Selección de Resúmenes de Menopausia

Semana del 25 al 31 de Marzo de 2015

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Addition of Exercise Increases Plasma Adiponectin and Release from Adipose Tissue.

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INTRODUCTION: Adiponectin is an adipose tissue-derived anti-inflammatory protein that is down-regulated in obesity. The effects of caloric restriction and exercise induced weight loss on adiponectin are not clear. PURPOSE: To determine whether addition of aerobic exercise training to caloric restriction has additive effects over caloric restriction alone on circulating adiponectin concentrations and adiponectin release from abdominal and gluteal adipose tissue. METHODS: Overweight or obese (body mass index=25-40 kg/m, waist>88 cm) postmenopausal women were randomized to 20-week caloric restriction with and without aerobic exercise (CR+EX, n=48 and CR, n=22). Blood samples were collected for measuring plasma adiponectin concentration, and abdominal and gluteal subcutaneous adipose tissue biopsies were performed in a subgroup to determine in vitro adiponectin release, before and after the interventions.RESULTS: The interventions elicited similar amounts of weight loss (CR+EX: -11.3±4.6 kg ; CR:-11.2±3.4 kg) and fat loss (CR+EX: -8.0±3.5 kg; CR:-7.4±2.7 kg). The two groups had differential changes in plasma adiponectin concentrations (p for interaction = 0.014); CR+EX increased (6.9±3.9 to 8.5±4.9 μg/ml, p= 0.0001), while CR did not alter (6.4±4.4 to 6.5±4.5 μg/ml, p=0.42), plasma adiponectin. Likewise, adiponectin release from abdominal and gluteal subcutaneous adipose tissue increased with CR+EX (p=0.0076 and 0.089, respectively), but did not change with CR (p=0.13 and 0.95, respectively). CONCLUSION: Despite similar reductions in body weight and fat mass, the addition of aerobic exercise to caloric restriction increased plasma adiponectin concentrations, which may be partly explained by increased adiponectin release from abdominal and gluteal subcutaneous adipose tissue.

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Osteoporosis: the emperor has no clothes.

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Current prevention strategies for low-trauma fractures among older persons depend on the notions that fractures are mainly caused by osteoporosis (pathophysiology), that patients at high risk can be identified (screening) and that the risk is amenable to bone-targeted pharmacotherapy (treatment). However, all these three notions can be disputed. PATHOPHYSIOLOGY: most fracture patients have fallen, but actually do not have osteoporosis. A high likelihood of falling, in turn, is attributable to an ageing-related decline in physical functioning and general frailty. SCREENING: currently available fracture risk prediction strategies including bone densitometry and multifactorial prediction tools are unable to identify a large proportion of patients who will sustain a fracture, whereas many of those with a high fracture risk score will not sustain a fracture. TREATMENT: the evidence for the viability of bone-targeted pharmacotherapy in preventing hip fracture and other clinical fragility fractures is mainly limited to women aged 65 to 80 years with osteoporosis, whereas the proof of hip fracture-preventing efficacy in women over 80 years of age and in men at all ages is meagre or absent. Further, the anti-hip fracture efficacy shown in clinical trials is absent in real-life studies. Many drugs for the treatment of osteoporosis have also been associated with increased risks of serious adverse events. There are also considerable uncertainties related to the efficacy of drug therapy in preventing clinical vertebral fractures, whereas the efficacy for preventing other fractures (relative risk reductions of 20-25%) remains moderate, particularly in terms of the low absolute risk reduction of fractures with this treatment.

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Sex-specific effects of cardiovascular risk factors on endothelium-dependent dilation and endothelin activity in middle-aged women and men.

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BACKGROUND: Healthy middle-aged postmenopausal women have higher endothelium-dependent dilation and lower vasoconstrictor activity of endothelin-1 than men. Whether these sex-specific differences extend to patients with cardiovascular risk factors has not been investigated. The current study aimed to determine whether, in patients with cardiovascular risk factors, sex-specific differences exist in endothelium-dependent dilation and endothelin-1 activity.

METHODS: Forearm blood flow responses were measured by strain-gauge plethysmography during the intra-arterial infusion of acetylcholine, sodium nitroprusside, and the selective endothelin type A receptor blocker BQ-123 in 50 women and 64 men with cardiovascular risk factors. RESULTS: Acetylcholine and sodium nitroprusside induced a significant vasodilation in women and men alike ($p < 0.01$ for both). Also BQ-123 caused a significant vasodilation ($p < 0.001$) in both groups. The vasodilator response to acetylcholine was greater in women compared to men; however there were no differences in the response to sodium nitroprusside and BQ-123 (p = NS for both) between the two sex groups. CONCLUSIONS: Middle-aged women with cardiovascular risk factors have significantly higher endothelium-dependent dilation than middle-aged men; however, vascular endothelin 1 activity is similar in the two groups. These findings suggest that the presence of cardiovascular risk factors is associated with sex-specific effects on endothelium-dependent dilation but not on endothelin 1 activity. Further study is needed to confirm our findings and to characterize the mechanisms underlying this sex-specific regulation of endothelial function.

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Hormone replacement therapy in heart failure.

