

Physiological and metabolic alterations across menopausal stages in sample of Iraqi women

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Abstract:

Menopausal transition is associated with significant metabolic changes, particularly in lipid profiles, which can increase cardiovascular risk. Hormonal fluctuations during this period, including changes in estradiol (E2) and follicle-stimulating hormone (FSH), along with variations in body mass index (BMI), contribute to these alterations. In addition to conventional lipid parameters, the Atherosclerosis Index (AI) serves as an important predictor of cardiovascular risk by reflecting the balance between protective and atherogenic lipoproteins. However, the specific patterns and relationships among these factors in Iraqi women remain under-explored. This comparative physiological study aimed to assess the impact of menopausal transition on lipid profile parameters, including the Atherosclerosis Index (AI), and to examine their associations with estradiol (E2), follicle-stimulating hormone (FSH), and body mass index (BMI) in Iraqi women. A cross-sectional comparative study was conducted involving Iraqi women categorized into three groups: pre-menopausal (n=30), menopausal (n=30), and post-menopausal (n=30). Serum levels of reproductive hormones (FSH and E2), lipid profiles (total cholesterol, triglycerides, HDL, LDL, and VLDL), and BMI were measured using standardized laboratory protocols. Statistical analysis was performed using one-way ANOVA to compare the differences among the groups, with significance level at $p \leq 0.05$. Significant differences ($p \leq 0.05$ or $p \leq 0.01$) were observed among the three groups in most lipid and hormonal variables. Postmenopausal women demonstrated the highest levels of total cholesterol, triglycerides, LDL, and VLDL, along with the lowest HDL levels. These lipid alterations coincided with elevated BMI and FSH levels. Interestingly, estradiol levels were also found to be elevated in the postmenopausal group, contrary to the expected postmenopausal decline. This study highlights the complex interplay between menopausal status, hormonal changes, and lipid metabolism in Iraqi women. Elevated lipid levels and BMI in postmenopausal women suggest increased cardiovascular risk despite elevated estradiol levels, possibly due to peripheral aromatization. Regular monitoring of lipid profiles and BMI is recommended to manage potential cardiovascular risks in this population.

Keywords: Atherosclerosis Risk, BMI, Hormonal Shifts, Iraqi women, Lipid profile, Menopausal stages.

التحولات الفسيولوجية والتمثيلية الغذائية عبر مراحل سن اليأس في عينة من النساء العراقيات

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مستخلص:

يرتبط الانتقال إلى سن اليأس بتغيرات استقلابية (أيضية) كبيرة، وخصوصاً في دهون الدم، مما قد يزيد من خطر الإصابة بأمراض القلب والأوعية الدموية. تسهم التقلبات الهرمونية خلال هذه الفترة، بما في ذلك التغيرات في مستويات الإستراديول (E2) والهرمون المنشط للجريبات (FSH)، إلى جانب التغيرات في مؤشر كتلة الجسم (BMI)، في هذه التحولات الفسيولوجية. بالإضافة إلى معايير الدهون التقليدية، يُعد مؤشر تصلب الشرايين (AI) مؤشراً مهماً للتنبؤ بمخاطر أمراض القلب، حيث يعكس التوازن بين البروتينات الدهنية الواقية وتلك المسببة للتصلب. ومع ذلك، لا تزال الأنماط المحددة والعلاقات بين هذه العوامل لدى النساء العراقيات غير مدروسة بشكل كافٍ.

هدفت هذه الدراسة الفسيولوجية المقارنة إلى تقييم تأثير الانتقال إلى سن اليأس على معايير دهون الدم، بما في ذلك مؤشر تصلب الشرايين (AI)، وفحص علاقتها بالإستراديول (E2) والهرمون المنشط للجريبات (FSH) ومؤشر كتلة الجسم (BMI) لدى النساء العراقيات. أُجريت دراسة مقطعية مقارنة شملت نساء عراقيات قُسمن إلى ثلاث مجموعات: ما قبل انقطاع الطمث (n=30)، انقطاع الطمث (n=30)، وما بعد انقطاع الطمث (n=30). تم قياس مستويات الهرمونات التناسلية (FSH و E2)، ودهون الدم (الكوليسترول الكلي، الدهون الثلاثية، HDL، LDL، و VLDL)، و BMI باستخدام بروتوكولات مخبرية قياسية. أُجري التحليل الإحصائي باستخدام تحليل التباين الأحادي (ANOVA) لمقارنة الفروقات بين المجموعات، باعتبار القيمة الاحتمالية $p \leq 0.05$ دلالة إحصائية.

