Sport and Fitness Journal
E-ISSN: 2654-9182 Volume 11, No.2, May 2023: 161-169

EFFECT OF ACUTE EXERCISE FOR GENITAL SEXUAL AROUSAL IN FEMALES: A LITERATURE REVIEW

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ABSTRACT

Females often give less attention to sexual dysfunction, which is mainly reduced genital sexual arousal. Acute exercise is reported to increase the expression of hormones that may have an indirect effect on the physiology of the genital system. Due to its practicality, acute exercise has the potential to become one of the therapeutical options for genital sexual arousal dysfunction. Therefore, we would like to perform a literature review to describe the effect of acute exercise on genital sexual arousal. Literature searching was performed using keywords (acute exercise) AND (sexual arousal) to obtain eligible articles. The literature search was performed in PubMed, EMBASE, CENTRAL, and Google Scholar. The findings in eligible articles were reviewed and described narratively. Acute exercise is reported to have an influence on the expression of hormones including estrogen, cortisol, prolactin, testosterone, and oxytocin. The cortisol, estrogen, and testosterone increase genital sexual arousal by direct effect. Genital sexual arousal occurs due to the increased thickness of the vaginal epithelium, increase vaginal lubrication, and increase genital sensations. Prolactin and oxytocin influence genital sexual arousal by indirect effect. Acute exercise also enhances the activity of the sympathetic nervous system which leads to increased genital sexual arousal in females. The increased sympathetic tone leads to increase cardiac output and vasodilatation. Vasodilatation causes an increase in blood flow to the genital organ. Acute exercise increases genital sexual arousal in females via hormonal and sympathetic nervous system pathways.

Keywords: acute exercise; genital; hormonal; sexual arousal, sympathetic

INTRODUCTION

Sexual activity is an important part of human life as reproduction and recreation function. Therefore it is very essential for either males or females. The sexual function are ranging from desire, arousal, orgasm, and resolution. Dysfunction of each sexual function can cause a reduction in the quality of life. Sexual dysfunction can occur both in males and females. Female sexual dysfunction is a common problem and its prevalence reaches 38 – 63% of all women. The prevalence of sexual arousal dysfunction is ranging from 30 – 50% of all female sexual dysfunction.[1] Male sexual dysfunction is quite common and extensively studied. Effective therapies for male sexual dysfunction are available. On the other hand, female sexual dysfunction is less understood and more complicated compared to male sexual dysfunction.[1,2]

Female in Indonesia pays less attention to their sexual function. It may be associated with culture, society, economy, and other factors that make sexual function and sexual satisfaction less important to females. Exercise has been well-known to increase health both physically and mentally in various studies.[2] Research to investigate the association between exercise and sexual function is fewer than on the impact of exercise on physical and mental health. The physiological mechanism during exercise has been reported to affect female sexual function, especially genital sexual arousal.

Exercise does not carry stigma so it could be the treatment of choice for female sexual dysfunction(s). People are usually not interested in seeking medical and non-medical helps with their sexual problems because of hesitation, shame, and fear. Primary care physicians usually have difficulties talking about sexual problems in the examination room which potentially causes misadvice on prevention and treatment recommendations.[2,3] Therefore, exercise could be a treatment of choice, either as a single treatment or a complement of other treatments.

Exercise itself can be classified into acute and chronic exercise. Regular exercise (chronic exercise) is better than acute exercise because of its regularity and causes physiological adaptation to the body. However, not everyone is able to perform regular exercise due to their busy activities. Therefore, we thought that acute exercise could be a potential answer to overcome the problem of sexuality. In this review, we aimed to describe narratively the effect of acute exercise on genital sexual arousal.

METHODS

This study was a review of the literature that was elaborated narratively. The PICO framework was initially constructed to identify the key to creating eligibility criteria. Based on the PICO framework, the population in this study was female and active sexually; the interest was acute exercise; the comparator was none; and the outcome was genital sexual arousal. The keys on the PICO framework were used further to create the keywords as the basis to perform literature searching in this review. The keywords used in this review were (acute exercise) AND (sexual arousal). We performed a literature search on several online databases which include PubMed, EMBASE, CENTRAL, and Google Scholar. Articles in English that describe acute exercise and its relation with genital sexual arousal in women were considered eligible. We included studies comparing acute exercise and reported assessment of genital sexual arousal in females which were published in English. The eligible studies were further reviewed and elaborated narratively.

