Journal of Chemical Health Risks

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JCHR (2026) 16(2), 2843 - 2849

Comparison of Dehydroepiandrosterone (DHEA) and Cortisol Hormone Levels in Premenopausal Women with Sexual Dysfunction

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(Received: 16 February 2026 Revised: 25 March 2026 Accepted: 05 April 2026)

KEYWORDS

Cortisol; DHEA; Premenopausal Women; Sexual Dysfunction; Hormones

ABSTRACT:

Introduction: Sexual dysfunction is common in premenopausal women. Dehydroepiandrosterone (DHEA) and cortisol are thought to play a key role in the mechanism of sexual dysfunction in premenopausal women by affecting the balance of androgens and estrogens.

Objectives: to compare the dehydroepiandrosterone (DHEA) and cortisol hormone levels in premenopausal women with sexual dysfunction.

Methods: This analytical observational study with a cross-sectional design was conducted at Wahidin Sudirohusodo Central General Hospital and Pepabri Health Center, Makassar, Indonesia, from January to June 2025. Seventy-eight premenopausal women aged 40-50 years were enrolled, consisting of 39 with sexual dysfunction and 39 without, determined by the Female Sexual Function Index (FSFI). Serum cortisol and DHEA levels were measured using enzyme-linked immunosorbent assay (ELISA).

Results: Cortisol levels were significantly higher in premenopausal women with sexual dysfunction compared to those without (median 9.05 vs 7.87 µg/dL). Conversely, DHEA levels were significantly lower in the sexual dysfunction group (median 0.82 vs 2.05 ng/mL). Cortisol levels were negatively correlated with sexual desire (r=-0.603) and orgasm (r=-0.349), while DHEA levels were positively correlated with sexual desire (r=0.423) and arousal (r = 0.347).

Conclusions: Elevated cortisol and reduced DHEA levels are associated with sexual dysfunction in premenopausal women, particularly affecting desire, arousal, and orgasm. These hormones may serve as potential biomarkers or therapeutic targets for improving sexual function.

1. Introduction

Sexual dysfunction refers to a condition in which an individual experiences difficulty in achieving satisfaction from sexual intercourse, thereby disrupting the normal sexual response cycle and impairing sexual enjoyment or fulfillment [1]. The prevalence of sexual dysfunction is age-related and commonly occurs during the premenopausal period, affecting approximately 30% to 50% of women [2]. Menopause represents a major physiological transition in a woman's life, characterized by the cessation of menstruation for at least 12 consecutive months due to a natural decline in estrogen levels. This phase typically occurs around the age of 51 and is marked by reduced ovarian follicle reserves and compensatory elevations in follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Hormonal imbalance resulting from estrogen deficiency may disrupt menstrual regularity and sexual health [3].

Dehydroepiandrosterone (DHEA) and its sulfate ester (DHEAS) play a key role in maintaining androgen and estrogen balance, influencing sexual function in women. They are endogenous steroid hormones produced by the zona reticularis of the adrenal cortex in response to adrenocorticotropic hormone (ACTH). DHEA production peaks between the ages of 25 and 35, followed by a gradual decline, reaching only 10-20% of its maximal concentration by the age of 70 [4]. Several studies have demonstrated associations between DHEA levels and female sexual function across different populations, but data specifically examining premenopausal women remain limited. Furthermore,Cortisol, commonly known as the stress hormone, also plays a significant role in hormonal homeostasis and may influence sexual function [5]. It is synthesized from cholesterol in the zona fasciculata of the adrenal cortex under the stimulation of ACTH secreted by the anterior pituitary gland. The majority of cortisol in circulation is bound to corticosteroid-binding globulin (CBG) or albumin, rendering it inactive. Activation occurs through the enzyme 11β-

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hydroxysteroid dehydrogenase type 1 (11β-HSD1), while inactivation occurs through 11β-HSD2 in specific tissues such as the kidneys and pancreas [6]. Dysregulation of cortisol metabolism has been linked to disturbances in sexual function, as abnormal cortisol responses can interfere with other hormonal pathways and diminish sexual desire, arousal, and satisfaction [7].

A previous study reported differential cortisol responses to sexual stimuli among women, particularly those with a history of sexual trauma. Women exhibiting heightened cortisol reactivity during sexual arousal displayed lower sexual function scores, including reduced desire, arousal, and satisfaction [8]. These findings underscore the potential role of cortisol imbalance in the pathophysiology of female sexual dysfunction. Sexual dysfunction in premenopausal women can substantially impair quality of life, affecting interpersonal relationships, self-esteem, and psychological well-being. Understanding the endocrine mechanisms underlying this condition particularly the roles of DHEA and cortisol is essential for improving prevention and management strategies. While prior research has explored the relationship between these hormones and sexual function, most studies have focused on postmenopausal or older populations. Therefore, data comparing DHEA and cortisol levels specifically in premenopausal women with and without sexual dysfunction remain scarce [9].

