Selección de Resúmenes de Menopausia

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The Menopause Transition: Signs, Symptoms, and Management Options

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Context: Menopause, the permanent cessation of menses, reflects oocyte depletion and loss of gonadal steroids. It is preceded by a transition state, the perimenopause, which is characterized by the gradual loss of oocytes, altered responsiveness to gonadal steroid feedback, wide hormonal fluctuations, and irregular menstrual patterns. The goal of this mini review is to discuss the basic pathophysiology of the menopausal transition and the hormonal and non-hormonal management of clinicopathology attributed to it. Evidence acquisition: A Medline search of epidemiologic, population-based studies and studies of reproductive physiology were sought. A total of 758 publications were screened. Evidence synthesis: The reproductive hormonal milieu of the menopausal transition precipitates bothersome vasomotor symptoms, mood disruption, temporary cognitive dysfunction, genitourinary symptoms, and other disease processes that reduce the quality of life of affected women. The endocrine tumult of the menopause transition also exposes racial and socioeconomic disparities in the onset, severity and frequency of symptoms. Hormone therapy (HT) treatment can be effective for perimenopausal symptoms but its use has been stymied by concerns about health risks observed in postmenopausal HT users who are older than 60 and/or women who have been postmenopausal for greater than 10 years. Conclusions: The menopause transition is a disruptive process that can last for over a decade and causes symptoms in a majority of women. It is important for clinicians to recognize early signs and symptoms of the transition and be prepared to offer treatment to mitigate these symptoms. Many safe and effective options, including HT, are available.

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Diet to Reduce the Metabolic Syndrome Associated with Menopause. The Logic for Olive Oil

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The rates of metabolic syndrome are increasing in parallel with the increasing prevalence of obesity, primarily due to its concomitant insulin resistance. This is particularly concerning for women, as the years around menopause are accompanied by an increase in visceral obesity, a strong determinant of insulin resistance. A fall in estrogens and increase in the androgen/estrogen ratio is attributed a determining role in this process, which has been confirmed in other physiological models, such as polycystic ovary syndrome. A healthy lifestyle, with special emphasis on nutrition, has been recommended as a first-line strategy in consensuses and guidelines. A consistent body of evidence has accumulated suggesting that the Mediterranean diet, with olive oil as a vital component, has both health benefits and acceptable adherence. Herein, we provide an updated overview of current knowledge on the benefits of olive oil most relevant to menopause-associated metabolic syndrome, including an analysis of the components with the greatest health impact, their effect on basic mechanisms of disease, and the state of the art regarding their action on the main features of metabolic syndrome.

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Physical activity energy expenditure and fat-free mass: Relationship with metabolic syndrome in overweight or obese postmenopausal women

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Menopause transition is associated with detrimental changes in physical activity, body composition and metabolic profile. Although physical activity energy expenditure (PAEE) is inversely associated with metabolic syndrome (MetS) in individuals at higher risk of CVD, the association is unknown in low-risk individuals. The aim of the study was to investigate the association between PAEE and MetS (prevalence and severity) in inactive overweight or obese postmenopausal women with a low Framingham Risk Score (FRS:< 10%). Cross-sectional data of 126 participants were divided into quartiles based on PAEE (Q1= lowest PAEE) while fat-free mass (FFM) and fat mass (FM) were measured by DXA. MetS prevalence was significantly different between Q1 and Q4 (37.9% vs 13.3%, p= 0.03). After controlling

for potential confounders, MetS severity was negatively associated with PAEE (B= -0.057, p< 0.01) and positively with FFM (B= 0.038, p< 0.001). Moderation analyses indicated that a greater FFM exacerbated the association between PAEE and MetS severity in Q1 and Q2 (PAEE*FFM; B= -0.004; p= 0.1). Our results suggest that displaying a low FRS and lower PAEE increase MetS prevalence and severity. In addition, greater FFM interacts with lower PAEE to worsens MetS severity, while higher PAEE lessened this effect. Novelty - Inactive individuals displaying higher daily PAEE also have a lower MetS prevalence - Greater fat-free mass is associated with a worse MetS severity where a higher PAEE mitigates this deleterious effect in our cohort.

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Compounded bioidentical menopausal hormone therapy - a physician perspective

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One of the defining moments of the 80-year evolution of menopausal hormone therapy (MHT) was the 2002 reveal of the initial results of the combined hormone therapy arm of the Women's Health Initiative (WHI) clinical trial. The exodus from regulatory approved MHT was prompt and profound and accompanied by a rapid acceleration of the compounding pharmacy 'bioidentical' hormone therapy industry. Compounders had recruited prescribers and promoted compounded bioidentical hormone therapy (cBHT) well before the WHI, yet the startling results provided a catalyst that enabled a leap in production of compounded hormones that were variably regulated, basically unstudied, and inconsistently labeled. In this review, the story of the rise of cBHT and the regulatory double standard is eclipsed only by the 2020 findings and recommendations of the US National Academies of Science, Engineering, and Medicine. Their investigation, commissioned by the US Food and Drug Administration, was tasked to: provide an evidence-based summary of the clinical utility of cBHT; evaluate whether the evidence of safety and efficacy supports the use of cBHT; and identify patient populations that might need cBHT in lieu of an approved drug product. Their conclusions are consistent with sound science and their recommendations are in harmony with global menopause societies.