The initial literature search using predefined keywords result in 93 articles. We removed the duplicate articles in each database. Then, we performed title and abstract screening of these 93 articles. One article was excluded due to examining chronic exercise. Therefore, we obtained four articles that were eligible for this review.

RESULTS

As can be seen in **Table 1**, we provided a summary of the findings of included articles.

Table 1. Summary of findings of included articles

Author	Sample	Intervention	Result
Meston and Gorzalka	Twelve women were sexually functional, 12 experienced significant impairments in sexual desire, and 12 experienced primary or secondary anorgasmia.	In 2 experimental sessions, 36 women viewed a neutral film followed by an erotic film. In 1 session, the women were exposed to 20 min of intense exercise before viewing the films.	Acute exercise significantly increased vaginal pulse amplitude (VPA) and vaginal blood volume (VBV) responses to an erotic film among sexually functional women and those with low sexual desire. Among anorgasmic women, exercise significantly decreased VPA but had no effect on VBV responses to an erotic film. Acute exercise had no significant effect on the women's perceptions of sexual arousal.
Meston, *et al*.	Thirty women between the ages of 18 and 45 years, no	Thirty sexually functional women participated in two	Clonidine significantly decreased vaginal pulse amplitude, vaginal

	use of medications known to affect vascular or sexual functioning, no history of treatment for sexual dysfunction, no medical condition that may put die subject at risk when exercising, no history of high or low blood pressure, and current involvement in a heterosexual relationship.	experimental sessions in which subjective (self-report) and physiological (vaginal photoplethysmograph) sexual responses to erotic stimuli were measured after either clonidine (0.2 mg) or placebo administration in a randomized, double-blind, crossover protocol. Before viewing the experimental films, 15 subjects engaged in 20 minutes of intense exercise designed to elicit significant SNS activation.	blood volume, and subjective sexual responses to the erotic films in subjects who were in a state of heightened (via exercise), but not baseline (no exercise) sympathetic nervous system arousal.
Hamilton, *et al*.	Sixteen women aged 18 – 45 years old; pre-menopausal; free of sexual problems; free of any drugs or medical conditions or medications that could affect sexual arousal, symphatetic activity, or testosterone; and physically able to exercise	Sixteen participants came into the lab on two separate occasions. On 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.	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.
Lorenz and Meston.	Fourty-seven heterosexual or bisexual female, aged 18 years or older, taking an selective serotonin reuptake or selective noradrenaline reuptake inhibitor, and currently sexually active	Women reporting antidepressant-related sexual arousal problems (N = 47) participated in three counterbalanced sessions where they watched an erotic film while we recorded genital and sympathetic nervous system arousal. In two sessions, women exercised for 20 min, either 5 or 15 min prior to the films.	Genital arousal was significantly increased in the two exercise conditions relative to the no-exercise control, $F(1,43) = 8.63$, $p < 0.05$; moreover, genital arousal was significantly higher 5 min post-exercise than 15 min post-exercise, $F(1,43) = 3.20$, $p < 0.05$

DISCUSSION

Exercise is divided into acute and chronic phases. Acute exercise is any exercise (endurance, resistance, high-intensity training) which not repeated regularly. When the exercise is repeated regularly, then it is called chronic exercise. We did not find any clear time limitation or amount of repetition in the literature. Several articles even stated that chronic exercise is an exercise that is repeated regularly for years and becomes routine. In the acute phase, the exercise causes a physiological adjustment to maintain homeostasis. Meanwhile, in the chronic phase, the exercise cause physiological adaptation for the long term. Acute exercise causes an increase in the metabolic rate and is followed by proportional metabolic and

neuromuscular changes during and immediately after exercise. The metabolic changes during and immediately after the acute exercise are transient according to the demand for an increase in metabolic rate. The consumption of oxygen increased from 3 mL oxygen/kg/minute to 50 and 70 mL oxygen/kg/minute and back to the baseline a few hours after the exercise.[4] The exercise also causes an increased contraction of the muscle which causes higher flow of the blood to the active muscle. The higher blood flow leads to increased heart rate, respiratory rate, body temperature, and secretion of hormones due to stress such as adrenocorticotrophic, cortisol, and catecholamine.[5] The goal of these adjustments is to maintain homeostasis during exercise.[4]