أظهرت النتائج وجود فروقات معنوية ($p \leq 0.05$ أو $p \leq 0.01$) بين المجموعات الثلاث في معظم المتغيرات الهرمونية والدهنية. أظهرت النساء بعد انقطاع الطمث أعلى مستويات للكوليسترول الكلي، والدهون الثلاثية، وLDL و VLDL، إلى جانب أدنى مستويات HDL. وتزامنت هذه التغيرات مع ارتفاع في BMI و FSH. ومن اللافت أن مستويات الإستراديول كانت مرتفعة في مجموعة ما بعد انقطاع الطمث، وهو ما يتعارض مع الانخفاض المتوقع بعد سن اليأس.

تُبرز هذه الدراسة التفاعل المعقد بين حالة المرأة الهرمونية وتغيرات الدهون في الجسم لدى النساء العراقيات. وتشير المستويات المرتفعة للدهون و BMI في النساء بعد انقطاع الطمث إلى زيادة خطر الإصابة بأمراض القلب، رغم ارتفاع مستويات الإستراديول، والذي قد يُعزى إلى التحول المحيطي لهرمون الأندروجين إلى الإستراديول. ويوصى بالحرص المنتظم لمستويات دهون الدم و BMI لإدارة المخاطر القلبية المحتملة لدى هذه الفئة من النساء.

الكلمات المفتاحية: مخاطر تصلب الشرايين، مؤشر كتلة الجسم، التغيرات الهرمونية، النساء العراقيات، ملف الدهون، مراحل سن اليأس.

1. Introduction:

Menopause is a natural biological process marking the end of female reproductive capacity, typically occurring in the mid-forties to early fifties and accompanied by hormonal changes most notably a decline in ovarian estrogen which trigger major physiological and metabolic shifts (World Health Organization, 2024).

Cardiovascular disease (CVD) risk increases significantly in women during their fifth decade of life, coinciding with the onset of menopause. This transition is closely linked to hormonal changes resulting from ovarian aging. Menopause is no longer seen merely as a reproductive milestone, but rather as a critical period marked by profound metabolic and cardiovascular changes (Uddenberg *et al.*, 2024).

Menopause is also a critical window for adverse metabolic changes, particularly dyslipidemia. Studies show rises in total cholesterol, LDL-C, triglycerides, and apolipoprotein B (apoB) levels, independent of age (Kodoth, *et al.*, 2022). These changes reflect a complex interaction between ag-

ing, hormonal shifts, and lifestyle.

Following menopause, reduced ovarian estrogen is partly offset by peripheral conversion of androgens to estrogens in adipose tissue via aromatase, especially in obese women, leading to mildly elevated estrogen levels (Janowska *et al.*, 2024). Concurrently, FSH levels rise up to 14-fold and are increasingly recognized for their non-reproductive roles (Spicer *et al.*, 2025).

Emerging evidence links elevated FSH to cardiovascular risk through mechanisms like endothelial inflammation, lipid disturbances, and fat accumulation (Kim *et al.*, 2024; Wang *et al.*, 2024)). FSH receptors are present in adipose and other non-reproductive tissues, where high FSH levels correlate with obesity, dyslipidemia, CVD, Alzheimer's, and cancer risk (Li *et al.*, 2024).

Vascular dysfunction, including reduced endothelial function and arterial stiffness, starts around ages 47–48 and correlates partly with rising FSH, while the direct effect of estrogen remains less clear (Wenner *et al.*, 2024).

Thus, FSH plays a central role in metabolic alterations during and after menopause, acting beyond its classical role in reproduction by affecting fat distribution, lipid metabolism, and cardiovascular risk (Kim *et al.*, 2024).

