ABSTRACT

World Congress on Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (WCO-IOF-ESCEO 2021): Educational Lectures Abstracts

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EL1 DIETARY PATTERNS AND FRACTURE RISK S. Maggi¹ ¹CNR Aging Branch-IN, Padua, Italy

Objective: This study aims to review the role of selected dietary patterns in the pathogenesis of fragility fractures. Material and methods: This is a narrative review of concepts and controversies in the most recent scientific literature. Results: Osteoporosis and sarcopenia are two important determinants of falls and fragility fractures that recognize several common risk factors, including malnutrition. In particular, malnutrition is associated to a higher risk of osteoporosis and sarcopenia in a life-course perspective, leading to an increased risk of fragility fracture at older ages. Diet is therefore one of the modifiable lifestyle factors that could represent an effective preventive strategy. Besides calcium and vitamin D, proteins, magnesium, and potassium, zinc, and possibly vitamins A, C, and K play a role. Good dietary intakes of calcium (1,000 mg/day before age 50, 1,200 mg/day beyond 50) and vitamin D (600 IU/day before the age of 70 and 800 IU/day thereafter) are crucial to prevent fragility fractures. Moreover, scientific evidence supports the protective role of n-3 fatty acids, flavonoids, and antioxidants and a negative role of saturated fatty acids and sugar. However, we consume selected foods within a dietary pattern, and recent epidemiological studies have focused on the assessment of selected dietary patterns and risk of fractures, more than of single components. This approach is very important, for two main reasons: first, dietary pattern that prevent not only musculoskeletal diseases, but also cardiovascular diseases, diabetes, and inflammatory bowel diseases might lessen the risk of fractures; therefore, we might consider several pathways, besides those typical of musculoskeletal health, explaining the association of diet and fractures. Specifically, bone loss might occur as a result of impaired calcium homeostasis, if the supply of calcium with the diet is inadequate, but also as a consequence of increased inflammatory cytokines and oxidative stress. Second, the analysis of dietary pattern, more than of single components, makes easier to translate the research findings into dietary recommendations for the general population.

One of the most studied dietary patterns is the Mediterranean diet, assessed usually through the Mediterranean diet scores. Although in general there is a strong evidence supporting a protective effect of the Med diet on fracture risk, the comparison between studies is difficult, due to the different scoring systems used and the differences in the definition of the Med diet (traditional versus alternative). Evidence of the negative association between "healthy diets" (such as the Med diet) and the positive association between the dietary inflammatory index (DII) and fractures has been reported recently in large, longitudinal studies in the USA, Europe and China. Therefore, diets rich on fruits and vegetables, and low on saturated fats and sugars, are consistently reported as protective, but some concerns, and the need for further studies, are expressed for strict vegetarian, and vegan diets, which seems to be have deleterious effects on bone. **Conclusions:** According to this literature review, the adherence to a dietary pattern according to the Mediterranean diet principles, supporting large intake of vegetables, fruits, and cereals, daily intake of one/ two services of dairy, and moderate to low intake of meat and wine, seems to offer the best life-course approach to the prevention of fragility fractures. However, several methodological problems, related to the design, sampling, and instruments assessing the dietary pattern, must be acknowledged, and more studies are needed to fully elucidate the pathophysiological association with fragility fractures.

EL2

MEET THE EVIDENCE OF PHARMA-GRADE CHONDROI-TIN SULPHATE

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Objectives: Osteoarthritis (OA) represents the major cause of chronic pain and disability among the musculoskeletal diseases with a considerable impact on the public health burden. According to ESCEO algorithm, the initial therapy for the treatment of knee OA is the use of SYSADOAs, in line also with other international recommendations. In particular, the ESCEO affords a strong recommendation for the use of prescription, pharma-grade chondroitin sulphate (CS) as long-term background therapy.

Material and methods: The MEDLINE and PubMed databases were searched for randomized controlled trials (RCTs), meta-analyses and review articles on pharma-grade CS to evaluate its benefit–risk profile in OA.

Results: The available data shows that CS 800mg/day is effective and safe in the treatment of knee and hand OA, with increasing evidences available for hip OA. The evidence so far suggested an efficacy comparable between CS and some NSAIDs (i.e. celecoxib). The chronic use (2 years) of CS for knee OA is associated not only with a positive effect on symptoms, but also on articular cartilage. The pharma-grade CS is thus endowed of both SYSADOA and SMOAD effects. Even at higher dosages (1200 mg/day), CS of pharma-grade shows a favourable benefit risk profile together with an improved compliance thanks to a new oral gel pharmaceutical formulation. CS high tolerability is also supported by its long marketing experience in different countries worldwide. Several products containing CS are available on the market, but the scientific evidences and considerations here analysed cannot be extrapolated to support other forms of CS (e.g. food supplements) but only attributed to CS of pharma-grade.

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Conclusions: In line with the recent ESCEO algorithm and thanks to a robust scientific background, pharma-grade CS confirms its role as reference drug in the management of OA acting positively on signs, symptoms and structural changes of the disease.

References

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EL3

PEAK BONE MASS AND PRIMARY FRACTURE PREVENTION

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Osteoporosis is a skeletal disorder characterized by reduced bone strength predisposing to an increased risk of fracture. Bone strength comprises both bone density and bone quality. Bone density in any given individual is determined by the interaction between peak bone mass (PBM) and the amount of subsequent bone loss. Much of the focus in osteoporosis has been on bone loss following menopause and with ageing. However, PBM is at least as important, with some studies suggesting it explains up to 60% of osteoporosis risk. Thus, an individual not attaining optimal PBM during childhood and adolescence may develop osteoporosis without having accelerated bone loss.