The initial sign that can be observed as a sign of genital arousal in females is vaginal lubrication.[6] The vaginal transudate is produced by glands and epithelia on both sides of the vagina. The vaginal transudate or basal fluid causes moisturization of the vaginal surface to prevent adhesion and pain during penetration. Increasing sexual arousal causes vascular congestion in the tissue, leading to increased hydrostatic capillary pressure. The increase in hydrostatic capillary pressure leads to an increased volume of fluid on the vaginal epithelium surface.[7]

Estrogen has an important role in facilitating the growth and function of smooth muscles, neurons, vessels, and cells in the endothelium and epithelium by regulating the cellular process within the vaginal tissue. Therefore, estrogen receptors in the smooth muscle cells and on the vaginal epithelium are an important structure to maintaining vaginal lubrication, the integrity of the vaginal tissue, the thickness of the vagina wall, and maintaining the transmission of impulses along the nerve from the vagina to the brain.[8] The thicker epithelium of the vagina had a high amount of microvascular which caused higher blood flow and leads to improvement in vaginal lubrication.[9,10]

The natrium and kalium in the vaginal tissue also influence vaginal lubrication.[11] The fluid in the vagina contains a high concentration of kalium and a low concentration of natrium in a steady state.[11,12] When sexual arousal is initiated, the vaginal epithelial cell enhances the capacity for sodium transportation.[12] The sodium is on the vaginal surface and causes base environment in the canal of the vagina.[13,14]

Androgens such as dehydroepiandrosterone (DHEA) can increase vaginal lubrication through aromatization to estrogens.[15] The majority of endogenous estrogens (75 – 100%) in females before and after menopause is DHEA and its DHEA sulfate.[16,17] Reduced circulating estrogen cause a thickness reduction of the epithelial of the vagina and smooth muscle atrophy in the vaginal wall. These changes lead to decrease vasodilation, lubrication, and genital sensations.[18]

Following sexual stimulation, vaginal lubrication occurs and is followed by increased vaginal vascular congestion.[19] In the early phase of sexual arousal, dilatation of the precapillary artery occurs gradually which leads to increased venous output.[20] The high blood flow due to vascular dilatation cause swelling of the vestibule and venous plexus around the vagina and makes the vaginal wall manifest as dark purple color. Vascular dilatation occurs because of increased cardiac output and smooth muscle relaxation of the arteries supplying the genitalia.[21] Complete vaginal vascular congestion occurs when the outer third of the vagina has expanded completely.[6]

The higher blood volume can be initiated by peripheral sensory input which leads to the activation of the central nervous system (CNS). The peripheral sensory input or stimulation such as manual, oral, and genital is essential for genital sexual arousal. The peripheral nerves that contain afferent and efferent fibers regarding genital regulation are including the pudendal, vagal, hypogastric, and pelvic nerves.[22-24]

The vaginal vascular congestion during sexual arousal is also facilitated by the parasympathetic and sympathetic nervous systems. The sacral parasympathetic motor neurons

innervate the pelvic organs and are controlled by the pelvic organ stimulating center (POSC) which is located in the pons. The activation of sacral parasympathetic motor neurons by POSC leads to vascular congestion and vagina lubrication.[25] In the later stage of sexual arousal, the sympathetic nervous system is taking over and leads to a higher heart rate and increased blood pressure during orgasm.[26,27]

The sex hormones such as estradiol and testosterone increased the blood flow to the genital organ by regulating vasoactive intestinal polypeptide (VIP) and nitric oxide synthase (NOS) activity.[28-30] The muscle fibers in the vagina have an abundant supply of immunoreactive VIP and NOS that are regulated by estrogen and androgen.[31] The expression as well as the activity of neural and endothelial NOS are regulated by estradiol, which further regulates the smooth muscle relaxation of the vagina and clitoris.[32] A study reported VIP only increased vaginal blood flow.[30] Testosterone plays role in the regulation of NOS and enhancing the smooth muscle relaxation of the vagina due to the regulation of VIP.[33] Prolactin and oxytocin also play an indirect role in genital sexual arousal. Increased serum prolactin may send negative feedback signaling to limit sexual arousal in order to stop sexual activity.[34] Oxytocin is reported to be related to orgasm than to genital sexual arousal.[35,36]

Several research has reported a positive association between acute exercise and genital sexual arousal where acute exercise can increase genital sexual arousal in women.[37] Acute exercise is reported to have an influence on the expression of hormones such as prolactin, cortisol, oxytocin, estrogen, and testosterone.[38-43] All of those hormones have an association with genital sexual arousal as shown above.