This study aims to compare the levels of DHEA and cortisol hormones in premenopausal women with sexual dysfunction and those without, to elucidate the hormonal patterns associated with sexual function in this population. The findings may provide valuable insights for optimizing the diagnosis and management of sexual dysfunction in premenopausal women and contribute to improving their overall sexual well-being and quality of life.

2. Objectives

The purpose of this study was to evaluate the comparison of dehydroepiandrosterone (DHEA) and cortisol hormone levels in premenopausal women with sexual dysfunction**.**

3. Methods

Study Design and Participants

This study employed an analytical observational design with a cross-sectional approach. The research was conducted at Wahidin Sudirohusodo Central General Hospital, the Educational Network Hospital of the Department of Obstetrics and Gynecology, Faculty of Medicine, Hasanuddin University, and the Pepabri Health Center, Makassar, Indonesia. The data collection period was carried out from January to June 2025 until the required number of samples was achieved.

Study population are all premenopausal women living in Makassar. The research samples were selected from premenopausal women attending the participating health facilities who met the inclusion and exclusion criteria. Sampling was conducted using a sequential sampling technique, where all eligible subjects who met the inclusion criteria during the research period were recruited until the sample size was fulfilled. The minimum sample size was calculated based on the formula for comparing mean values between two populations, namely women with and without sexual dysfunction. The inclusion criteria consisted of premenopausal women aged 40-50 years, with and without sexual dysfunction as assessed by the Female Sexual Function Index (FSFI). Participants were required to provide informed consent prior to inclusion. Exclusion criteria included a history of mental disorders, diabetes mellitus, chronic venous insufficiency, urogenital surgery, or gynecological surgery, as well as the use of hormone replacement therapy or hormonal contraceptives. Samples that were damaged or participants who withdrew consent were excluded from the analysis

Data Collection

After identifying research subjects who met the research criteria, all subjects completed the FSFI questionnaire. An FSFI score of ≤ 26.55 was considered sexual dysfunction, and a score of >26.55 was considered normal sexual function. All subjects then underwent cortisol and DHEA levels tests using the enzyme-linked immunosorbent assay (ELISA) method. Venous blood samples (3 mL) were collected from each participant and stored in tubes containing EDTA as an anticoagulant. The samples were transported to the laboratory for hormonal analysis. Serum levels of DHEA and cortisol were determined using ELISA kits following standardized procedures. Serum was first separated by centrifugation at 2000-3000 rpm for approximately 30 minutes. Reagents and samples were prepared at room temperature, and the ELISA procedure was performed according to the manufacturer's instructions. The optical density (OD) of each well was measured at 450 nm using a microplate reader, and the results were used to calculate hormone concentrations.

Statistical Analysis

Data were tabulated in Microsoft Excel and subsequently analyzed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize demographic and clinical characteristics, including frequency, percentage, median, minimum, and maximum values. The Shapiro-Wilk test was used to assess data normality. Because the distribution was non-normal, the Mann-Whitney U test was employed to compare mean hormone levels between groups. Categorical variables were analyzed using the chi-square test, with a significance level set at $p < 0.05$.

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Ethical Clerance

This research was conducted after obtaining ethical approval from the Biomedical Research Ethics Commission of the Faculty of Medicine, Hasanuddin University, Makassar, Indonesia. All participants received an explanation regarding the study purpose, benefits, and procedures. Participation was voluntary, and respondents retained the right to withdraw at any stage without penalty. The confidentiality of participants' identities and data was strictly maintained throughout the study.

4. Results

Basic Characteristics of the Subject

In this study, 78 premenopausal women were collected, consisting of 39 premenopausal women with sexual dysfunction and 39 premenopausal women with no sexual dysfunction who met the inclusion and exclusion criteria. The characteristics of the subjects of this study are presented in Table 1. Table 1 shows that age, occupation, education, nutritional status and parity do not differ significantly between the sexual dysfunction and non-sexual dysfunction groups with a p> value of 0.05. Thus, the characteristics of the two groups of research subjects are homogeneous.