Many factors may diminish accrual of PBM. In young women and adolescents, late menarche, oligomenorrhea, or amenorrhea is not uncommon. These menstrual disturbances may occur in the context of strenuous athletic training, emotional stress, and low body weight. Anorexia nervosa is an extreme example in which profound undernutrition and nutritional factors combine with a hypogonadal state to result in failure to achieve PBM, accelerated bone loss, and increased fracture rates. The lack of correction of nutritional factors by oestrogen replacement explains its failure to correct bone loss in anorexia nervosa. Chronic disease in childhood and adolescence may also diminish PBM. Cystic fibrosis, coeliac disease, and inflammatory bowel disease are examples of conditions associated with malabsorption and resultant osteopenia in some young individuals. The osteoporosis of cystic fibrosis is also related to the frequent need for glucocorticoids as well as to other factors, including genetic factors. Glucocorticoids used to treat other inflammatory conditions, such as asthma and arthritis, in children can also inhibit growth and attainment of PBM. In Turner syndrome, both oestrogen deficiency and karyotype (SHOX haploinsufficiency) contribute to small bone size and skeletal fragility. Testosterone production (and its conversion to oestradiol) in adolescent boys and young men is similarly important in achieving PBM. Pathologic delay in the onset of puberty is a risk factor for suboptimal PBM in men.

Genetic factors exert the predominant effect on PBM, but environmental and modifiable lifestyle factors are also important. Among these are adequate nutrition and body weight and physical activity. Thus, maximizing PBM creates a critical opportunity to reduce the impact of subsequent age-related bone loss. The prepubertal years and puberty are also critical times for the development of healthy lifestyle habits. Cigarette smoking, which usually starts in adolescence, should be avoided. A balanced diet, adequate calories, and appropriate calcium and vitamin D nutrition are required to achieve PBM. Excessive pursuit of thinness should be discouraged. The Institute of Medicine recommends children aged 1–3 years need 500 mg of calcium daily, and those aged \geq 4 years need 800 mg daily. To support bone growth, adolescents need approximately 1,300 mg daily; however, only a minority meet these recommendations. Factors contributing to low calcium intakes are restriction of dairy products, a generally low level of fruit and vegetable consumption, and a high intake of low calcium beverages such as soft drinks. A recommended vitamin D intake of 400 to 600 IU/day has been established for young adults. Supplementation of calcium and vitamin D may be necessary in those unable to meet dietary requirements. High dietary protein, caffeine, phosphorus, and sodium can adversely affect calcium balance, but their effects are mitigated in individuals with adequate calcium intakes.

There is strong evidence that physical activity early in life contributes to higher PBM with resistance and high impact exercise likely being the most beneficial. It is estimated that a one standard deviation increase in PBM can result in a 50% decrease in osteoporosis fracture risk. The timing of exercise creates a window of opportunity to increase PBM with the strongest evidence for prepubertal exercise conferring a long-term benefit. Prepubertal boys with a higher protein intake and higher weight-bearing physical activity levels achieved 10% higher PBM, which was maintained over the next 15 years into young adulthood. Optimising both nutrition and physical activity in the prepubertal years is therefore critical to increasing PBM and to reducing the risk of osteoporosis and fractures later in life.

EL4

INTEGRATED CARE PATHWAYS FOR BONE HEALTH: IMPROVING CARE AROUND THE WORLD M. E. Bussell¹

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don, United Kingdom

Research sponsored by Amgen: As people age, maintaining mobility and memory become increasingly imperative; therefore, addressing bone health is the most important way to preserve mobility. Fractures cause reduced quality of life due to the loss of mobility, social isolation, depression and pain. Poor bone health encompasses a broad spectrum of diseases, but it is most often quantified as the cumulative burden of osteoporosis and osteoporotic fractures, also known as fragility fractures. The rates of these fractures have been increasing and are expected to continue rising globally, attributed to the anticipated rise in the worldwide population over age 60. From a population perspective, there is a lack of understanding, and misconceptions need to be demystified: poor bone health is not an inevitable part of ageing. Among health professionals, bone health does not garner the same level of attention as other longterm conditions leading to a fragmentation in the care pathway in bone health, missed opportunities in the delivery of care and a lack of health system preparedness.

No single strategy will be sufficient to address this global public health issue. Coordination across all stakeholder groups is vital to decrease the health and socioeconomic burden of poor bone health. For health professionals, building multidisciplinary care teams to ensure the right approach - health promotion, disease awareness, prevention, diagnosis and treatment — is delivered to the right patient at the right time. Among payors, we need to incentivise and reimburse care for bone health. Among policy-makers, we need to ensure that there is a better understanding of the socioeconomic and health consequences of poor bone health to promote better policies to address needs. Building a more resilient health system approach to bone health based on the evidence and sound policy-making will not only improve population health but will provide cost savings to health systems by preventing poor bone health in the first place. Health systems around the world must prepare for this surge by prioritising bone health to preserve mobility and wellbeing. Working across the life span, we can all benefit from improved bone health throughout the ages and stages of our lives.