Selección de Resúmenes de Menopausia

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Is Vaginal Estrogen Treatment Associated with Risk of Thrombosis in Postmenopausal Women? A Scoping Review

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Although the thromboembolic risk associated with oral estrogen is recognized, the risk profile of vaginal estrogen treatment remains inconclusive. This scoping review aims to explore available evidence on the association between vaginal estrogen treatment in postmenopausal women and the risk of thrombosis. We conducted a systematic search of PubMed and Embase, investigating vaginal estrogen treatment and thrombosis, for all records up to May 21, 2025. The process was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis Extension for Scoping Reviews (PRISMA-ScR) and a preregistered protocol (osf.io/hgecd). We excluded systemic estrogen treatments, contraception, fertility treatments, gender-affirming therapies, and hormone treatments other than estrogen. Eligible study designs included clinical trials, randomized controlled trials, observational, case-control, and cohort studies. The search identified 866 articles, whereof 8 were eligible. Manual searching through citations and references yielded 3 additional studies, resulting in 11 included articles, all observational. One study reported a reduced risk of recurrent myocardial infarction (MI) following discontinuation of vaginal estrogen treatment (adjusted hazard ratio [aHR]: 0.54; 95% confidence interval [CI]: [0.34-0.86]). Remaining studies found no thrombosis risk in vaginal estrogen users. For venous thromboembolism (n = 6), effect estimates ranged from null to reduced risk (aHRs: 0.68 [0.36-1.28], to 1.06 [0.58-1.93]), similar for MI (n = 5; aHRs: 0.52 [0.31-0.85] to 0.83 [0.77-0.89]) and stroke (n = 6, aHRs: 0.68 [0.62-0.70] to 0.96 [0.93-0.99]). This scoping review, including studies of generally moderate to high methodological quality, indicates no increased risk of thrombosis associated with vaginal estrogen use in postmenopausal women. Further prospective, high-quality clinical trials, including high-risk populations and women with prior thrombosis, are warranted.

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System failure and health inequity in plain sight: Menopause, misdiagnosis, and the cost to women's physical, social, economic, mental health and wellbeing

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Women make up half of the global population, yet their health continues to be overlooked, underfunded, and insufficiently studied. At the same time, they represent 70-80% of the worldwide health care workforce. As a result, Europe is not only dealing with an ageing population but also an ageing health workforce. This means the very foundation of health care systems is itself being neglected and inadequately supported. The situation is further worsened by the lack of education on menopause among health care providers, as it is often not part of the medical curriculum. This gap in knowledge, combined with limited systemic support, leaves many women in menopause feeling uncertain, overwhelmed, and alone. The consequences for both their physical and mental well-being can be significant and concerning. That is why we are calling for multi-pronged action; i) Comprehensive Menopausal Education and Training ii) Increased Ring-Fenced Research Funding iii) Women-Led Research and Empowerment iv) Menopausal-Friendly Workplace Policies and v) Allyship of All Genders.

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The Effect of Menopausal Symptoms on Subjective Well-Being

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Background: Menopausal symptoms may adversely affect women's overall health and well-being. Aim: This study investigated the effects of menopausal symptoms on subjective well-being in women in the 40-65 age group. Methods: The study sample consisted of 510 women, with 318 postmenopausal and 192 perimenopausal participants. Data were gathered using a Sociodemographic Information Form, the Menopause Rating Scale (MRS), and the Subjective Well-being Scale (SWBS), all administered as self-report instruments. Menopausal status was determined using the Stages of