Table 1. Frequency distribution of the characteristics of the research subject

Characteristic	Sexual dysfunction (n = 39)	Non-sexual dysfunction (n = 39)	p-value
Age (Years)[a]	46.0 (41.0-49.0)	45.0 (41.0-50.0)	0.230
Occupation[b]			
Working	28 (71.8)	24 (61.5)	0.337
Not working	11 (28.2)	15 (38.5)	
Education[b]			
Junior high school	2 (5.1)	4 (10.3)	
Senior high school	33 (84.6)	35 (89.7)	0.094
College	4 (10.3)	0 (0.0)	
Nutritional Status[b]			
Normal	20 (51.3)	15 (38.5)	
Overweight	12 (30.8)	13 (33.3)	0.440
Obesity	7 (17.9)	11 (28.2)	
Parity[b]			
Nullipara	5 (12.8)	1 (2.5)	
Multipara	33 (84.6)	34 (87.2)	0.106
Grand Multipara	1 (2.6)	4 (10.3)	

[a]Data presented in median (min-max), Mann Whitney test; [b]Data is presented in n (%), chi square test

The results of comparison of cortisol and dehydroepiandrosterone levels between sexual dysfunction and non-sexual dysfunction are presented in Table 2. Table 2 shows that there is a significant difference in cortisol levels between the sexual dysfunction and non-sexual dysfunction groups with a p< value of 0.05. The cortisol levels of premenopausal women with sexual dysfunction were higher than those of premenopausal women with no sexual dysfunction. There was a significant difference in dehydroepiandrosterone levels between the sexual dysfunction and non-sexual dysfunction groups with a p value of < 0.05. The levels of dehydroepiandrosterone in premenopausal women with sexual dysfunction were lower than in premenopausal women with no sexual dysfunction.

Table 3 shows that there is a significant relationship between cortisol levels and the domain of sexual dysfunction in sexual desire and orgasm with a p< value of 0.05. The correlation coefficient in the desire dimension of -0.603 indicates a negative and strong correlation between cortisol levels and disturbances in sexual desire. Negative correlation means that the lower the score of the sexual desire dimension, the higher the cortisol level. The correlation coefficient in the orgasm dimension of -0.349 indicates a negative and weak correlation between cortisol levels and disturbances in orgasm. Negative correlation means that the lower the orgasm dimension score, the higher the cortisol level. A lower score of the sexual dysfunction dimension indicates a higher sexual dysfunction disorder. Thus, higher sexual desire and orgasm disorders are associated with higher cortisol levels.

There was a significant relationship between dehydroepiandrosterone levels and the domain of sexual dysfunction in sexual desire and arousal with a p< value of 0.05. The correlation coefficient in the desire dimension of 0.423 indicates a positive and moderate correlation between dehydroepiandrosterone levels and disturbances in sexual desire. Positive correlation means that the lower the score of the sexual desire dimension, the lower the level of dehydroepiandrosterone. The correlation coefficient in the stimulus dimension of 0.347 indicates a positive and weak

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correlation between dehydroepiandrosterone levels and disturbances in sexual stimulation. Positive correlation means that the lower the score of the sexual stimulation dimension, the lower the level of dehydroepiandrosterone. A lower score of the sexual dysfunction dimension indicates a higher sexual dysfunction disorder. Thus, higher disturbances of desire and sexual arousal are associated with lower levels of dehydroepiandrosterone.

Table 1. Comparison of premenopausal female hormones cortisol and dehydroepiandrosterone levels between sexual dysfunction and non-sexual dysfunction

Marker	Sexual dysfunction (n = 39)	Non-sexual dysfunction (n = 39)	p-Value
	Median (min-max)	Median (min-max)	
Cortisol (mcg/dL)	9,05 (5,96-22,35)	7.87 (1.33-26.54)	0.004
Dehydroepiandrosterone (ng/mL)	0,82 (0,19-2,77)	2.05 (0.41-4.34)	< 0.001

Mann Whitney Test

Table 2. Correlation of cortisol and dehydroepiandrosterone levels with each domain of sexual dysfunction in premenopausal women with sexual dysfunction

Dimensions of sexual dysfunction	Cortisol levels		Dehydroepiandrosterone	
	r	p-value	r	p-value
Desire	-0.603	< 0.001*	0.423	0.012*
Stimulation	0.274	0.091	0.347	0.030*
Lubrication	0.312	0.053	0.119	0.471
Orgasm	-0.349	0.029*	0.155	0.345
Satisfaction	0.211	0.198	-0.056	0.733
Pain	0.287	0.076	-0.029	0.861

