Consequences of maternal vitamin D deficiency

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Osteoporosis is a skeletal disease characterised by low bone mass and susceptibility to fracture. Preventive strategies against osteoporotic fracture can be targeted throughout the life course. Although there is evidence to suggest that peak bone mass is inherited, current genetic markers are able to explain only a small proportion of the variation in individual bone mass or fracture risk. Evidence has begun to accrue that fracture risk might be modified by environmental influences during intrauterine or early postnatal life: (1) Epidemiological studies which confirm that subjects who are born light and whose growth falters in the first year of postnatal life, have significantly lower bone size and mineral content, at age 60 to 75 years; (2) Cohort studies demonstrating that subsequent lower trajectories of childhood growth are associated with an increased risk of hip fracture among such men and women; (3) Detailed physiological studies of candidate endocrine systems which might be programmed have shown that birthweight and growth in infancy alter the functional settings of the GH/IGF-1, and vitamin D/PTH axes; (4) Studies characterising the nutrition, body build and lifestyle of pregnant women which relate these to the bone mass of their newborn offspring, have identified a number of important determinants of reduced fetal mineral accrual: these include maternal smoking, excessive weight bearing physical activity in late pregnancy, and low maternal fat stores. More recently, maternal vitamin D insufficiency during mid and late gestation has been associated with bone mineral content and areal BMD in the offspring at age 9 years. As a consequence, large randomised controlled trials of vitamin D supplementation in pregnancy have been instituted and the results of these will inform public health interventions aiming to reduce the frequency of maternal vitamin D deficiency.