

## Selección de Resúmenes de Menopausia

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### **Isoflavone combined with exercise on bone mineral density in postmenopausal women: A systematic review and meta-analysis of randomized controlled trials.**

Qiu S1, Ma Y1, Jiang C2.

**BACKGROUND:** This meta-analysis of randomized control trials (RCTs) aimed to evaluate the effects of isoflavones supplementation combined with exercise training on bone mineral density (BMD) in postmenopausal women. **METHODS:** Two reviewers did a complete search of two electronic database (Medline, PubMed) records up to January 31, 2019. Risk of bias was classified based on the Cochrane Collaboration tool. The pooled standard mean difference (SMD) combined with 95% confidence interval (CI) was used as the effect size of BMD values. **RESULTS:** A total of four RCTs with 609 participants were included for meta-analysis. The BMD did not differ significantly between isoflavone supplementation combined with exercise training group and placebo group (sub-whole body: SMD = 0.00, 95% CI, -0.23 to 0.24; lumbar spine: SMD = 0.15, 95% CI, -0.30 to 0.60; total hip: SMD = 0.05, 95% CI, -0.18 to 0.298; femoral neck: SMD = 0.10, 95% CI, -0.23 to 0.43; trochanter: SMD = 0.09, 95% CI, -0.14 to 0.33; ward's triangle: SMD = -0.03, 95% CI, -0.24 to 0.30). In addition, combined intervention did not provide additive effects on BMD improvements compared with exercise or isoflavone supplementation alone. The trials included in this meta-analysis were small and some had methodological limitations. **CONCLUSION:** The present meta-analysis reveals that isoflavone supplements combined with exercise training do not significantly increase BMD in postmenopausal women. In addition, combined intervention does not provide additive effects on BMD improvements compared with exercise or isoflavone supplementation alone.

**Calcif Tissue Int. 2020 Jul 2. doi: 10.1007/s00223-020-00713-3. [Epub ahead of print]**

### **Prevalence of Frailty in Older Men and Women: Cross-Sectional Data from the Geelong Osteoporosis Study.**

Tembo MC1, Holloway-Kew KL2, Sui SX2, Dunning T3,4, Low ACH3, Yong SJ3, Ng BL3, Brennan-Olsen SL5,6,7,8, Williams LJ2, Kotowicz MA2,3,6, Pasco JA2,3,6,9.

Few studies have investigated the prevalence of frailty in the Australian general population. This study determined the prevalence of frailty in a population-based sample of older adults and examined the relationship between frailty and comorbid conditions. Men (n = 347) and women (n = 360) aged  $\geq 60$  year from the Geelong Osteoporosis Study (GOS) were assessed between 2016-2019 and 2011-2014, respectively. Frailty was identified using a modified Fried frailty phenotype. Prevalence estimates were standardised to the 2011 Australian population. Kruskal-Wallis test and  $\chi^2$  test were used to analyse data. For women, mean standardised prevalence estimates were 18.3% (14.1-22.5) for frail, 54.1% (47.3-60.8) pre-frail and 22.9% (18.9-26.8) robust. Corresponding estimates for men were 13.1% (9.8-16.3) frail, 47.8% (42.0-53.6) pre-frail and 27.3% (22.7-31.8) robust. Women who were frail were older, shorter, tended to have a higher body mass index (BMI) and used more medications compared to other groups. Compared to robust women, those who were frail were more likely to have cardio-metabolic (OR 3.5 (0.7-20.0)), pulmonary (OR 3.5 (1.5-8.4)) and musculoskeletal (OR 10.1 (2.1-48.0)) conditions. Frail men were older, had a higher BMI and were more likely to have musculoskeletal conditions (OR 5.8 (2.8-12.3)) and tended to be from a lower SES. No further associations were observed. This study reported the prevalence of frail and pre-frail individuals in a population-based sample of Australian men and women. Frailty was associated with musculoskeletal conditions for both men and women; however, associations with cardio-metabolic and pulmonary comorbidities were evident in women only.

