Selección de Resúmenes de Menopausia

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The association between triglyceride-glucose index and all-cause mortality in postmenopausal women: A cohort study from NHANES

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Elevated triglyceride-glucose (TyG) index has been linked to cardiovascular risk and metabolic disorders, but its relationship with all-cause mortality in postmenopausal women remains unclear. We aimed to investigate the association between baseline TyG index and all-cause mortality among postmenopausal women, and to determine whether a nonlinear (threshold) relationship exists. We conducted a prospective cohort study using data from the U.S. National Health and Nutrition Examination Survey 2001-2014. Participants were 3925 postmenopausal women (weighted mean age 63.1 years). Fasting subsample survey weights were applied, and the complex multistage sampling design (stratification and clustering) was fully accounted for in all analyses. TyG index was calculated from fasting triglyceride and glucose measurements. The main outcome was all-cause mortality. We used restricted cubic splines and design-based two-piecewise Cox proportional hazards regression to model nonlinear associations and identify a potential threshold. Over a median follow-up of approximately 9.9 years, 1088 deaths occurred (27.7% of participants). The association was nonlinear: the two-piecewise Cox analysis identified a significant threshold at TyG = 9.06 (95% confidence interval [CI] 8.92-9.26). Below the threshold, TyG index was not significantly associated with mortality (hazard ratio 0.88, 95% CI 0.69-1.11). Above this threshold, each 1-unit increase in TyG was associated with a 44% higher risk of all-cause mortality (hazard ratio 1.44, 95% CI 1.05-1.96, P = .020) after full adjustment for covariates. In this cohort of U.S. postmenopausal women, an elevated TyG index above 9.06 was associated with increased all-cause mortality. The relationship was nonlinear, with a risk threshold. This threshold should be considered exploratory and requires external validation before clinical use.

Editora**: Cálculo TyG: Triglicéridos 180 mg/dL; Glucosa 100 mg/dL. 180×100=18.000/2= 9.000. Log Nat 9000= 9.1.* ***TyG 9.1

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Dyslipidemia across the menopause transition: Mechanisms, trajectories, and opportunities for cardiovascular prevention

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The menopause transition is associated with a notable increase in the risk of cardiovascular disease (CVD), making this period a critical window for early prevention and intervention. This heightened CVD risk is driven in part by adverse changes in lipid metabolism. As estrogen levels decline, women experience significant increases in low-density lipoprotein cholesterol (LDL-C) and triglycerides, along with alterations in high-density lipoprotein cholesterol (HDL-C) composition and function that reduce its cardioprotective properties. These lipid changes occur independent of chronological aging, with the most pronounced increases occurring within one year of the final menstrual period. Dyslipidemia is an important modifiable risk factor for CVD during this life stage. Evidence-based lifestyle interventions, such as Mediterranean and Dietary Approaches to Stop Hypertension (DASH) dietary patterns along with regular aerobic or resistance training, can help mitigate these adverse lipid changes. Additional behavioral modifications, including smoking cessation and limiting alcohol intake, further reduce cardiovascular risk. When lifestyle interventions are insufficient, statins remain first-line therapy for dyslipidemia management. While menopausal hormone therapy favorably affects lipid profiles, current guidelines do not recommend its use for dyslipidemia management or CVD prevention due to inconsistent benefits and potential risks. This review evaluates current evidence on dyslipidemia across the menopause transition, distinguishing menopause-related lipid changes from those of chronological aging, and examines the impact of modifiable lifestyle factors and therapeutic interventions on lipid trajectories during this reproductive stage.

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Insurance Type and Menopausal Hormone Therapy Use Among US Women

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Importance: Access to menopausal hormone therapy (MHT) in the US remains low. Understanding whether insurance type is associated with MHT underuse and racial disparities in menopausal care is critical for designing interventions that increase evidence-based and equitable menopause care. Objective: To examine whether Medicaid compared with private insurance is associated with lower MHT use among US women. A secondary objective explored the degree that payer type is associated with observed racial differences in MHT use. Design, setting, and participants: This cross-sectional study used the 2013-2014, 2015-2016, and 2017-2020 cycles of the National Health and Nutrition Examination Survey (NHANES) to identify a survey-weighted analytic subset of eligible respondents of a population-based survey of the US civilian, noninstitutionalized population. The study included women aged 45 to 64 years with Medicaid or private insurance coverage who were eligible for MHT. Statistical analyses were performed between March 26, 2024, and December 2, 2025. MHT use was the primary outcome. A secondary analysis assessed whether insurance status was associated with observed racial differences in MHT use. Associations were estimated using multivariable logistic regression. Results: The unweighted sample included 1666 National Health and Nutrition Examination Survey respondents (n = 22 275 545, weighted). Of the weighted final sample, 91.4% had private insurance, and 8.6% had Medicaid. Individuals insured by Medicaid were less likely than those with private insurance to report a history of MHT use (10.7% [95% CI, 6.9%-16.0%] vs 20.7% [95% CI, 18.1%-23.5%]). After adjustment, Medicaid-insured women had lower odds of MHT use than privately insured women (OR, 0.50 [95% CI, 0.28-0.87]). In a secondary analysis, Black participants showed lower odds of MHT than White participants (OR, 0.68 [95% CI, 0.45-0.95]), but this difference was attenuated once insurance status was included in the model (adjusted OR, 0.72 [95% CI, 0.50-1.04]). Medicaid coverage was associated with significantly lower MHT use, suggesting that insurance type may play a role in menopausal care disparities. Insurance status may also partially explain previously observed racial gaps in MHT use. These findings highlight the need for policy and clinical interventions to address insurance-related barriers and to promote equitable menopausal care.