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Menopause and Brain Health: Hormonal Changes Are Only Part of the Story

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Most studies of menopause and brain aging have focused on the role of the sex steroid hormone, estradiol, as a key mechanisms contributing to cognitive and brain aging in women. An emerging literature demonstrates that beyond endogenous estradiol levels, menopausal symptoms, particularly vasomotor symptoms (VMS), are also key determinants of menopause-related changes in cognition and brain function. Critically, that literature shows the importance of using objective techniques to identify associations of VMS with memory performance, brain structure, and brain function. While self-report measures are important patient-centered outcomes in women's health research, objective measures of VMS typically relate more strongly to indices of cognitive and brain health. Currently, it is premature to make a causal claim about VMS and memory dysfunction, but initial findings raise the possibility that women with VMS might experience an improvement in cognition with VMS treatment. More generally, these findings underscore the utility of investigating female-specific risk factors for cognitive decline.

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17α-Estradiol prevents ovariectomy-mediated obesity and bone loss

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Menopause is a natural physiological process in older women that is associated with reduced estrogen production and results in increased risk for obesity, diabetes, and osteoporosis. 17α-estradiol (17α-E2) treatment in males, but not females, reverses several metabolic conditions associated with advancing age, highlighting sexually dimorphic actions on age-related pathologies. In this study we sought to determine if 17α-E2 could prevent ovariectomy (OVX)-mediated detriments on adiposity and bone parameters in females. Eight-week-old female C57BL/6J mice were subjected to SHAM or OVX surgery and received dietary 17α-E2 during a six-week intervention period. We observed that 17α-E2 prevented OVX-induced increases in body weight and adiposity. Similarly, uterine weight and luminal cell thickness

were decreased by OVX and prevented by 17α-E2 treatment. Interestingly, 17α-E2 prevented OVX-induced declines in tibial metaphysis cancellous bone. And similarly, 17α-E2 improved bone density parameters in both tibia and femur cancellous bone, primarily in OVX mice. In contrast, to the effects on cancellous bone, cortical bone parameters were largely unaffected by OVX or 17α-E2. In the non-weight bearing lumbar vertebrae, OVX reduced trabecular thickness but not spacing, while 17α-E2 increased trabecular thickness and reduced spacing. Despite this, 17α-E2 did improve bone volume/tissue volume in lumbar vertebrae. Overall, we found that 17α-E2 prevented OVX-induced increases in adiposity and changes in bone mass and architecture, with minimal effects in SHAM-operated mice. We also observed that 17α-E2 rescued uterine tissue mass and lining morphology to control levels without inducing hypertrophy, suggesting that 17α-E2 could be considered as an adjunct to traditional hormone replacement therapies.

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The Future of Metformin in the Prevention of Diabetes-Related Osteoporosis

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As a worldwide aging population is on the rise, osteoporosis (OS) is becoming a global health burden. Therefore, many researchers and health authorities are looking into the potential prevention and treatment of OS. Although previously regarded as two separate pathological processes, diabetes (DM) and OS are now regarded as two conditions that can occur together. It is now believed that OS can develop as a complication of DM. This relationship is further evidenced through a reduction in bone mineral density in type-1 diabetes with a resulting increased risk of fracture. Although bone mineral density in type-2 diabetes mellitus is normal or increased, there is also increased fragility due to decreased bone quality. These abnormal bone qualities tend to occur through the production of reduced bone microvasculature and advanced glycation end product, AGE. Interestingly, one of the most common treatments for DM, metformin (MF), shows a promising result on the protection of diabetes and non-diabetes related bone turnover. It is believed that MF modulates its effect through the adenosine monophosphate-activated protein kinase (AMPK) pathway. Recent data regarded AMPK as a vital mediator of homeostasis. It is involved not only in glucose metabolism but also in osteogenesis. AMPK can directly influence the production of mature and good quality bone by decreasing osteoclasts, increasing osteoblast formation, and enhancing bone mineral deposition. As an activator of AMPK, MF also upregulates osteogenesis. Furthermore, MF can influence osteogenesis through a non-AMPK pathway, such as the fructose 1-6 phosphatase pathway, by reducing glucose levels. While already recognized as a safe and effective treatment for DM, this article discusses whether MF can be used for the prevention and treatment of OS.