The role of dietary calcium in bone health

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Approximately 99 % of body Ca is found in bone, where it serves a key structural role as a component of hydroxyapatite. Dietary requirements for Ca are determined by the needs for bone development and maintenance, which vary throughout the life stage, with greater needs during the periods of rapid growth in childhood and adolescence, during pregnancy and lactation, and in later life. There is considerable disagreement between expert groups on the daily Ca intake levels that should be recommended, reflecting the uncertainty in the data for establishing Ca requirements. Inadequate dietary Ca in early life impairs bone development, and Ca supplementation of the usual diet for periods of ≤3 years has been shown to enhance bone mineral status in children and adolescents. However, it is unclear whether this benefit is long term, leading to the optimisation of peak bone mass in early adulthood. In later years inadequate dietary Ca accelerates bone loss and may contribute to osteoporosis. Ca supplementation of the usual diet in post-menopausal women and older men has been shown to reduce the rate of loss of bone mineral density at a number of sites over periods of 1–2 years. However, the extent to which this outcome reduces fracture risk needs to be determined. Even allowing for disagreements on recommended intakes, evidence indicates that dietary Ca intake is inadequate for maintenance of bone health in a substantial proportion of some population groups, particularly adolescent girls and older women.

Dietary calcium: Bone health: Life stage

Ca, which accounts for 1–2 % of the adult human body weight, is a major component of mineralised tissues, which contain > 99 % of the total body Ca. Ca is required for normal growth, development and maintenance of the skeleton, where it provides strength and structure. The remainder is present in blood, extracellular fluid, muscle and other tissues, where it plays a role in mediating vascular contraction and vasodilation, muscle contraction, nerve transmission and glandular secretion (Institute of Medicine, 1997).

As bone contains a high proportion of the body Ca and is the major reservoir for Ca, its development and maintenance are the major determinant of Ca needs. Thus, unlike other nutrients, the requirement for Ca relates not to the maintenance of the metabolic function of the nutrient but to the maintenance of an optimal reserve and the support of the reserve's function (i.e. providing structural rigidity to the skeleton; Heaney, 1997). Ca requirements vary throughout an individual's life, with greater needs during the periods of rapid growth in childhood and adolescence, during pregnancy and lactation, and in later life.

Ca is a threshold nutrient, i.e. at suboptimal intakes the ability of the body to store Ca as bone tissue is limited by the intake of Ca, but increasing Ca intake above that required as optimal for genetic or mechanical purposes does not result in further increases in stores (Heaney, 1997). Thus, Ca can only be stored as bone, and increasing Ca intake above that which produces optimal bone mass will not result in more bone.

There are important genetic and environmental influences on Ca requirements. Genetic influences include such factors as bone architecture and geometry, and responsiveness of bone to hormones that mediate the function of bone as the body's Ca reserve (Heaney, 1997). Environmental influences include factors such as dietary constituents and the extent of mechanical loading imposed on the skeleton in everyday life. As a result of their effects on urinary Ca losses, high intakes of both Na and protein increase dietary Ca requirements (Shortt & Flynn, 1990; Cashman & Flynn, 2003; Dawson Hughes, 2003).

Recommended calcium intakes

Traditionally, daily Ca requirements have been estimated by a factorial approach. This practice involves estimating the average physiological requirement for absorbed Ca derived from balance data and adjusting for incomplete utilisation and variation in requirements among individuals and in the bioavailability of Ca among food sources. This approach is subject to considerable uncertainty, as is evident from the wide variation in estimates of daily Ca requirements made by different expert authorities. For example, the US and UK authorities have established very different recommendations for Ca intake (National Research Council, 1989; Department of Health, 1991; see Table 1). Much of this divergence arose as a result of different interpretations of available data, e.g. different estimates of absorption efficiencies and obligatory losses of Ca were used by the two authorities (Cashman & Flynn, 1999).

More recently, the US Food and Nutrition Board (Institute of Medicine, 1997) established recommendations for daily Ca intake for different population groups, principally using data from metabolic balance studies to develop a non-linear regression model to predict the lowest value of intake at which mean maximal Ca retention is attained. This approach is based on the principle that maximising the Ca reserve in the skeleton is required in order to maximise skeletal strength, and that an adequate Ca intake is needed to achieve and preserve these genetically-determined upper limits of both skeletal size and density (Institute of Medicine, 1997). A major limitation of this approach is the limited balance data available, and this shortage of data resulted in the establishment of an 'adequate intake' rather than a recommended dietary allowance, since no estimate of average requirement could be derived (Table 1).