There are several factors affecting the effect of exercise on hormonal response in females, which includes the type of exercise, menopause status, and menstrual cycle.[37] Moderate- to high-intensity (60 – 80% VO2 max) exercise cause increased cortisol level, while low-intensity exercise reduces the cortisol level.[38] There was an increase in estradiol during the luteal phase following 30 minutes of moderate exercise (60% VO2 max).[44] Moderate to vigorous aerobic exercise leads to an increase in estrogen metabolism.[39]

Exercise also increases other hormones. There was an increase in prolactin concentrations after physical activity.[45] Furthermore, there was an increase in oxytocin levels after prolonged endurance exercise. However, it was reported that in short high-intensity exercise or stable runs, the oxytocin level did not increase.[41] Testosterone level increases after aerobic exercise[46] but not after resistance exercise.[47,48]

As described above in the physiology of genital sexual arousal, these hormones directly or indirectly affect genital sexual arousal in females. The cortisol levels during sexual arousal in females were increased.[49] Estrogen has been known as an important hormone as the regulator of genital sexual arousal in females. This hormone improves nerve transmission from the peripheral to the central nervous system. The thickness of the epithelium of the vagina and the mass of smooth muscle of the vagina are maintained by estrogen. The thickness reduction of the vaginal epithelium and vaginal smooth muscle atrophy cause lesser vasodilatation and reduction of sensation in the genital.[18] Prolactin, as described above, has an indirect effect on genital sexual arousal in females. A high level of prolactin leads to negative feedback that reduces sexual arousal to stop sexual activity.[34,50] Oxytocin is reported to facilitate orgasm function.[35,36] However, the orgasm function gives positive feedback to increase genital sexual arousal.[37] The androgen hormone (testosterone and dihydrotestosterone) also increased genital sexual arousal via aromatization to estrogens.[15]

Acute exercise also increases the activation of the sympathetic nervous system which caused increased genital sexual arousal in females.[37] As has been known, increased activity of the sympathetic leads to higher heart rate and increased blood pressure during orgasm.[26,27] Increased genital sexual arousal is also associated with increased norepinephrine.[51]

It was reported that exercise activating the sympathetic nervous system increases genital arousal. The 20 minutes of intense exercise which activates the domination of the sympathetic nervous system could increase the blood flow to the genital and prepare the vagina for sexual arousal.[52] However, genital sexual arousal is inhibited immediately after the exercise and increases within 15 – 30 minutes after the exercise. It is caused by the shift of blood flow to the contracting skeletal muscle to maintain homeostasis.[53]

The moderate increase in activity of the sympathetic nervous system is the optimal level and most beneficial for increasing genital sexual arousal. Furthermore, they found that significant relationship between heart rate variability and sexual arousal in females that indicate the association between the sympathetic nervous system and genital sexual arousal.[54] Heart rate variability (HRV) is a noninvasive index of the relative balance between the sympathetic and parasympathetic nervous systems.[55] The low resting HRV is predictive of sexual arousal problems in women.[56] Increasing HRV increases genital sexual arousal in females.[57]

As described above, acute exercise influences the expression of cortisol, estrogen, prolactin, oxytocin, and testosterone. The cortisol, estrogen, and testosterone increase genital sexual arousal by direct effect due to the increased thickness of the vaginal epithelium, increase vaginal lubrication, and increase genital sensations. Prolactin and oxytocin influence genital sexual arousal by indirect effect where an increase of this hormone cause negative feedback to stop the sexual activity. Acute exercise also increases the sympathetic nervous system's activation, leading to increased genital sexual arousal by increased cardiac output and vasodilatation. Vasodilatation causes an increase in blood flow to the genital organ, therefore causing increased vaginal lubrication and genital engorgement. On the other hand, chronic exercise cause chronic changes in the physiology of the human body. Chronic exercise causes an improvement of the cardiovascular system which improves the circulation to the genital. The improvement of circulation to the genital cause enhanced genital sexual arousal due to better vascular congestion to the clitoral and vaginal.[58]

The limitation of this study was that we did not investigate which type of acute exercise has the most benefit for genital sexual arousal. We did not investigate that because we did not find any description regarding the type of exercise in the included studies. Therefore, future studies should investigate the type of acute exercise (endurance, resistance, high-intensity training) that greatly enhances genital sexual arousal. Furthermore, future studies should investigate the optimum heart rate during exercise that gives benefit to genital sexual arousal.