Reproductive Aging Workshop +10 criteria. Descriptive statistics, Chi-square test, Pearson correlation, and regression analyses were used. Results: Three regression models were specified to investigate the relationship between menopausal symptoms and subjective well-being. Model 1 demonstrated that overall menopausal symptoms were significant negative predictors of subjective well-being (B = -0.749, SE = 0.156, β = -0.260, t = -4.788, p < 0.001, 95% CI [-1.06, -0.44], R2 = 0.068). Model 2 showed that both urogenital symptoms (B = -1.208, SE = 0.517, β = -0.139, t = -2.336, p = 0.020, 95% CI [-2.22, -0.20]) and somatic symptoms (B = -2.068, SE = 0.731, β = -0.168, t = -2.830, p = 0.005, 95% CI [-3.50, -0.64]) were significant negative predictors. Model 3 indicated that psychological symptoms significantly and negatively predicted subjective well-being (B = -1.114, SE = 0.262, β = -0.233, t = -4.253, p < 0.001, 95% CI [-1.63, -0.60], R2 = 0.054). Conclusions: The findings highlight the importance of comprehensive health strategies and demonstrate that psychological symptoms significantly impact overall well-being.

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Sex Differences in Mitochondrial Function: Endocrine Regulation, Immunometabolic Signaling, and Implications for Health and Disease

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Mitochondria are central regulators of cellular bioenergetics, redox balance, and signaling pathways that integrate metabolic and immune responses. Emerging evidence indicates that biological sex is an important determinant of mitochondrial function, in part through the regulatory effects of sex hormones on mitochondrial biogenesis, oxidative phosphorylation, reactive oxygen species production, and quality control mechanisms. Estrogen, testosterone, and progesterone differentially modulate mitochondrial dynamics, substrate utilization, antioxidant capacity, and immune signaling, resulting in distinct mitochondrial phenotypes that may influence disease susceptibility across the lifespan. In this review, we synthesize current knowledge on the mechanistic basis of sex differences in mitochondrial function and highlight mitochondria as key mediators linking endocrine signaling to immunometabolic regulation. We discuss how mitochondrial-derived signals, including mitochondrial reactive oxygen species, mitochondrial DNA release, and cardiolipin exposure, activate inflammatory pathways such as NF-κB, cGAS-STING, and NLRP3 inflammasome signaling. These pathways may contribute to chronic inflammation, gut barrier dysfunction, and systemic metabolic disruption. We further examine the impact of major endocrine transitions, including pregnancy, the postpartum period, menopause, and androgen imbalance in conditions such as polycystic ovary syndrome, on mitochondrial function and disease risk. Particular emphasis is placed on the gastrointestinal tract as a metabolically active and mitochondria-dependent interface, where mitochondrial dysfunction may contribute to epithelial barrier disruption, microbial dysbiosis, and systemic inflammation. Finally, we discuss emerging therapeutic strategies targeting mitochondrial function, including exercise, hormone-based therapies, mitochondria-targeted antioxidants, and interventions aimed at improving mitochondrial quality control. Understanding sex-specific mitochondrial regulation may provide a framework for improved endocrine stratification, mitochondrial phenotyping, and precision medicine approaches across diverse clinical contexts.

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Perimenopausal migraine: a narrative review

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Migraine is a highly prevalent primary headache disorder that disproportionately affects women owing to the effects of sex hormones, particularly estrogen. Hyperestrogenism during early perimenopause, coupled with anovulatory cycles, is associated with an increased frequency and severity of migraine attacks and an increased prevalence of perimenstrual migraine. The natural decline in estrogen levels after menopause is generally associated with migraine improvement; however, this is variable and often complicated by chronic migraine, vasomotor symptoms, cardiovascular risk and use of menopause hormone therapy (MHT). Although there is a trend toward increasing the use of MHT during perimenopause, there are no research trials regarding migraine in this population, although it appears to have a negative clinical effect. This could be explained by the addition of MHT to the already high and fluctuating endogenous estrogen levels. More appropriate during perimenopause is hormone cycle control. This can be achieved using continuous combined hormonal contraception for women with migraine without aura, whereas progestogen-only regimens, with the option of additional transdermal estrogen, can be used in women with migraine with and without aura. For women choosing to use MHT postmenopause, continuous transdermal regimens are better tolerated than cyclical preparations.