Additionally, ethnicity, geography, and lifestyle influence menopausal symptoms and metabolic outcomes. Studying Iraqi women offers insights into these effects within a culturally and genetically distinct context (Skibiak *et al.*, 2025). Factors like sex, ethnicity, and socioeconomic status also shape fat distribution particularly subcutaneous, visceral, and cardiac fat which is crucial to cardiovascular health (Assheuer, 2025).

Aim of study:

The study aimed to study the relation of atherogenesis rate with hormonal changes within premenopause and postmenopause stages.

2. Materials and Methods:

2.1 Study Design and Participants:

This cross-sectional comparative study was conducted at the Department of Biology, College of Science, University of Baghdad, between No-

vember 1, 2024, and January 15, 2025. Ethical approval was granted by the Ethics Committee of the Department of Biology, College of Science, University of Baghdad (Reference No.CSEC/0625/0074).

A total of 90 Iraqi women voluntarily participated in the study and were stratified into three equal groups (n = 30 each) based on menopausal status:

- Premenopausal group (control group): women aged 25–30 years with regular menstrual cycles.
- Menopausal transition group: women aged 45–50 years with noticeable changes in menstrual cycle regularity
- Postmenopausal group: women aged 50–55 years with no menstruation for 12 consecutive months, without any pathological cause.

All participants provided written informed consent before enrollment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

2.2 Inclusion and Exclusion Criteria:

The inclusion criteria comprised Iraqi women aged between 25 and 55

years, with intact uterus and ovaries, and in generally good health. All participants were required to be free of chronic medical conditions.

Exclusion criteria included a history of hypertension, diabetes mellitus, osteoporosis, polycystic ovary syndrome (PCOS), or thyroid disorders. Women were also excluded if they were pregnant, infertile, or had undergone oophorectomy or thyroidectomy.

2.3 Data Collection:

Comprehensive medical and demographic information was obtained using a structured questionnaire. Collected data included age, marital status (married or unmarried), number of children, and menstrual history, which was used to classify participants into premenopausal (25–30), menopausal transition(45–50), and postmenopausal(50–55) groups. Physical examination involved direct measurement of height and weight to calculate body mass index (BMI).

2.4 Laboratory Analyses:

Lipid profile(mg/dl), FSH(IU/L) and E2(pg/ml) was measured by using serum(10µL). The complete lipid profile, FSH, E2, and BMI were measured.

Hormonal assays were conducted using the sandwich ELISA technique for FSH and E2 (Sunlongbiotech, China). Additionally, lipid profile analyses were performed using a semi-automatic analyzer (Semi-Auto Spectrophotometer) For total cholesterol (Ref/CS005 2100, Bio Research, Jordan), and for Triglycerides (Ref/10164, SGM, Italy) while for HDL (Ref/CS009 1100, Bio Research, Jordan). LDL and VLDL concentrations were calculated using the Friedewald formula, while the Atherosclerosis Index (AI) was calculated as the ratio of LDL to HDL.

2.5 Quality Control and Statistical Analysis:

Statistical analysis was performed using SPSS software (version2019). Data normality was assessed using the Shapiro-Wilk test. Results were presented as means ± standard error (SE). Differences among the three groups were evaluated using one-way analysis of variance (ANOVA), followed by Least Significant Difference (LSD) post hoc test to identify specific group differences. Statistical significance was set at $p < 0.05$, with highly significant values reported at $p < 0.01$.

3. Results:

3.1 Body Mass Index (BMI) Analysis

Significant differences were observed in body mass index (BMI) among the study groups (Table 1). Higher increase in menopause and postmenopause group BMI values compared to the premenopausal con-

trol group (24.74 ± 3.29 , $p \leq 0.01$). This suggests an increase in adiposity concurrent with menopausal transition and postmenopausal status, potentially contributing to metabolic and cardiovascular risks, In addition the mean age show higher increase in postmenopause group compared to the menopause and premenopause groups with ($p \leq 0.01$).

Table 1: Comparison between different groups in Body mass index and Ages

Groups	Means \pm SE (kg/m ²)	Means \pm SE (yrs)
	BMI (kg/m ²)	Age (years)
Pre-menopause (Control)	24.74 \pm 3.29 a	28.6 \pm 1.04 a
Menopause	30.81 \pm 5.87 b	46.0 \pm 0.74 b
Post-menopause	32.29 \pm 6.13 b	53.5 \pm 1.21 c
L.S.D.	2.70**	2.0**
P-value	0.0001	0.0001
Means having with the different letters in the same column differed significantly. ** (P \leq 0.01).		

