

Selección de Resúmenes de Menopausia

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Impact of high body fat and low muscle mass on bone mineral density in postmenopausal women with type 2 diabetes: A DXA-based cross-sectional study

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Background and objective: Sarcopenic obesity, defined as the coexistence of reduced muscle mass and excess adiposity, may adversely affect bone health, especially in postmenopausal women with type 2 diabetes mellitus (T2DM). This study aimed to explore the association between different body composition phenotypes, particularly sarcopenic obesity, and bone mineral density (BMD) in this population. Methods: We retrospectively analyzed 573 postmenopausal women with T2DM hospitalized between September 2020 and October 2024. Based on appendicular skeletal muscle mass index (ASMI) and body fat percentage (BFP) measured by dual-energy X-ray absorptiometry (DXA), participants were classified into control, obesity alone, sarcopenia alone, and sarcopenic obesity groups. Associations between phenotypes and BMD were assessed using Spearman correlation, logistic regression, and restricted cubic spline (RCS) models. Results: The obesity group had the highest BMD, while the sarcopenia group had the lowest. Sarcopenic obesity was associated with significantly lower lumbar spine and hip BMD and a higher risk of osteoporosis. Obesity showed a protective effect (OR = 0.515, P = 0.002), whereas sarcopenic obesity increased osteoporosis risk (OR = 3.368, P = 0.007). RCS analysis revealed nonlinear relationships between ASMI, BFP, and BMD. Conclusion: Sarcopenic obesity significantly increases osteoporosis risk in postmenopausal women with T2DM, while isolated obesity may protect bone mass. Routine body composition assessment is essential for early identification and targeted intervention.

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The hypothalamic-pituitary-ovarian axis, ovarian disorders and brain aging: a review

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The hypothalamic-pituitary-ovarian (HPO) axis is a complex endocrine feedback mechanism controlling ovulation in female vertebrates. Balance of the HPO axis requires correct secretion of sex steroids from the ovarian follicle to inhibit release of gonadotropins from the pituitary. Several conditions of ovarian dysfunction such as menopause, Primary Ovarian Insufficiency (POI) and Polycystic Ovary Syndrome (PCOS) involve imbalances in the HPO axis, contributing to infertility. Intriguingly, these disorders also share a higher incidence of cognitive and emotional dysregulations, as well as a heightened risk of certain neurodegenerative conditions with age. It is understood that estradiol exerts neuroprotective functions, but gonadotropin signaling is less understood. High concentrations of circulating Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH) have shown to contribute to neurodegenerative disease states, but are not addressed as part of traditional Hormone Replacement Therapy (HRT). To identify the mechanistic connections between ovarian disorders and heightened susceptibility of the brain to pathological aging, a multi-system experimental approach is required, considering each HPO axis player as an individual effector. In this review, we will summarize current knowledge on the effects of estradiol, progesterone, FSH and LH on neuronal susceptibility to pathology. We will describe ways in which the HPO axis becomes imbalanced during ovarian dysfunction, and how systemic inflammation can become an additional HPO axis effector. Finally, we will recommend solutions to the presented gaps in knowledge, and suggest avenues of future research to pursue development of therapeutics targeting both ovarian and brain health in patients.

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Association between modified mediterranean diet score and menopause-specific quality of life and symptoms: a cross-sectional study

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In recent times, research has delved into the impact of dietary elements on alleviating menopausal symptoms. We endeavored to examine the correlation between the modified Mediterranean diet score (mMDS) and quality of life, menopausal symptoms, discriminative ability and determination of the optimal cut-off point. A total of one hundred forty-nine postmenopausal females were selected to participate in the cross-sectional study from June 2021 to June 2022. Upon completion of data collection through interviews, the relevant variables were calculated. The assessment of participants' food consumption in the preceding year was conducted using a 117-item food frequency questionnaire. postmenopausal women who were referred to healthcare clinics in Lar. The participants were exclusively women aged 40 and above who had not undergone a menstrual cycle for a minimum of 12 months. We observed that mMDS associated with the severity of sexual and vasomotor symptoms. The third tertile of mMDS had a significantly lower odds ratio for severe to moderate sexual (OR: 0.17; 95% CI: 0.068, 0.450) and vasomotor (OR: 0.20; 95% CI: 0.064, 0.658) symptoms compared to the first tertile. Also, mMDS showed significant discriminative ability in identifying women with severe to moderate poor quality of life (AUC = 0.997, 95% CI = 0.988-1.000; p-value = 0.001), vasomotor (AUC = 0.727, p-value < 0.001), psychological (AUC = 0.867, p-value < 0.001), physical (AUC = 0.934, p-value < 0.001) and sexual symptoms (AUC = 0.753; p-value < 0.001). There is promising yet incongruous data indicating the potential of the Mediterranean diet in the management of vasomotor symptoms in menopausal women. Further comprehensive, extended research is essential for the development of precise dietary recommendations.

