Importance of calcium, vitamin D and vitamin K for osteoporosis prevention and treatment. Susan A. Lanham-Newa. Proc Nutr Soc 2008;67(2):163-167.

Abstract Throughout the life cycle the skeleton requires optimum development and maintenance of its integrity to prevent fracture. Bones break because the loads placed on them exceed the ability of the bone to absorb the energy involved. It is now estimated that one in three women and one in twelve men aged >55 years will suffer from osteoporosis in their lifetime and at a cost in the UK of >£1·7×109 per year. The pathogenesis of osteoporosis is multifactorial. Both the development of peak bone mass and the rate of bone loss are determined by key endogenous and exogenous factors. Ca supplements appear to be effective in reducing bone loss in women late post menopause (>5 years post menopause), particularly in those with low habitual Ca intake (<400 mg/d). In women early post menopause (<5 years post menopause) who are not vitamin D deficient, Ca supplementation has little effect on bone mineral density. However, supplementation with vitamin D and Ca has been shown to reduce fracture rates in the institutionalised elderly, but there remains controversy as to whether supplementation is effective in reducing fracture in free-living populations. Re-defining vitamin D requirements in the UK is needed since there is evidence of extensive hypovitaminosis D in the UK. Low vitamin D status is associated with an increased risk of falling and a variety of other health outcomes and is an area that requires urgent attention. The role of other micronutrients on bone remains to be fully defined, although there are promising data in the literature for a clear link between vitamin K nutrition and skeletal integrity, including fracture reduction. **Key Words:** Osteoporosis; Fracture; Vitamin D; Calcium; Vitamin K

Vitamin D status and its determinants in adolescents from the Northern Ireland Young Hearts 2000 cohort. Tom R. Hilla *et al.* Br J Nutr 2008;99(5):1061-1067.

Abstract: Despite recent concerns about the high prevalence of sub-clinical vitamin D deficiency in adolescents, relatively few studies have investigated the underlying reasons. The objective of the present study was to investigate the prevalence and predictors of vitamin D inadequacy among a large representative sample of adolescents living in Northern Ireland (54–55°N). Serum concentrations of 25-hydroxyvitamin D (25(OH)D) were analysed by enzyme-immunoassay in a subgroup of 1015 of the Northern Ireland Young Hearts 2000 cohort; a cross-sectional study of 12 and 15 year-old boys and girls. Overall mean 25(OH)D concentration throughout the year was 64·3 (range 5–174) nmol/l; 56·7 and 78·1 nmol/l during winter and summer, respectively. Reported intakes of vitamin D were very low (median 1·7 μg/d). Of those adolescents studied, 3 % and 36 % were vitamin D deficient and inadequate respectively, as defined by serum 25(OH)D concentrations < 25 and < 50 nmol/l. Of the subjects, 46 % and 17 % had vitamin D inadequacy during winter and summer respectively. Gender differences were also evident with 38 % and 55 % of boys and girls respectively classified as vitamin D inadequate during winter ($P < 0·001$). Predictors of vitamin D inadequacy during winter were vitamin D intake and gender. In conclusion, there is a high prevalence of vitamin D inadequacy in white-skinned adolescents in Northern Ireland, particularly during wintertime and most evident in girls. There is a clear need for dietary recommendations for vitamin D in this age group and for creative strategies to increase overall vitamin D status in the population. **Key Words:** Serum 25-hydroxyvitamin D; Vitamin D insufficiency; Determinants; Season; Gender; Adolescents.

Vitamin D deficiency: a worldwide problem with health consequences. Michael F Holick and Tai C Chen. Am J Clin Nutr 2008;87(4):1080S-1086S

Vitamin D deficiency is now recognized as a pandemic. The major cause of vitamin D deficiency is the lack of appreciation that sun exposure in moderation is the major source of vitamin D for most humans. Very few foods naturally contain vitamin D, and foods that are fortified with vitamin D are often inadequate to satisfy either a child's or an adult's vitamin D requirement. Vitamin D deficiency causes rickets in children and will precipitate and exacerbate osteopenia, osteoporosis, and fractures in adults. Vitamin D deficiency has been associated with increased risk of common cancers, autoimmune diseases, hypertension, and infectious diseases. A circulating level of 25-hydroxyvitamin D of >75 nmol/L, or 30 ng/mL, is required to maximize vitamin D's beneficial effects for health. In the absence of adequate sun exposure, at least 800–1000 IU vitamin D3/d may be needed to achieve this in children and adults. Vitamin D2 may be equally effective for maintaining circulating concentrations of 25-hydroxyvitamin D when given in physiologic concentrations.

Vitamin D Deficiency and Risk of Cardiovascular Disease
Thomas J. Wang *et al.* Circulation 2008;117(4):503-511.

Background: Vitamin D receptors have a broad tissue distribution that includes vascular smooth muscle, endothelium, and cardiomyocytes. A growing body of evidence suggests that vitamin D deficiency may adversely affect the cardiovascular system, but data from longitudinal studies are lacking. **Methods and Results:** We studied 1739 Framingham Offspring Study participants (mean age 59 years; 55% women; all white) without prior cardiovascular disease. Vitamin D status was assessed by measuring 25-dihydroxyvitamin D (25-OH D) levels. Prespecified thresholds were used to characterize varying degrees of 25-OH D deficiency (<15 ng/mL, <10 ng/mL). Multivariable Cox regression models were adjusted for conventional risk factors. Overall, 28% of individuals had levels <15 ng/mL, and 9% had levels <10 ng/mL. During a mean follow-up of 5.4 years, 120 individuals developed a first cardiovascular event. Individuals with 25-OH D <15 ng/mL had a multivariable-adjusted hazard ratio of 1.62 (95% confidence interval 1.11 to 2.36, *P*=0.01) for incident cardiovascular events compared with those with 25-OH D ≥15 ng/mL. This effect was evident in participants with hypertension (hazard ratio 2.13, 95% confidence interval 1.30 to 3.48) but not in those without hypertension (hazard ratio 1.04, 95% confidence interval 0.55 to 1.96). There was a graded increase in cardiovascular risk across categories of 25-OH D, with multivariable-adjusted hazard ratios of 1.53 (95% confidence interval 1.00 to 2.36) for levels 10 to <15 ng/mL and 1.80 (95% confidence interval 1.05 to 3.08) for levels <10 ng/mL (*P* for linear trend=0.01). Further adjustment for C-reactive protein, physical activity, or vitamin use did not affect the findings. **Conclusions:** Vitamin D deficiency is associated with incident cardiovascular disease. Further clinical and experimental studies may be warranted to determine whether correction of vitamin D deficiency could contribute to the prevention of cardiovascular disease.