3.2 Hormonal Profile Analysis

Analysis of hormonal levels revealed significant differences across menopausal stages (Table 2). Follicle-stimulating hormone (FSH) levels were significantly elevated in postmenopausal women (1.715 ± 0.05 pg/ml) compared to both menopausal (0.979 ± 0.11 pg/ml) and premenopausal groups (1.438

± 0.05 pg/ml), with a highly significant difference ($p \leq 0.01$). These findings highlight the hormonal shift associated with ovarian senescence. As for estradiol (E2), postmenopausal women also showed the highest levels (14.68 ± 0.57 pg/ml) compared to menopausal (10.96 ± 1.97 pg/ml), with the observed difference reaching statistical signifi-

cance ($p \leq 0.05$). This elevation in E2 during the postmenopausal phase may reflect peripheral estrogen production rather than ovarian activity.

Table 2: Comparison between different groups in Hormones level

Groups	Means \pm SE (IU/L)	Means \pm SE (pg/ml)
	FSH (IU/L)	E2 (pg/ml)
Pre-menopause (Control)	0.958 \pm 0.05 a	12.01 \pm 0.43 ab
Menopause	0.652 \pm 0.11 b	10.96 \pm 1.97 a
Post-menopause	1.143 \pm 0.05 c	14.68 \pm 0.57 b
L.S.D.	0.216 **	3.406 *
P-value	0.0001	0.0498
Means having with the different letters in same column differed significantly. * ($P \leq 0.05$), ** ($P \leq 0.01$).		

3.3 Lipid Profile Analysis

Comprehensive analysis of lipid parameters revealed significant variations across the menopausal stages (Table 3). Postmenopausal women exhibited the most adverse lipid profile, with markedly elevated concentration of total cholesterol (294.13 ± 14.38 mg/dl) compared to both premenopausal (240.30 ± 9.40 mg/dl) and menopausal women (258.93 ± 9.56 mg/dl) ($p \leq 0.05$; $LSD = 31.923^{**}$).

Similarly, triglyceride concentration increased significantly with menopausal progression, reaching the highest

in postmenopausal women (254.83 ± 18.86 mg/dl) compared to premenopausal groups (162.23 ± 10.38 mg/dl) ($p \leq 0.01$; $LSD = 40.356^{**}$).

High-density lipoprotein (HDL) concentration declined with age and menopausal status, being significantly lower in postmenopausal women (42.80 ± 1.97 mg/dl) than in premenopausal counterparts (48.40 ± 1.95 mg/dl), while menopausal women showed non significant differences (45.23 ± 2.01 mg/dl) ($p \leq 0.05$; $LSD = 5.554^*$).

Low-density lipoprotein (LDL) concentrations show significant differ-

ence in premenopause (159.45 ± 9.61 mg/dl) compared to postmenopause (200.36 ± 14.63 mg/dl) at ($p \leq 0.05$; $LSD = 32.471^*$).

Very-low-density lipoprotein (VLDL) concentration were significantly higher in both menopausal (43.19 ± 2.48 mg/dl) and postmenopausal women (50.97 ± 3.77 mg/dl) compared to premenopausal women (32.44 ± 2.07 mg/dl) ($p \leq 0.01$; $LSD = 8.071^{**}$).

Atherosclerosis Index (AI) values

were progressively elevated across the groups, with postmenopausal women showing the highest mean (5.51 ± 0.71 mg/dl) compared to premenopausal women (3.77 ± 0.45 mg/dl), indicating a significant difference ($LSD = 1.57$; $\Delta = 1.74$). However, no significant differences were observed between premenopausal and menopausal (4.34 ± 0.48 mg/dl), or between menopausal and postmenopausal women, despite the rising trend ($p > 0.05$; $LSD = 1.57$ NS).