CONCLUSION

Acute exercise increases genital sexual arousal in females via hormonal and sympathetic nervous system pathways. Acute exercise influences the expression of hormones such as prolactin, estrogen, cortisol, oxytocin, and testosterone. Acute exercise also increases sympathetic tone which further causes increased genital sexual arousal. Further, we suggested conducting research to explore which specific exercise (endurance, resistance, high-intensity training) causes more genital sexual arousal.

CONFLICT OF INTEREST

The authors declared the absence of a conflict of interest regarding the preparation of this manuscript.

FUNDING

The authors declare received no funding to sponsor the process of the preparation of this manuscript.

AUTHOR CONTRIBUTION

All the authors had equal contributions during the preparation of this manuscript and agree to accept equal responsibility regarding the content of the article.

REFERENCES

1. Jaafarpour M, Khani A, Khajavikhan J, Suhrabi Z. Female sexual dysfunction: Prevalence and risk factors. *J Clin Diagn Res*. 2013; 7(12): 2877 – 2880.
2. Parish SJ, Hahn SR. Hypoactive sexual desire disorder: A review of epidemiology, biopsychology, diagnosis, and treatment. *Sex Med Rev*. 2016; 4(2): 103 – 120.
3. Muhammad R, Horey D, Liamputtong P, Low WY. Managing women with sexual dysfunction: Difficulties experienced by Malaysian Family Physicians. *Arch Sex Behav*. 2019; 48(3): 949 – 960.
4. Lambert MI. General adaptations to exercise: acute versus chronic and strength versus endurance training. In: Vaamonde D, ed. Exercise and human reproduction. New York, NY: Springer Science þ Business Media; 2016: p. 93 – 100.
5. Gottschall JS, Davis JJ, Hastings B, Porter HJ. Exercise time and intensity: How much is too much? *Int J Sports Physiol Perform*. 2020; 15(6): 808 – 815.
6. Yeung J, Pauls RN. Anatomy of the vulva and the female sexual response. *Obstet Gynecol Clin North Am*. 2016; 43(1): 27 – 44.
7. Pastor Z, Chmel R. Differential diagnostics of female "sexual" fluids: A narrative review. *Int Urogynecol J*. 2018; 29(5): 621 – 629.
8. Santoro N, Worsley R, Miller KK, Parish SJ, Davis S. Role of estrogens and estrogen-like compounds in female sexual function and dysfunction. *J Sex Med.* 2016; 13: 305 – 316.
9. Sun Q, Huang J, Yang D-L, Cao X-N, Zhou W-L. Activation of β-adrenergic receptors during sexual arousal facilitates vaginal lubrication by regulating vaginal epithelial Cl (-) secretion. *J Sex Med*. 2014; 11(8): 1936 – 48.
10. Cappelletti M, Wallen K. Increasing women's sexual desire: The comparative effectiveness of estrogens and androgens. *Horm Behav*. 2016; 78: 178 – 93.
11. O'Hanlon DE, Moench TR, Cone RA. Vaginal pH and microbiocidal lactic acid when lactobacilli dominate the microbiota. PLoS One. 2013; 8(11): e80074.
12. Pastor Z, Chmel R. Differential diagnostics of female "sexual" fluids: a narrative review. *Int Urogynecol J*. 2018; 29(5): 621 – 629.
13. Zeinalzadeh S, Mohagheghzadeh AA, Ahmadinezhad F. Comparison of the effect of Elaeagnus angustifolia flower capsule and sildenafil citrate tablet female sexual interest/arousal disorder in clinical trial study. *J Family Med Prim Care*. 2019; 8(11): 3614 – 3620.
14. Jannini EA, Buisson O, Rubio-Casillas A. Beyond the G-spot: Clitourehtrovaginal complex anatomy in female orgasm. *Nat Rev Urol*. 2014; 11(9): 531 – 8.
15. Calabro RS, Cacciola A, Bruschetta D, Milardi D, Quattrini F, Sciarrone F, *et al*. Neuroanatomy and function of human sexual behavior: A neglected or unknown issue? *Brain Behav.* 2019; 9(12): e01389.
16. Kamin HS, Kertes DA. Cortisol and DHEA in development and psychopathology. *Horm Behav*. 2017; 89: 69 – 85.
17. Pitsava G, Maria AG, Faucz FR. Disorders of the adrenal cortex: Genetic and molecular aspects. *Front Endocrinol (Lausanne)*. 2022; 13: 931389.
18. Levin RJ, Both S, Georgiadis J, Kukkonen T, Park K, Yang CC. The physiology of female sexual function and the pathophysiology of female sexual dysfunction (Committee 13A). *J Sex Med*. 2016; 13(5): 733 – 59.