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PURPOSE OF REVIEW: Despite major advances in medical treatments, survival rates of chronic heart failure (CHF) have not significantly changed in the past 50 years, making it imperative to search for novel pathophysiological mechanisms and therapeutic targets. In this article, we summarize the current knowledge regarding the possibility to treat such anabolic deficiencies with hormone replacement therapy (HRT).RECENT FINDINGS: Mounting evidence supports the concept that CHF is a disease characterized not only by excessive neurohormonal activation but also by a reduced anabo ic drive that carries functional and prognostic significance. The recent demonstration of overall beneficial effects of HRT in CHF may pave the way to slow the disease progression in patients with coexisting CHF and hormone deficiencies. The hypothesis is to identify a considerable subset of CHF patients also affected with hormone deficiency and to treat them with HRT. SUMMARY: Single or multiple HRT may in theory be performed in CHF. Such a novel approach may improve left ventricular architecture, function, and physical capacity as well as quality of life. Larger randomized, controlled trials are needed to confirm this working hypothesis.

Menopause. 2015 Mar 23. [Epub ahead of print]

Estradiol-based postmenopausal hormone therapy and risk of cardiovascular and all-cause mortality.

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OBJECTIVE: Data on the health benefits and risks of postmenopausal hormone therapy (HT) are derived mainly from the use of conjugated equine estrogens. Estradiol-based regimens may have a different risk-benefit profile. We evaluated the risk of death caused by coronary heart disease (CHD), stroke, or any disease among users of estradiol-based HT regimens in a nationwide study in Finland. METHODS: A total of 489,105 women who used HT from 1994 to 2009 (3.3 million HT exposure years), as indicated in the nationwide reimbursement register and the national Cause of Death Register, were followed. A total of 28,734 HT users died during follow-up; among the deaths, 3,843 were caused by CHD and 2,464 were caused by stroke. Mortality risk in HT users with varying duration of exposure (≤1 y, >1 to 3 y, >3 to 5 y, >5 to 10 y, or >10 y) was compared with that in an age-matched background population. RESULTS: Risk of CHD death was significantly reduced by 18% to 54% in HT users and was positively related to HT exposure time. Risk of stroke death was also reduced by 18% to 39%, but this reduction was not clearly related to HT exposure time. Risk of all-cause mortality was reduced in HT users by 12% to 38%, almost in linear relationship with duration of exposure. All these risk reductions were comparable in women initiating HT before age 60 years and women initiating HT at age 60 years or older. CONCLUSIONS: In absolute terms, the risk reductions mean 19 fewer CHD deaths and 7 fewer stroke deaths per 1,000 women using any HT for at least 10 years.

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A systematic review and meta-analysis of physical activity and endometrial cancer risk.

Schmid D, Behrens G, Keimling M, Jochem C, Ricci C, Leitzmann M.

Physical activity is related to decreased endometrial cancer risk. However, a comprehensive investigation of activity domains, intensities, time periods in life, and potential interaction with body mass index is unavailable. We performed a meta-analysis of physical activity and endometrial cancer studies published through October 2014. We identified 33

eligible studies comprising 19,558 endometrial cancer cases. High versus low physical activity was related to reduced endometrial cancer risk [relative risk (RR) = 0.80; 95 % confidence interval (CI) 0.75-0.85]. The corresponding RRs for recreational activity, occupational activity, household activity, and walking were 0.84 (95 % CI 0.78-0.91), 0.81 (95 % CI 0.75-0.87), 0.70 (95 % CI 0.47-1.02), and 0.82 (95 % CI 0.69-0.97), respectively ([Formula: see text]). Walking/biking for transportation, walking for recreation, and walking without specification revealed summary RRs of 0.70 (95 % CI 0.58-0.85), 0.94 (95 % CI 0.76-1.17), and 0.88 (95 % CI 0.52-1.50), respectively ([Formula: see text]). Inverse associations were noted for light (RR 0.65; 95 % CI 0.49-0.86), moderate to vigorous (RR 0.83; 95 % CI 0.71-0.96), and vigorous activity (RR 0.80; 95 % CI 0.72-0.90; [Formula: see text]). A statistically significant inverse relation was found for postmenopausal (RR 0.81; 95 % CI 0.67-0.97), but not premenopausal women (RR 0.74; 95 % CI 0.49-1.13; [Formula: see text]). Physical activity performed during childhood/adolescence, young adulthood/midlife, and older age yielded RRs of 0.94 (95 % CI 0.82-1.08), 0.77 (95 % CI 0.58-1.01), and 0.69 (95 % CI 0.37-1.28), respectively ([Formula: see text]). An inverse relation was evident in overweight/obese (RR 0.69; 95 % CI 0.52-0.91), but not normal weight women (RR 0.97; 95 % CI 0.84-1.13; [Formula: see text]). In conclusion, recreational physical activity, occupational physical activity, and walking/biking for transportation are related to decreased endometrial cancer risk. Inverse associations are evident for physical activity of light, moderate to vigorous, and vigorous intensities. The inverse relation with physical activity is limited to women who are overweight or obese.

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Insights into bone fragility in diabetes: the crucial role of bone quality on skeletal strength [Review].

Yamamoto M.

Meta-analyses have revealed that the relative risk of hip fractures in patients with type 1 and type 2 diabetes mellitus is higher than that in non-diabetic subjects. The risk of fracture in patients with diabetes mellitus increases along with a decrease in bone mineral density (BMD) similarly to those in non-diabetic patients. However, the observed risk of fracture is higher than expected one by BMD in both type 1 and type 2 diabetic patients, indicating that precise estimation of bone fragility by BMD values in patients with diabetes is difficult. Bone strength consists of BMD and bone quality, for this reason, poor bone quality is a most suitable and explicable cause for elevated fracture risk in this population. This bone fragility observed in patients with diabetes mellitus is caused by unique pathogenesis in diabetes, suggesting that osteoporosis in diabetic patients may be one of the diabetic complications and that specific diagnostic criteria for this osteoporosis is required. Bone quality indicators closely related to bone fragility are required to be identified to establish a diagnostic method for osteoporosis in patients with diabetes mellitus.