Table 3: Comparison between difference groups in Lipid profile

Groups	Means \pm SE (mg/dl)					
	Cholesterol (mg/dl)	Triglyceride (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)	AI
Pre-menopause (Control)	240.30 ± 9.40 a	162.23 ± 10.38 a	48.40 ± 1.95 a	159.45 ± 9.61 a	32.44 ± 2.07 a	3.77 ± 0.45 a
Menopause	258.93 ± 9.56 a	215.93 ± 12.44 b	45.23 ± 2.01 ab	170.91 ± 9.67 ab	43.19 ± 2.48 b	4.34 ± 0.45 a
Post-menopause	294.13 ± 14.38 b	254.83 ± 18.86 b	42.80 ± 1.97 b	200.36 ± 14.63 b	50.97 ± 3.77 b	5.51 ± 0.71 a
L.S.D.	31.923 **	40.356 **	5.554 *	32.471 *	8.071 **	1.57 NS
P-value	0.0043	0.0001	0.0488	0.0401	0.0001	0.087
Means having with the different letters in same column differed significantly. * ($P \leq 0.05$), ** ($P \leq 0.01$). NS: Not Significant (no significant difference)						

4. Discussion:

The present study revealed a statistically significant increase in E2 levels in the postmenopausal group compared to the menopausal group ($P \leq 0.05$). This unexpected elevation in E2 is unlikely to originate from ovarian activity but is more plausibly attributed to the peripheral aromatization of androgens into estrogens via the aromatase enzyme, whose activity is known to be heightened in adipose tissue. The higher body mass index (BMI) observed among postmenopausal participants may have facilitated this enhanced peripheral conversion, thereby contributing to increased circulating estrogen levels (Fux-Otta *et al.*, 2025; Üstün, 2025).

An increased E2 levels may related to decrease in the level of sex hormone-binding globulin (SHBG), SHBG is also considered a contributing factor, as this protein plays a crucial role in transporting and binding sex hormones in the bloodstream. When SHBG levels decline, the amount of free, biologically active estrogen increases, which in turn enhances its physiological effects in the body (Ichikawa *et al.*, 2025; Szy-

biak-Skora *et al.*, 2025).

In this study, a clear increase in FSH levels was found in all three groups after menopause ($P \leq 0.01$), which agrees with the natural physiological changes that usually start a few years before the last menstrual cycle (Spicer *et al.*, 2025). This rise in FSH is mainly due to the reduced function of the ovaries, as the drop in estrogen production weakens the negative feedback to the pituitary gland. As a result, the pituitary releases more FSH to compensate (Mou *et al.*, 2025).

Moreover, advancing age is also associated with a decline in the liver's metabolic efficiency due to decreased activity of cytochrome P450 (CYP) enzymes. This reduction can lead to a buildup of different forms of estrogen in the bloodstream, as they are not properly metabolized into their active or inactive forms. As a result, higher levels of circulating estradiol may not deliver sufficient negative feedback to the hypothalamic-pituitary axis, which helps explain why FSH levels remain elevated even when estrogen appears to be adequate (Konstandi and Johnson, 2023).

Furthermore, this hormonal change is also linked to an increase in subcutaneous fat and disruptions in glucose metabolism, which together raise the risk of developing cardiovascular and metabolic diseases (Saei Ghare Naz *et al.*, 2024).

One key hormonal factor linked to weight gain after menopause is follicle-stimulating hormone (FSH). Higher levels of FSH have been found to directly affect fat metabolism by stimulating the formation of fat cells, especially in visceral fat tissue. This process contributes to central obesity and an increase in BMI (Korkmaz *et al.*, 2025).

In addition to hormonal factors, menopause is associated with significant changes in body composition, including decreased muscle mass and increased visceral fat. These changes lead to a reduction in resting metabolic rate (RMR), contributing further to increased BMI. Aging and hormonal decline both play roles in this metabolic slowdown, and interventions such as hormone replacement therapy and physical exercise have been suggested to mitigate these effects (Juppi *et al.*,

2025).

The concerning lipid profile findings highlight a consistent pattern of dyslipidemia that emerges during the menopausal transition, with the most pronounced changes observed in postmenopausal women. This shift in lipid profile calls for a deeper physiological understanding to elucidate the underlying mechanisms linking menopause to the increased risk of cardiovascular disease.