Spearman Correlation Test, *Significant at p < 0.05, r = 0-0.19 (very weak), r= 0.20-0.39 (weak), r= 0.40-0.59 (moderate), r= 0.60-0.79 (strong), r= 0.80-1.0 (very strong)

The results of the analysis of factors related to premenopausal female cortisol levels are presented in Table 4. Table 4 shows that occupation, education, nutritional status and parity were not significantly related to cortisol and dehydroepiandrosterone levels with p> values of 0.05. Thus, employment, education, nutritional status and parity were not confounding factors in this study

Table 3. Results of analysis of factors related to cortisol and dehydroepiandrosterone levels in premenopausal women

Characteristic	Cortisol (mcg/dL) Median (min-max)	p-value	Dehydroepiandrosterone (ng/mL) Median (min-max)	p-value
Occupation				
Working	8,67 (1,33-26,54)	0,458	1,37 (0,19-4,34)	0,849
Not working	9,44 (2,16-23,34)		1,23 (0,37-3,67)	
Education				
Junior high school	11,79 (5,73-23,24)		1,20 (0,60-2,52)	
Senior high school	8,82 (1,33-26,54)	0,413	1,29 (0,19-4,34)	0,956
College	9,79 (7,38-21,88)		1,52 (0,47-2,41)	
Nutritional status				
Normal	8,74 (1,33-22,38)		1,31 (0,32-4,34)	
Overweight	9,57 (4,82-23,34)	0,436	1,30 (0,19-3,69)	0,773
Obesity	9,10 (1,40-26,54)		1,36 (0,41-3,69)	
Parity				
Nullipara	9,98 (7,87-15,76)		0,98 (0,72-1,31)	
Multipara	8,81 (1,33-26,54)	0,620	1,45 (0,19-4,34)	0,639
Grand Multipara	9,91 (2,76-14,77)		2,27 (0,92-3,67)	

Mann Whitney Test

5. Discussion

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This study compared serum cortisol and DHEA levels between premenopausal women with and non-sexual dysfunction. The study population consisted of women with relatively homogeneous baseline characteristics in terms of age, occupation, education, nutritional status, and parity. The median age of participants was 46 years in the sexual dysfunction group and 45 years in the non-sexual dysfunction, consistent with the premenopausal period when hormonal fluctuations are most pronounced. The results demonstrated that cortisol levels were significantly higher in premenopausal women with sexual dysfunction than in those without dysfunction. Although studies focusing specifically on premenopausal women are limited, previous research in other populations has shown similar associations. A study among perimenopausal women reported that those with sexual dysfunction had higher cortisol levels compared to those without dysfunction [10]. Cortisol responses were associated with reduced sexual desire, arousal, and satisfaction in women aged 21-50 years. Similarly with observed a negative correlation between cortisol levels and the orgasm domain of FSFI in women with polycystic ovary syndrome (PCOS) [11]. These findings collectively suggest that elevated cortisol levels are consistently associated with the presence and severity of sexual dysfunction across different female populations, and the present study expands this evidence to premenopausal women [12].

High cortisol levels may contribute to sexual dysfunction through several neuroendocrine mechanisms. Dysregulation of the hypothalamic pituitary adrenal (HPA) axis has been implicated in decreased sexual desire and arousal. Cortisol can inhibit luteinizing hormone (LH) secretion and suppress estradiol activity [13]. Estradiol normally enhances the expression of serotonin transporters; thus, suppression of estradiol may reduce transporter expression and increase central serotonin concentration, which is known to inhibit sexual motivation and orgasm. Moreover, elevated cortisol is associated with altered brain activation patterns. Increased cortisol levels have been linked to hyperactivity in the precentral gyrus, caudate nucleus, and insula, regions involved in the regulation of emotional and somatosensory processing [14]. The posterior insula, which mediates sensations of desire, shows hypoactivity in individuals with elevated cortisol and negative mood states. Consequently, high cortisol may impair sexual desire by reducing posterior insula activation. Cortisol also influences orgasmic function [15]. During orgasm, activation occurs in the cerebellum, anterior cingulate cortex, and dopaminergic pathways connecting the ventral tegmental area and nucleus accumbens [10]. Elevated cortisol can suppress amygdala and prefrontal cortex activity and decrease functional connectivity between these regions, which may explain the observed reduction in orgasmic function in women with high cortisol levels [16].