19. Worsley R, Bell RJ, Gartoulla P, Davis SR. Prevalence and predictors of low sexual desire, sexually related personal distress, and hypoactive sexual desire dysfunction in a community-based sample of midlife women. *J Sex Med*. 2017; 14(5): 675 – 686.
20. Faubion SS, Rullo JE. Sexual dysfunction in women: A practical approach. Am Fam Physician. 2015; 92(4): 281 – 8.
21. Graziottin A, Gambini D. Anatomy and physiology of genital organs- women. *Handb Clin Neurol.* 2015; 130: 39 – 60.
22. Cour F, Droupy S, Faix A, Methorst C, Giuliano F. Anatomy and physiology of sexuality. *Prog Urol*. 2013; 23(9): 547 – 61.
23. Pelliccioni G, Piloni V, Sabbatini D, Fioravanti P, Scarpino O. Sex differences in pudendal somatosensory evoked potentials. Tech Coloproctol. 2014; 18(6): 565 – 9.
24. Meston CM, Stanton AM. Understanding sexual arousal and subjective-genital arousal desynchrony in women. *Nat Rev Urol*. 2019; 16(2): 107 – 120.
25. Holstege G. How the emotional motor system controls the pelvic organs. *Sex Med Rev.* 2016; 4: 303 – 328.
26. Bouchard KN, Dawson SJ, Shelley AJ, Pukall CF. Concurrent measurement of genital lubrication and blood flow during sexual arousal. *Biol Psychol*. 2019; 145: 159 – 166.
27. Brody S, Klapilova K, Krejcova L. More frequent vaginal orgasm is associated with experiencing greater excitement from deep vaginal stimulation. *J Sex Med.* 2013; 10(7): 1730 – 6.
28. Oliver JL, Kavoussi PK, Smith RP, Woodson RI, Corbett ST, Costabile RA, *et al*. The role of regulatory proteins and S-nitrosylation of endothelial nitric oxide synthase in the human clitoris: Implications for female sexual function. *J Sex Med*. 2014; 11(8): 1927 – 35.
29. Vink JY, Qin S, Brock CO, Zork NM, Feltovich HM, Chen X, *et al*. A new paradigm for the role of smooth muscle cells in the human cervix. *Am J Obstet Gynecol*. 2016; 215(4): 478.
30. Hu J-M, Cheng X, Wang L, Zhu J-N, Zhou L-H. Vasoactive intestinal peptide expression in the vaginal anterior wall of patients with pelvic organ prolapse. *Taiwan J Obstet Gynecol*. 2013; 52(2): 233 – 40.
31. Li T, Liao Q, Zhang H, Gao X, Li X, Zhang M. Anatomic distribution of nerves and microvascular density in the human anterior vaginal wall: Prospective study. *PLoS One*. 2014; 9(11): e110239.
32. Sawatsky ML, Dawson SJ, Lalumiere ML. Genital lubrication: A cue-specific sexual response? *Biol Psychol*. 2018; 134: 103 – 113.
33. Palacios S. Expression of androgen receptors in the structures of vulvovaginal tissue. *Menopause*. 2020; 27(11): 1336 – 1342.
34. Parada M, Gerard M, Larcher K, Dagher A, Binik YM. Neural representation of subjective sexual arousal in men and women. *J Sex Med*. 2016; 13(10): 1508 – 22.
35. Motta-Mena NV, Puts DA. Endocrinology of human female sexuality, mating, and reproductive behavior. *Horm Behav*. 2017; 91: 19 – 35.
36. Behnia B, *et al*. Differential effects of intranasal oxytocin on sexual experiences and partner interactions in couples. *Horm Behav.* 2014; 65: 308 – 318.
37. Stanton AM, Handy AB, Meston CM. The effects of exercise on sexual function in women. *Sex Med Rev*. 2018; 6(4): 548 – 557.
38. Duclos M, Tabarin A. Exercise and the hypothalamo-pituitary-adrenal axis. *Front Horm Res*. 2016; 47: 12 – 26.
39. Smith AJ, Phipps WR, Thomas W, Schmitz KH, Kurzer MS. The effects of aerobic exercise on estrogen metabolism in healthy premenopausal women. *Cancer Epidemiol Biomarkers Prev.* 2013; 22: 756 – 764.