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### **Risk of fragility fractures in obesity and diabetes: a retrospective analysis on a nation-wide cohort.**

Adami G1, Gatti D2, Rossini M2, Orsolini G2, Pollastri F2, Bertoldo E2, Viapiana O2, Bertoldo F3, et al.

This study aims to investigate the role of obesity and diabetes on bone health in a nation-wide cohort of women with high risk of fracture. **INTRODUCTION:** The role of obesity and diabetes on fracture risk is yet poorly understood. Body mass index (BMI) and bone mineral density (BMD) are strongly correlated; however, patients with elevated BMI are not protected against fractures, configuring the obesity paradox. A similar controversial association has been also found in diabetic patients. Herein, we present a retrospective analysis on 59,950 women. **METHODS:** Using a new web-based fracture risk-assessment tool, we have collected demographic (including BMI), densitometric, and clinical data (including history of vertebral or hip and non-vertebral, non-hip fractures, presence of comorbidities). We performed a propensity score generation with 1:1 matching for patients in the obese (BMI  $\geq 30$ ) and non-obese (BMI  $< 30$ ) groups, in the diabetics and non-diabetics. Propensity score estimates were estimated using a logistic regression model derived from the clinical variables: age, lumbar spine T-score, and femoral neck T-score. **RESULTS:** We found an association between diabetes and fractures of any kind (OR 1.3, 95% CI 1.1-1.4 and 1.3, 95% CI 1.2-1.5 for vertebral or hip fractures and non-vertebral, non-hip fractures, respectively). Obesity, on the other hand, was significantly associated only with non-vertebral, non-hip fractures (OR 1.3, 95% CI 1.1-1.6). To estimate the individual effect of obesity and diabetes on bone health, we ran sensitivity analyses which included obese non-diabetic patients and non-obese diabetic patients, respectively. **CONCLUSIONS:** Non-obese diabetics had the highest risk of vertebral or hip fracture, whereas obese non-diabetics predominantly had non-vertebral, non-hip fracture's risk. These results should raise awareness in clinical practice when evaluating diabetic and/or obese patients.

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### **3-Year effect of weight loss via severe versus moderate energy restriction on body composition among postmenopausal women with obesity - the TEMPO Diet Trial.**

Seimon RV1, Wild-Taylor AL1, McClintock S1, Harper C1, Gibson AA1, Johnson NA1,2, et al

We have previously shown that a severely energy-restricted diet leads to greater loss of weight, fat, lean mass and bone mineral density (BMD) at 12 months in postmenopausal women with obesity than a moderately energy-restricted diet. We now aim to evaluate whether these effects are sustained longer term (ie, at 36 months). 101 postmenopausal women were randomized to either 12 months of moderate (25 to 35%) energy restriction with a food-based diet (moderate intervention), or 4 months of severe (65 to 75%) energy restriction with a total meal replacement diet followed by moderate energy restriction for 8 months (severe intervention). Body weight and composition were measured at 0, 24 and 36 months. Participants in the severe intervention lost ~1.5 to 1.7 times as much weight, waist circumference, whole-body fat mass and visceral adipose tissue compared to those in the moderate intervention, and were 2.6 times more likely (42% versus 16%) to have lost 10% or more of their initial body weight at 36 months ( $P < 0.01$  for all). However, those in the severe versus moderate intervention lost ~1.4 times as much whole-body lean mass ( $P < 0.01$ ), albeit this was proportional to total weight lost and there was no greater loss of handgrip strength, and they also lost ~2 times as much total hip BMD between 0 and 36 months ( $P < 0.05$ ), with this bone loss occurring in the first 12 months. Thus, severe energy restriction is more effective than moderate energy restriction for reducing weight and adiposity in postmenopausal women in the long term (3 years), but attention to BMD loss in the first year is required.

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### **Neuroprotective effects of vitamin D and 17 $\beta$ -estradiol against ovariectomy-induced neuroinflammation and depressive-like state: Role of the AMPK/NF- $\kappa$ B pathway.**

Zhang WY1, Guo YJ2, Wang KY3, Chen LM1, Jiang P4.

