MTE5
CALCIUM-VITAMIN D: STILL A ROLE IN OSTEOPOROSIS MANAGEMENT?

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Supplementary calcium and vitamin D are often prescribed alongside antiosteoporosis medication, and may play an important role in the management of older institutionalized adults. In the first randomized controlled trial to consider the efficacy of these agents, a daily dose of calcium (1200 mg) and vitamin D3 (800 IU) in community dwelling elderly women normalized serum parathyroid hormone and 25(OH)D levels and apparently led to a reduced bone loss and decreased risk of hip fracture but in a subsequent study which used a 400 IU daily vitamin D dose, a nonsignificant reduction in hip fractures was observed, possibly as a consequence of the lower doses used.
In recent years, calcium supplementation has been controversial, with some but not all studies suggesting that there may be an increased risk of cardiovascular disease among women prescribed therapy. However recent studies show no association between risk of cardiovascular diseases and calcium supplementation in physiological doses, which can be considered safe. The most recent systematic review of the effectiveness of calcium and vitamin D combined supplementation observed a reduction in hip and total fracture which appeared more marked in the elderly, patients with low body weight and increased fracture risk and concluded that the minimum effective dose of calcium is 1200 mg while vitamin D should not be below 800 IU. Previous studies, including previous systematic reviews, have yielded conflicting results; methodological factors may be the explanation for these differing results, highlighting the need to look at the details of each study. In general, the combination of calcium with vitamin D is well tolerated, although increased frequency of urinary and renal tract stones has been reported and many patients report mild gastrointestinal irritation with calcium supplementation.
This workshop will discuss the available literature, and consider how we can incorporate current knowledge into clinical practice.
Conflict of interest
ED has received honoraria from UCB, Lilly, and Pfizer.

MTE6
REHABILITATION AFTER FRAGILITY FRACTURE

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Fragility fractures are associated with pain, loss of bone mineral density and muscle mass, disability, reduced quality of life, increased risk of subsequent fracture, and death. Guidance for the prevention, management, and treatment of osteoporosis has been developed by multiple national and regional organizations, and international campaigns exist to reduce the morbidity and mortality associated with osteoporosis. The treatment of individuals post fracture is multifactorial. Moreover, other risk factors exist for future fractures, such as sarcopenia, frailty, low supply of dietary protein, poor muscle strength and power, inadequate dynamic balance, and environmental risks such as safe walking environments. The management of most of these risk factors falls broadly within the scope of rehabilitation. Multimodal exercise post fragility fracture to the spine and hip is strongly recommended to reduce pain, improve physical function, and improve quality of life. Outpatient physiotherapy post hip fracture has a stronger evidence base than outpatient physiotherapy post-vertebral fracture. Appropriate nutritional care after fragility fracture provides a large range of improvement in morbidity and mortality. Education increases understanding of osteoporosis which in turn increases utilization of other rehabilitation services. Education may improve other health outcomes such as pain and increase a patient's ability for self-advocacy. Rehabilitation interventions are inter-reliant and research investigating these relationships may increase the relevance of rehabilitation research to clinical care.

MTE7
FRACTURES DURING CHILDHOOD AND ADOLESCENCE

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Growth during early life, through childhood and adolescence is an important determinant of peak bone strength, and thus, risk of later-life osteoporosis. During adolescence, individuals gain 20% of their adult height, 50% adult weight, and 40% of their peak bone mass. During childhood, fractures peak during infancy and in early adolescence. In contrast to later life, males tend to fracture more than females during infancy and adolescence. Data from longitudinal studies show there is an offset in peak growth rates, where height and lean mass occur first, followed by bone area and finally a period of consolidation where bone mineral continues to be accrued. The peak period of fractures occurs after peak height growth and while bone mineral consolidation occurs. The timing of puberty, and rate of both height and weight growth impact peak bone mass and later life fracture risk.
Genetics account for ~ 60–85% of variation in peak bone mass, with environment contributing the remainder.