Following menopause, significant hormonal shifts occur that affect lipid metabolism and increase the risk for cardiovascular disease (CVD). Although E2 levels may be elevated postmenopause, this estrogen is often non-ovarian in origin and has reduced bioactivity. As a result, its regulatory influence on lipid metabolism, particularly via estrogen receptors such as ER α in adipose tissue, becomes impaired. This receptor insensitivity or down regulation disrupts normal adipocyte function by diminishing lipid oxidation, enhancing lipogenesis, and promoting pro-inflammatory cytokine production (Sánchez-García *et al.*, 2025). Consequently, visceral fat accu-

mulation increases, leading to heightened risk of metabolic syndrome, CVD, and obesity-related complications.

Despite the concurrent rise in BMI, FSH, and E2 levels after menopause, the key contributor to metabolic dysfunction remains the accumulation of visceral adipose tissue (VAT) rather than subcutaneous adipose tissue (SAT) (Gallardo and Andrés, 2025). VAT accumulation is closely associated with insulin resistance, dyslipidemia, and increased risk of type 2 diabetes. These metabolic alterations are further influenced by declining regulatory roles of estrogen and androgens postmenopause.

The impaired ability of E2 to regulate fat distribution and blood pressure contributes to the development of metabolism-related cardiac conditions, including heart failure with preserved ejection fraction (HFpEF) (Du *et al.*, 2025; Kuznetsova *et al.*, 2025). Under normal physiological conditions, estrogen favors gynoid fat distribution. Postmenopausal loss of this effect results in a shift toward android (visceral) fat deposition, intensifying metabolic disturbances and elevating the

risk of CVD and diabetes (Colleluori *et al.*, 2020).

Estrogen receptors ESR1 and ESR2 play central roles in lipid and glucose metabolism. ESR1 promotes adipocyte glucose uptake and lipolysis, while ESR2 has a more complex function, with studies showing links to both insulin resistance and metabolic protection (Ahmed *et al.*, 2025). Notably, elevated E2 in late postmenopausal women was found to inhibit glucose uptake in SAT, partially mediated by ESR2, suggesting receptor-mediated resistance as a mechanism underlying persistent visceral adiposity and dyslipidemia.

FSH has also been linked to dyslipidemia and obesity. Elevated FSH levels postmenopause contribute to VAT accumulation and cholesterol profile disturbances. While some studies report inconsistent associations between FSH and type 2 diabetes, evidence suggests that FSH plays a role in glucose and lipid regulation, influencing the development of metabolic dysfunctions (Xu *et al.*, 2022). The study results indicate that elevated levels of FSH and E2, along with increased body mass index

and lipid profile disturbances, reflect a hormonal and metabolic imbalance that enhances the risk of chronic diseases in postmenopausal women. This underscores the importance of monitoring these markers to assess overall health and to guide prevention and treatment strategies (Ghudhaib *et al.*, 2014).

5. Conclusion:

This study provided a comprehensive assessment of both endocrine and metabolic changes during the menstrual stages in Iraqi women and focused on BMI, FSH, E2, lipid profile and atherosclerosis index. The results showed marked gradual deterioration in lipid profile with age. The most important are increase LDL, VLDL, triglycerides, cholesterol and decrease HDL levels.

The study highlighted a significant positive correlation between high FSH and BMI, along with the deterioration of the lipid profile to the risk of critical nutritional representation in menopause.

Given the cross-sectional nature of the study and a relatively small sample size, these findings emphasize the need for future longitudinal research

involving larger, more homogeneous cohorts. The results underscore the urgent need for targeted lifestyle interventions including dietary modifications, increased physical activity, and routine hormonal and lipid monitoring to mitigate cardiovascular risks in menopausal Iraqi women. Overall, this work establishes essential baseline data to inform culturally appropriate preventive health strategies tailored to this population.