In this study, the median cortisol level among premenopausal women with sexual dysfunction was 9.05 µg/dL, consistent with previous findings reporting levels of 10.89 µg/dL in perimenopausal women with dysfunction. Normal morning cortisol levels in adults typically range from 10 to 25 µg/dL, suggesting that although levels in this study were within the physiological range, women with sexual dysfunction exhibited relatively higher cortisol compared with those without dysfunction [17]. Morning cortisol measurement was chosen because cortisol secretion peaks after awakening and declines throughout the day. The results indicate that even within normal physiological limits, elevated cortisol may signal early neuroendocrine alterations related to sexual dysfunction in premenopausal women. Future research should aim to establish specific cortisol threshold values predictive of sexual dysfunction in this population [18].

Conversely, this study found that DHEA levels were significantly lower in premenopausal women with sexual dysfunction compared to those without. Although previous studies focusing exclusively on premenopausal women are scarce, the current results are in line with findings in other populations [19]. Women with Cushing's syndrome exhibited lower FSFI scores and decreased DHEA levels compared with healthy controls. Similarly, women with urinary incontinence and sexual dysfunction had significantly lower DHEA levels, while hypoactive sexual desire disorder (HSDD) was associated with reduced DHEA concentrations [20]. A systematic review further supported these findings, demonstrating that DHEA supplementation improved sexual interest, arousal, lubrication, and orgasm in women, particularly those with premenopausal and postmenopausal sexual dysfunction [21].

The decline in DHEA during premenopause can be attributed to reduced ovarian activity and irregular ovulation beginning in the mid-30s, which leads to decreased progesterone and DHEA secretion. Low DHEA levels may also reflect HPA axis dysfunction, as DHEA and cortisol are both products of adrenal steroidogenesis. Neurobiologically, DHEA exerts both direct and indirect effects on neurotransmission [22]. It can be converted into DHEA sulfate (DHEAS), estradiol, and testosterone, modulating dopaminergic pathways involved in sexual motivation and reward. DHEA also enhances dopamine release, and reduced DHEA or

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DHEAS concentrations can lead to diminished dopaminergic signaling and lower sexual arousal [23].

In the present study, decreased DHEA was most strongly associated with lower scores in the desire and arousal domains of the FSFI. This relationship can be explained by dopaminergic mechanisms, as dopamine pathways within the ventral tegmental area and nucleus accumbens are critical for sexual motivation and reward processing. Lower DHEA may lead to reduced dopamine activity, thereby diminishing sexual desire. Furthermore, DHEA conversion into androgens plays a key role in maintaining genital tissue function and sexual response. Androgens influence clitoral and vaginal physiology by regulating smooth muscle tone and promoting sexual desire [24]. Through conversion to testosterone, DHEA supports sexual interest by enhancing dopaminergic activity in the hypothalamus. Testosterone, in turn, contributes to vaginal trophic integrity, neurovascular relaxation, and nerve fiber density, which are essential for sexual stimulation. The interrelationship between DHEA, testosterone, and estradiol underscores the complex endocrine basis of female sexual function [25].

DHEA concentration in premenopausal women with sexual dysfunction was 0.82 ng/mL. Previous reports have shown variable DHEA levels, ranging from 0.42 ng/mL in premenopausal women to 57.83 ng/mL in women with sexual dysfunction. DHEA cut-off value of 2.85 ng/mL as a potential predictor of sexual desire disorders in women aged 27-43 years [26]. Although no specific reference value for premenopausal women has been established, the consistently lower DHEA levels observed among those with sexual dysfunction in this study suggest that DHEA may serve as a potential biomarker for sexual dysfunction in premenopausal women [27].

This study only measured cortisol and dehydroepiandrosterone levels at one point in time. This research was only conducted in Makassar with a small number of samples. Another limitation of this study is the measurement of sexual dysfunction using FSFI which is a subjective measurement. This study could not rule out specific stress factors by using questionnaires but based on anamnesis.

6. Conclusion

Cortisol and DHEA play important roles in sexual dysfunction. Elevated cortisol levels and decreased DHEA levels are associated with sexual dysfunction. These results suggest that cortisol and serum dehydroepiandrosterone levels may provide therapeutic support for the treatment of sexual dysfunction in premenopausal women

Acknowledgements

The authors would like to thank you to Department of Obstetrics and Gynecology, Faculty of Medicine, Hasanuddin University, and the staff of Wahidin Sudirohusodo Central General Hospital and Pepabri Health Center, Makassar, for their valuable assistance during data collection and laboratory analysis. Special thanks are also extended to all the premenopausal women who willingly participated in this study. This research received ethical approval from the Biomedical Research Ethics Committee, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia.

Conflict Of Interest

The authors declare no potential conflict of interest with respect to the research, authorship, and/or publication of this article.

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