40. Hackney AC, Saeidi A. The thyroid axis, prolactin, and exercise in humans. *Curr Opin Endocr Metab Res*. 2019; 9: 45 – 50.
41. Broderick TL, Jankowski M, Gutkowska J. The effects of exercise training and caloric restriction on the cardiac oxytocin natriuretic peptide system in the diabetic mouse. *Diabetes Metab Syndr Obes*. 2017; 10: 27 – 36.
42. Hackney AC, Walz EA. Hormonal adaptation and the stress of exercise training: The role of glucocorticoids. *Trends Sport Sci*. 2013; 20(4): 165 – 171.
43. Hooper DR, Kraemer WJ, Focht BC, Volek JS, DuPont WH, Caldwell LK, Maresh CM. Endocrinological roles for testosterone in resistance exercise responses and adaptations. *Sports Med*. 2017; 47(9): 1709 – 1720.
44. Cho GJ, Han SW, Shin J-H, Kim T. Effects of intensive training on menstrual function and certain serum hormones and peptides related to the female reproductive system. *Medicine* (Baltimore). 2017; 96(21): e6876.
45. Schumann M, Mykkanen O-P, Doma K, Mazzolari R, Nyman K, Hakkinen K. Effects of endurance training only versus same-session combined endurance and strength training on physical performance and serum hormone concentrations in recreational endurance runners. *Appl Physiol Nutr Metab*. 2015; 40(1): 28 – 36.
46. Lane A, O'Leary C, Hackney A. Menstrual cycle phase effects free testosterone responses to prolonged aerobic exercise. *Acta Physiol Hung*. 2015; 102: 336 – 341.
47. Herman-Montemayor JR, Hikida RS, Staron RS. Early-phase satellite cell and myonuclear domain adaptations to slow-speed vs. traditional resistance training programs. *J Strength Cond Res*. 2015; 29(11): 3105 – 14.
48. Smilios I, Tsoukos P, Zafeiridis A, Spassis A, Tokmakidis SP. Hormonal response after resistance exercise performed with maximum and submaximum movement velocities. *Appl Physiol Nutr Metab*. 2014; 39(3): 351 – 7.
49. Alley J, Diamond LM, Lipschitz DL, Grewen K. Women's. cortisol stress responsivity, sexual arousability, and sexual history. *Arch Sex Behav*. 2020; 49(5): 1489 – 1503.
50. Paterson LQ, Jin ES, Amsel R, Binik YM. Gender similarities and differences in sexual arousal, desire, and orgasmic pleasure in the laboratory. *J Sex Res*. 2014; 51(7): 801 – 13.
51. Levin RJ. Recreation and procreation: A critical view of sex in the human female. *Clin Anat.* 2015; 28(3): 339 – 54.
52. Stanton AM, Lorenz TA, Pulverman CS, Meston CM. Heart rate variability: A risk factor for female sexual dysfunction. *Appl Psychophysiol Biofeedback*. 2015; 40(3): 229 – 7.
53. Teixeira AL, Vianna LC. The exercise pressor reflex: An update. *Clin Auton Res*. 2022; 32(4): 271 – 290.
54. Handy AB, Stanton AM, Pulverman CS, Meston CM. Differences in perceived and physiological genital arousal between women with and without sexual dysfunction. *J Sex Med*. 2018; 15(1): 52 – 63.
55. Cygankiewicz I, Zareba W. Heart rate variability. *Handb Clin Neurol*. 2013; 117: 379 – 93.
56. Stanton AM, Lorenz TA, Pulverman CS, Meston CM. Heart rate variability: a risk factor for female sexual dysfunction. *Appl Psychophysiol Biofeedback.* 2015; 40: 229 – 237.
57. Stanton AM, Meston CM. A single session of autogenic training increases acute subjective and physiological sexual arousal in sexually functional women. *J Sex Marital Ther.* 2016; 43: 601 – 617.
58. Stanton AM, Handy AB, Meston CM. The effects of exercise on sexual function in women. *Sex Med Rev*. 2018; 6(4): 548 – 557.