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References:

- Ahmed, F., Hetty, S., Laterveer, R., Surucu, E.B., Mathioudaki, A., Hornbrinck, E., et al. (2025) 'Altered expression of aromatase and estrogen receptors in adipose tissue from men with obesity or type 2 diabetes', *Journal of Clinical Endocrinology & Metabolism*, dgaf038. doi:10.1210/clinem/dgaf038
- Assheuer, J.A. (2025) The assessment of adipose tissue distribution among patients with ischemic heart disease from different social and demographic groups [Master's thesis]. Vilnius: Vilniaus universitetas.
- Colleluori, G., Chen, R., Turin, C.G., Zhou, J., Coughlin, J.W., Muniyappa, R. and Ferrando, A.A. (2020) 'Aromatase inhibitors plus weight loss improve the hormonal profile of obese hypogonadal men without causing major side effects', *Frontiers in Endocrinology (Lausanne)*, 11, p.277. doi:10.3389/fendo.2020.00277
- Du, J., Liu, J., Wang, X., Wang, X., Ma, Y., Zhang, S., et al. (2025) 'Difference for the risk factors of heart failure with preserved ejection fraction', *Biology Direct*, 20(1), p.28. doi:10.1186/s13062-025-00618-x
- Fux-Otta, C., Torre, D., Chedraui, P., Melgarejo, B., Ramos, N., Di Carlo, M., et al. (2025) 'Hyperandrogenism after menopause: diagnostic evaluation', *Climacteric*, 28(1), pp.61–68. doi:10.1080/13697137.2024.2423874
- Gallardo, N. and Andrés, A. (2025) 'Effect of Aromatase and ESR1 Expression in SAT on Insulin Resistance and T2D in Obese Men', *Journal of Clinical Endocrinology & Metabolism*, dgaf167. doi:10.1210/clinem/dgaf167
- Ghudhaib, K.K., Turaki, K.M. and Muzal, S.A. (2014) 'Estimation of serum osteocalcin levels in osteoporotic postmenopausal Iraqi women with type 2 diabetes mellitus', *Baghdad Science Journal*, 11(4), pp.1549–1555. doi:10.21123/bsj.2014.11.4.1549-1555
- Ichikawa, T., Okada, H., Hamaguchi, M., Hara, M., Takashima, N., Suzuki, S., et al. (2025) 'Association of sex hormone-binding globulin and dyslipidemia with Japanese postmenopausal women: A cross-sectional study', *Lipids in Health and Disease*, 24(1), pp.1–8. doi:10.1186/s12944-025-02634-2
- Janowska, S., Holota, S., Lesyk, R.

and Wujec, M. (2024) 'Aromatase inhibitors as a promising direction for the search for new anticancer drugs', *Molecules*, 29(2), p.346. doi:10.3390/molecules29020346

Juppi, H-K., Karppinen, J.E. and Laakkonen, E.K. (2025) 'Menopause and body composition: A complex field', *Seminars in Reproductive Medicine*. (In press). doi:10.1055/s-0045-1809531

Kim, S-M., Sultana, F., Sims, S., Gimenez-Roig, J., Laurencin, V., Pallapati, A., et al. (2024) 'FSH, bone, belly and brain', *Journal of Endocrinology*, 262(1). doi:10.1530/JOE-23-0377

Konstandi, M. and Johnson, E.O. (2023) 'Age-related modifications in CYP-dependent drug metabolism: Role of stress', *Frontiers in Endocrinology (Lausanne)*, 14, p.1143835. doi:10.3389/fendo.2023.1143835

Korkmaz, F., Gimenez-Roig, J., Sultana, F., Laurencin, V., Sen, F., Cullen, L., et al. (2025) 'Targeting FSH for osteoporosis, obesity, and Alzheimer's disease', *Trends in Molecular Medicine*. (In press). doi:10.1016/j.molmed.2025.05.001

Kodoth, V., Scaccia, S. and Aggarwal, B. (2022) 'Adverse changes in body composition during the menopausal transition and relation to cardiovascular risk: A contemporary review', *Women's Health Reports (New Rochelle)*, 3(1), pp.573–581. doi:10.1089/whr.2021.0119

Kuznetsova, E.A., Fedorov, N.S., Zakyrjanova, G.F., Malomouzh, A.I. and Petrov, A.M. (2025) '25-Hydroxycholesterol as a negative regulator of diaphragm muscle contractions via estrogen receptor and Ca²⁺-dependent pathway', *Histochemistry and Cell Biology*, 163(1), pp.1–15. doi:10.1007/s00418-025-02370-9