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The Comparison of Insulin Resistance Between Normal and Early Menopause Women Younger than Fifty Years Old by Machine Learning Methods

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Background: The prevalence of type 2 diabetes (T2D) is on the rise, and insulin resistance (IR) is one of the key risk factors for developing T2D. This paper seeks to identify risk factors for IR in women with normal menstrual cycles (NM) and early menopausal women (EM). Methods: EM women between 30 and 50 years old were compared with an NM control group. Four machine learning (ML) methods were trained using comprehensive physiological and lifestyle data to estimate a homeostasis model for insulin resistance (HOMA-IR dependent variable). Traditional multiple linear regression (MLR) was used as a benchmark for comparison. Results: A total of 948 participants were enrolled (NM: 410, EM: 538). On average, ML outperformed MLR, identifying the six key risk factors in the EM group (from most to least important) as waist-hip ratio (WHR), triglyceride (TG), glutamic-pyruvic transaminase (GPT), glutamic oxaloacetic transaminase (GOT), HDL-Cholesterol (HDL-C), and lactic dehydrogenase (LDH). Rankings differed in the NM group, with WHR identified as the leading risk factor, followed by C-reactive protein (CRP), HDL-C, total bilirubin (TBIL), diastolic blood pressure (DBP), and white blood cell count (WBC). Conclusions: Using ML, we found that WHR and HDL-C are the common denominators in both EM and NM women, with additional correlations with TG, liver enzymes and LDH for EM women. These results clearly indicate the importance of estrogen protection, suppressing less important factors (TG, liver enzyme, and LDH), and only the stronger inflammatory markers become important (CRP, TBIL, and WBC). Once estrogen's protection disappears, the suppression of CRP, TBIL, and WBC would become weaker. Since these 3 features are significantly correlated with body weight, for women under 50, reducing body weight is the most important factor in preventing hyperglycemia.

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The Final Phases of Ovarian Aging: A Tale of Diverging Functional Trajectories

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Ovarian aging is characterized by a gradual decline in both reproductive and endocrine functions, ultimately culminating in the cessation of ovarian activity around the age of 50, when most women experience natural menopause. The decline begins early, as follicular attrition is initiated in utero and continues throughout childhood and reproductive life. Most follicles undergo atresia without progressing through substantial stages of growth. With increasing age, a pronounced reduction occurs in the population of resting follicles within the ovarian reserve, accompanied by a decline

in the size of growing follicular cohorts. Around the age of 38, the rate of follicular depletion accelerates, sometimes resulting in diminished ovarian reserve (DOR). The subsequent menopausal transition involves complex, irregular hormonal dynamics, manifesting as increasingly erratic menstrual patterns, primarily driven by fluctuations in circulating estrogens and a rising incidence of anovulatory cycles. In parallel with the progressive depletion of the follicular pool, the serum concentrations of anti-Müllerian hormone (AMH) decline gradually, while reductions in inhibin B levels become more apparent during the late reproductive years. The concomitant decline in both inhibin B and estrogen levels leads to a compensatory rise in circulating follicle-stimulating hormone (FSH) concentrations. Together, these endocrine changes, alongside the eventual exhaustion of the follicular reserve, converge in the onset of menopause, which is defined by the absence of menstruation for twelve consecutive months. The mechanisms contributing to ovarian aging are complex and multifactorial, involving both the oocyte and the somatic cells within the follicular microenvironment. Oxidative stress is thought to play a central role in the age-related decline in oocyte quality, primarily through its harmful effects on mitochondrial DNA integrity and broader aspects of cellular function. Although granulosa cells appear to be relatively more resilient, they are not exempt from age-associated damage, which may impair their hormonal activity and, given their close functional relationship with the oocyte, negatively influence oocyte competence. In addition, histological changes in the ovarian stroma, such as fibrosis and heightened inflammatory responses, are believed to further contribute to the progressive deterioration of ovarian function. A deeper understanding of the biological processes driving ovarian aging has facilitated the development of experimental interventions aimed at extending ovarian functionality. Among these are the autologous transfer of mitochondria and stem cell-based therapies, including the use of exosome-producing cells. Additional approaches involve targeting longevity pathways, such as those modulated by caloric restriction, or employing pharmacological agents with geroprotective properties. While these strategies are supported by compelling experimental data, robust clinical evidence in humans remains limited.