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The post-reproductive ovary shifts from a reproductive to an immune-like organ

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Reproductive aging in females is characterized by the irreversible depletion of ovarian follicles, yet the structure and function of the post-reproductive ovary remain poorly defined. Using paired histological and bulk transcriptomic analyses of ovaries from reproductively young (2 m), reproductively old (18 m), and post-reproductive (24 m) mice, we mapped how ovarian identity evolves beyond follicle exhaustion. As expected, follicle loss, stromal remodeling, and increased collagen deposition were observed in the reproductively old and post-reproductive cohorts. Transcriptomic analyses revealed a shift from reproductive functionality to an immune-dominant signature with age. Correspondingly, post-reproductive ovaries exhibited increased infiltration of T cells, macrophages, and multinucleated giant cells. Although old and post-reproductive ovaries diverged substantially from young ovaries, they also showed discrete transcriptomic differences, indicating that the ovary continues to undergo molecular changes after reproductive senescence. Lastly, age-dependent changes in ovarian factors that are predicted to be secreted suggest that the post-reproductive ovary could be a source of pro-inflammatory signaling mediators with the potential to modulate extra-ovarian tissues. These findings challenge the assumption that the post-reproductive ovary is inert, instead indicating that it acquires an immune identity with potential endocrine and paracrine influence on whole-body aging.

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Prevalence of osteosarcopenia in postmenopausal women: a systematic review and meta-analysis

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Background: Osteosarcopenia (OSP) is defined as the coexistence of osteoporosis (OP) and sarcopenia (SP), and is associated with increased risks of falls, fractures, and frailty. Postmenopausal women are particularly susceptible due to estrogen deficiency, however, its overall prevalence in this population remains unclear. Methods: This systematic review and meta-analysis followed PRISMA guidelines and was registered in PROSPERO (CRD420251142908). PubMed, Embase, Web of Science, and the Cochrane Library were searched from inception to 15 October 2025. Eligible studies reported OSP prevalence (or data enabling its calculation) in postmenopausal women, with OP and SP defined by recognized criteria (WHO, EWGSOP, AWGS or equivalents). Two reviewers independently performed study selection, data extraction, and quality assessment using the JBI checklist. Pooled prevalence was estimated using generalized linear mixed models and random-effects meta-analysis with Hartung-Knapp-Sidik-Jonkman adjustment. Heterogeneity, influence analyses, publication bias, and exploratory subgroup analyses by diagnostic criteria, economic level, geographic region, and population source were conducted. Meta-regression evaluated the effect of mean age. Results: Sixteen studies involving 7,631 postmenopausal women and 1,421 OSP cases were included. The pooled prevalence of OSP was 15.6% (95% CI: 9.6-24.3%; I^2=97.1%). Using consistent criteria, prevalence was 20.1% (95% CI: 10.7-34.4%) for WHO + AWGS 2019 and 12.7% (95% CI: 2.3-47.5%) for WHO + EWGSOP 2018. Exploratory subgroup analyses showed higher prevalence estimates in developing versus developed regions (18.0% vs. 12.7%), Asian versus non-Asian countries (18.7% vs. 8.2%), and hospital-based versus community-based populations (26.4% vs. 8.7%). No significant study-level association between mean age and OSP prevalence was observed in meta-regression analyses based on the 14 studies with available mean age data. Most studies were of high quality, and no significant publication bias was detected. Conclusion: This meta-analysis found a pooled OSP prevalence of 15.6% among postmenopausal women across included studies. Given high heterogeneity, this estimate should be interpreted with caution, as it reflects diverse settings and diagnostic approaches. Exploratory subgroup analyses suggested higher prevalence in developing regions, Asian populations, and hospital-based settings. Standardized criteria and integrated bone-muscle assessment may improve early identification and management of OSP in this high-risk population.