Li, C., Ling, Y. and Kuang, H. (2024) 'Research progress on FSH-FSHR signaling in the pathogenesis of non-reproductive diseases', *Frontiers in Cell and Developmental Biology*, 12, p.1506450. doi:10.3389/fcell.2024.1506450

Mou, H., Zhang, J., Guo, Y., Xu, L. and Luo, X. (2025) 'Effects of key physiological parameters on cardiovascular disease and osteoporosis risk in perimenopausal and postmenopausal women', *Scientific Reports*, 15(1),

p.2814. doi:10.1038/s41598-025-86613-8

Saei Ghare Naz, M., Farhadi-Azar, M., Noroozzadeh, M., Farahmand, M. and Ramezani Tehrani, F. (2024) 'Follicle-stimulating hormone and diabetes in postmenopausal women: A systematic review and meta-analysis', *Journal of Clinical Endocrinology & Metabolism*, 109(8), pp.2149–2160. doi:10.1210/clinem/dgae198

Sánchez-García, M., León-Wu, K., de Miguel-Ibáñez, R., López-Juárez, N., Ramírez-Rentería, C., Espinosa-Cárdenas, E., et al. (2025) 'Metabolic changes in patients with premature ovarian insufficiency: Adipose tissue focus—A narrative review', *Metabolites*, 15(4), p.242. doi:10.3390/metabo15040242

Skibiak, K., Dębski, J., Przybyłowski, J., Walędziak, M. and Różańska-Walędziak, A. (2025) 'The influence of menopausal status on sleep quality in different populations – a narrative review', *Menopause Review/Przegląd Menopauzalny*, 24(1), pp.53–65. doi:10.5114/pm.2025.150450

Spicer, J., Malaspina, D., Blank, S.V. and Goosens, K.A. (2025) 'Fol-

licle-stimulating hormone: More than a marker for menopause: FSH as a frontier for women's mental health', *Psychiatry Research*, 345, p.116239. doi:10.1016/j.psychres.2024.116239

Szybiak-Skora, W., Cyna, W. and Lacka, K. (2025) 'New insights in the diagnostic potential of sex hormone-binding globulin (SHBG)—Clinical approach', *Biomedicines*, 13(5), p.1207. doi:10.3390/biomedicines13051207

Üstün, B. (2025) 'The role of androgens in health and disease in females: A narrative review', *Anatolian Journal of Obstetrics & Gynecology Research*. (In press). doi:10.4274/ana-jog.galenos.2025.58066

Uddenberg, E.R., Safwan, N., Saadeldine, M., Hurtado, M.D., Faubion, S.S. and Shufelt, C.L. (2024) 'Menopause transition and cardiovascular disease risk', *Maturitas*, 185, p.107974. doi:10.1016/j.maturitas.2024.107974

Wang, Q., Han, J., Liang, Z., Geng, X., Du, Y. and Zhou, J., et al. (2024) 'FSH is responsible for androgen deprivation therapy-associated atherosclerosis in mice by exaggerating endothelial inflammation and monocyte

adhesion', *Arteriosclerosis, Thrombosis, and Vascular Biology*, 44(3), pp.698–719. doi:10.1161/ATVBAHA.123.319426

Wenner, M.M., Shenouda, N., Shoemaker, L., Kuczmarski, A., Haigh, K. and Del Vecchio, A., et al. (2024) 'Characterizing vascular and hormonal changes in women across the life span: A cross-sectional analysis', *American Journal of Physiology-Heart and Circulatory Physiology*, 327(5), pp.H1286–H1295. doi:10.1152/ajpheart.00373.2024

World Health Organization (2024) Menopause [Internet]. Geneva: WHO. Available at: <https://www.who.int/news/item/28-11-2024-menopause> (Accessed: 28 June 2025).

Xu, Z., Gu, S., Wu, X., Zhou, Y., Li, H. and Tang, X. (2022) 'Association of follicle stimulating hormone and serum lipid profiles in postmenopausal women', *Medicine (Baltimore)*, 101(39), p.e30920. doi:10.1097/MD.00000000000030920