Circ Popul Health Outcomes. 2026 Jul 17:e013433. doi: 10.1161/CIRCOUTCOMES.126.013433.

Associations of Sociodemographic and Neighborhood Vulnerability With Cardiovascular Health in Midlife Women in the United States

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Background: Many women have suboptimal cardiovascular health (CVH), which declines during midlife. Few studies have characterized CVH across the menopausal transition or identified its sociodemographic and neighborhood determinants. Methods: We analyzed a prospective cohort of women in eastern Massachusetts enrolled during pregnancy (1999-2002) and followed to midlife (2019-2024). Exposures included household income, education, race and ethnicity, and neighborhood Social Vulnerability Index (categorized from very low [<20th percentile] to very high [≥80th percentile]; higher categories=greater neighborhood vulnerability). Women self-reported their menopause status using questionnaires. Using Life's Essential 8, we derived CVH scores (0-100 points; higher score=better CVH) at 3-, 8-, 13-, 18-, and 23-year follow-up visits. Linear spline mixed-effect models examined associations of sociodemographics and neighborhood Social Vulnerability Index with differences in CVH across different menopause stages (premenopause, perimenopause, and postmenopause). Results: Among 1200 women (mean enrollment age, 32.1 years; 67.5% Non-Hispanic White), 15.4% had household incomes ≤$40 000/y, 8.8% had ≤high school education, and 17.4% resided in very high Social Vulnerability Index neighborhoods. After covariate adjustment, women with lower income, lower education, or identifying as Non-Hispanic Black exhibited lower CVH across follow-up. Independent of individual sociodemographics, continued residence in vulnerable neighborhoods over time was associated with lower CVH and unfavorable CVH trajectories across follow-up. For example, residence in very high (versus very low) Social Vulnerability Index neighborhoods from enrollment to 3-year follow-up corresponded to mean CVH differences of -6.7 (95% CI, -12.3 to -1.2) at 3-year, -9.8 (95% CI, -15.6 to -4.0) at 8-year, -8.9 (95% CI, -13.9 to -3.9) at 13-year, -6.7 (95% CI, -12.9 to -0.5) at 18-year, and -7.2 (95% CI, -12.5 to -1.9) at 23-year follow-up, and with faster CVH score decline during premenopause (-0.62 points/y; 95% CI, -1.22 to -0.02). Conclusions: Women from disadvantaged sociodemographic backgrounds or residing in vulnerable neighborhoods exhibit poorer CVH across the menopausal transition, highlighting opportunities to optimize long-term CVH and mitigate cardiovascular disease risk.

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Menopause and epigenetic changes: a review

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Menopause is the biological process that occurs with the end of the menstrual cycle, not only involving hormonal changes but also leading to significant epigenetic transformations at the genomic level. Alterations in estrogen, progesterone and androgen levels positively or negatively influence epigenetic mechanisms such as DNA methylation, histone modifications, genome methylation and microRNA expression. These changes can alter the aging process by altering cell gene expression, and immunosenescence can increase susceptibility to osteoporosis, cardiovascular disease and some cancers. Furthermore, altering epigenetic mechanisms through environmental factors (such as nutrition, exercise and stress management) and lifestyle offers promising strategies for maintaining health during menopause and reducing the risk of chronic disease. Menopause-associated epigenetic alterations are not only linked to accelerated biological aging but also play a critical role in the pathogenesis of major chronic diseases, including cardiovascular disease, osteoporosis and hormone-related cancers. These epigenetic changes, particularly DNA methylation patterns and microRNA profiles, have emerged as promising biomarkers for early detection and risk stratification, as well as potential targets for personalized therapeutic interventions. This review aims to review the literature on epigenetic changes associated with menopause.

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Mechanistic redundancy and hierarchy of aging mechanisms: implications for strategies to extend healthspan and biomarker integration

Aging involves a network of interrelated biological processes that differ in causality and impact. This review proposes a hierarchical framework of primary, secondary, and tertiary drivers of human aging while emphasizing the extensive feedback loops and mechanistic redundancy. Primary mechanisms include molecular damage, particularly genomic and mitochondrial DNA damage, and mutation accumulation that cumulatively result in genomic instability, and telomere attrition, which represents a separate primary driver. Although rarely recognized as an independent aging mechanism, female sex hormone decline, particularly the abrupt loss of sex steroid signaling at menopause and earlier perimenopausal changes, may constitute a primary sex-specific driver of human aging as an evolved process that amplifies molecular and physiological deterioration. Mechanisms acting as both primary and secondary include damage to molecules other than DNA including protein damage with loss of proteostasis and lipid damage, which may arise directly from molecular insults or emerge as downstream consequences of DNA damage and other primary mechanisms, while also feeding back to accelerate upstream deterioration. Secondary mechanisms comprise cellular senescence, impaired macroautophagy, deregulated nutrient sensing, epigenetic alterations, mitochondrial dysfunction, and altered intercellular communication. These processes emerge downstream of initial damage and further reciprocally reinforce it. Tertiary mechanisms of aging comprise stem cell exhaustion, chronic inflammation, and dysbiosis, which represent the system-level deterioration exacerbating molecular dysfunction. This hierarchical network-based model suggests that many hallmarks of aging may represent manifestations of redundant upstream molecular insults. This review focuses on primary mechanisms as the causative drivers of aging and proposes that the strategy to extend healthspan may require preventive approaches targeting distinct redundant primary mechanisms. Complex preventive interventions that simultaneously reduce molecular damage, slow telomere attrition, and compensate for estrogen depletion may delay the initiation and amplification of secondary and tertiary aging mechanisms. This framework supports coordinated multi-target strategies for healthspan extension and underscores the need for validated biomarkers that reflect these upstream processes as part of an integrated preventive approach.