Estrogen replacement therapy (ERT) has been proven to relieve menopausal-related mental disorders including depression in postmenopausal women. However, the unsafety of ERT hinders its clinical use. In this study, we would evaluate whether vitamin D (VD), a hormone with optimal safety profile, could relieve the depressive-like symptom in ovariectomized (OVX) rats. Furthermore, we would determine whether vitamin D and 17 $\beta$ -estradiol (E2) exert neurological function through their immunomodulatory effect in OVX rats. Middle-aged female SD rats were randomly divided into four groups, namely, control (SHAM), OVX, OVX + VD, and OVX + E2. Vitamin D (calcitriol, 100 ng/kg) and 17 $\beta$ -estradiol (30  $\mu$ g/kg) had been daily gavaged in the OVX + VD and OVX + E2 group, respectively. After 10-week administration, vitamin D and 17 $\beta$ -estradiol both showed anti-depressive-like activity in the OVX rats. Using the method of immunofluorescent staining and western blot, vitamin D and 17 $\beta$ -estradiol were demonstrated to

upregulate each other's receptors, including VDR, ER $\alpha$ , and ER $\beta$  in the hippocampus of OVX rats. Additionally, the upregulation of VDR, calbindin-D28k, and calbindin-D9k suggested that the vitamin D signaling system was amplified by vitamin D and 17 $\beta$ -estradiol. Vitamin D and 17 $\beta$ -estradiol showed neuroprotective effects by decreasing OVX-induced apoptosis and neuronal damage, regulating the AMPK/NF- $\kappa$ B signaling pathway, and reducing the proinflammatory cytokines (IL-1 $\beta$ , IL-6, and TNF $\alpha$ ), as well as iNOS and COX-2 in the hippocampus of OVX rats. Collectively, the present study demonstrated that vitamin D and 17 $\beta$ -estradiol could upregulate each other's receptors and regulate the AMPK/NF- $\kappa$ B pathway to relieve the OVX-induced depressive-like state. The results could stimulate translational research towards the vitamin D potential for prevention or treatment of menopause-related depression.

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## **Prevalence of Hypoactive Sexual Desire Disorder Among Sexually Active Postmenopausal Women With Metabolic Syndrome at a Public Hospital Clinic in Brazil: A Cross-sectional Study.**

Dutra da Silva GM1, Rolim Rosa Lima SM2, Reis BF3, Macruz CF2, Postigo S2.

**AIMS:** To evaluate the prevalence of hypoactive sexual desire disorder (HSDD) among postmenopausal women diagnosed with metabolic syndrome (MS) and to compare it to that of a control group without MS. **METHODS:** This is a cross-sectional study carried out in 2 public tertiary hospitals in the state of São Paulo, Brazil, with a sample of 291 postmenopausal women aged between 40 and 65 years. Sexual function was evaluated using the Female Sexual Function Index (FSFI) questionnaire and Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, diagnostic criteria and was related to the diagnosis of MS, which was determined according to the guidelines defined by the Adult Treatment Panel. **MAIN OUTCOME MEASURES:** Analysis of sexual function with emphasis on sexual desire (HSDD), the incidence of MS, and the components of MS. **RESULTS:** The prevalence of HSDD was significantly higher among women diagnosed with MS than among women without MS ( $P = .01$ ). Women diagnosed with high blood pressure ( $P < .01$ ) and increased triglycerides ( $P = .03$ ) also had a higher prevalence of HSDD than did women without these conditions. The FSFI domain scores for desire, arousal, lubrication, orgasm, and satisfaction and the total FSFI score were significantly lower for postmenopausal women with MS, whereas the pain domain score was not significantly different between the groups ( $P = .913$ ). The incidence of female sexual dysfunction was significantly higher among women with MS, regardless of the diagnostic criteria used ( $P < .05$ ). **CONCLUSION:** Postmenopausal women diagnosed with MS have higher rates of HSDD than do women without MS.