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Menopause and risk of atherosclerotic cardiovascular disease: insights from a women's UK Biobank cohort

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Background: Menopause induces hormonal and metabolic changes that may increase the risk of atherosclerotic cardiovascular disease (ASCVD). However, the contribution of menopause to ASCVD risk, beyond aging, remains debated. This study investigated the association between menopause and ASCVD risk in a large population-based cohort. **Methods:** We analyzed data from 222,007 women in the UK Biobank, excluding those with prior cardiovascular disease or uncertain menopausal status. ASCVD risk was estimated using pooled cohort equations. High ASCVD risk was defined as a 10-year risk $\geq 7.5\%$. To address missing data, multiple imputations for the Area Deprivation Index (ADI) were performed using a fully conditional specification approach. Multivariable regression models were used to assess the association between menopause and ASCVD risk, adjusting for age as well as socioeconomic, lifestyle, metabolic, and clinical factors. **Results:** Postmenopausal women ($n = 158,572$) had significantly higher estimated ASCVD risk than premenopausal women ($n = 63,435$) (3.75% vs. 0.81% ; $p < 0.001$). The prevalence of high ASCVD risk was 9.65% in postmenopausal women versus 0.41% in premenopausal women ($p < 0.001$). After full adjustment, menopause was independently associated with a higher ASCVD risk ($\beta = 0.56\%$; 95% CI $0.54-0.58$) and an 18% increased likelihood of high ASCVD risk ($OR = 1.18$; 95% CI $1.01-1.37$). The association was strongest among women aged under 60 years, with no significant association observed beyond 60 years. **Conclusion:** Menopause is independently associated with increased ASCVD risk, particularly in women aged under 60 years. These findings highlight the importance of considering menopausal status in cardiovascular risk assessment and implementing targeted prevention strategies in midlife women.

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High-density lipoprotein cardio-protection function deteriorates as women transition through menopause: The SWAN HDL Study

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Background: Women show a rise in HDL cholesterol efflux capacity (CEC) as they traverse menopause. Whether this rise is associated with a lower risk of cardiovascular disease (CVD) overtime is not clear. **Objectives:** We tested

whether CEC association with subclinical vascular health, measured using a composite subclinical vascular health score based on levels of carotid-intima media thickness (cIMT), carotid-femoral pulse wave velocity (cfPWV), and presence of coronary artery calcium (CAC score >10), varies by time relative to the final menstrual period (FMP). Methods: 279 women (baseline age 51 ± 2.8 years; 68.5% White) who had CEC and outcome measures were included. The subclinical vascular health measures were related to CEC through a Bayesian hierarchical linear mixed effects model using the latent composite measure as the outcome, and time relative to FMP, CEC, and their interaction as explanatory variables. Differences by racial subgroups were explored. Results: Higher CEC was associated with a lower composite subclinical measure of vascular health at the time of the FMP. In both unadjusted and adjusted models, the inferred interaction effect (posterior probability >0.99) implies that the pre-FMP protective association of CEC diminishes after FMP. This was consistent across all components of the composite score. In Black women, the protective association of CEC diminished more rapidly compared to White women (posterior probability >0.90). Conclusions: In women, higher CEC is associated with a lower risk of subclinical vascular health only before menopause. Higher CEC is not a consistent indicator of greater CVD protection in women traversing menopause.