Bone structure and function

Bone is a specialised connective tissue that provides mechanical support for the body, facilitating muscle action and locomotion, and protecting the vital inner organs. Bone consists of a mineral phase of crystals of hydroxyapatite $Ca_{10}(PO_4)_6(OH)_2$ and other ions, an organic phase (osteoid) of collagen fibres, and a ground substance formed by glycoproteins and proteoglycans (Prentice *et al.* 2003). Three cell types produce and maintain bone:

- osteoblasts (bone-forming cells), which secrete osteoid and modulate the crystallisation of hydroxyapatite;
- osteoclasts (bone-resorbing cells), which are responsible for the resorption of bone for the repair of bone surfaces and the remodelling of bone;
- osteocytes, which are osteoblasts that have become embedded within the mineralised regions of bone and are involved in the sensing and translation of information about the internal bone environment.

There are two different types of bone: cortical (compact) bone, which is a thick and dense layer of calcified tissue that forms the outer surfaces of most bones and the shafts of the long bones; cancellous (trabecular) bone, which has a spongy appearance and consists of a lattice of thin calcified

Table 1. Recommended calcium intakes in the USA and UK*

US RDA (1989)		UK RNI (1991)‡		US AI (1997)	
Age-group (years)	(mg/d)	Age-group (years)	(mg/d)	Age-group (years)	(mg/d)
0–0·5	400	0–1	525	0–0·5	210
0–0·5	600			0·5–1	270
1–3	800	1–3	350	1–3	500
4–6	800	4–6	450	4–8	800
7–10	800	7–10	550		
11–14 M	1200	11–14 M	1000	9–13 M	1300
15–18 M	1200	15–18 M	1000	14–18 M	1300
11–14 F	1200	11–14 F	800	9–13 F	1300
15–18 F	1200	15–18 F	800	14–18 F	1300
19–24	1200	19–50	700	19–30	1000
25–50	800			31–50	1000
> 51	800	> 50	700	> 50	1200
Pregnancy	1200	Pregnancy	NI	Pregnancy	
				≤18	1300
				19–50	1000
Lactation	1200	Lactation	+550	Lactation	
				≤18	1300
				19–50	1000

RDA, recommended dietary allowances; RNI, reference nutrient intake; AI, adequate intake; M, males; F, females; NI, no increment.
*Estimates of Ca requirements refer to males and females unless stated otherwise.
†Based on a factorial approach (National Research Council, 1989).
‡Based on a factorial approach (Department of Health, 1991).
§Based principally on maximal Ca retention (Institute of Medicine, 1997).

trabeculae, and is found at the ends of long bones and within flat bones and the vertebrae. Cortical bone has 80–90 % of its volume calcified, while in cancellous bone the corresponding value is only 15–25 %. The main function of cancellous bone is regarded as metabolic, whereas the role of cortical bone is predominantly structural (Prentice *et al.* 2003).

Bone turnover is central to maintenance of the structural function of the skeleton. It is a cyclical process whereby the skeleton undergoes continual renewal by a phased sequence of bone resorption followed by bone formation, processes which are coupled but separated in time, resulting in the modelling of growing bone and the remodelling of existing tissue (Kanis, 1991).

Bone acts as a metabolic reservoir of Ca for the maintenance of extracellular homeostasis. The concentration of Ca^{2+} in blood serum is maintained within narrow limits (2·3–2·75 mmol/l) by the concerted action of the three calciotropic hormones, parathyroid hormone, 1,25-dihydroxyvitamin D and calcitonin. When serum Ca^{2+} concentration falls Ca^{2+}-sensing receptors on the parathyroid glands detect the change and stimulate an increase in parathyroid hormone secretion. Parathyroid hormone acts on bone to release Ca and on the kidney to reduce urinary Ca excretion and increase the production of 1,25-dihydroxyvitamin D. In turn, 1,25-dihydroxyvitamin D acts on the intestine to increase Ca absorption and on bone to further release Ca. Increases in serum Ca^{2+} are reversed by calcitonin, which is secreted by the thyroid gland, and by negative feedback by 1,25-dihydroxyvitamin D on parathyroid hormone secretion (Cashman & Flynn, 1999; Prentice *et al.* 2003).

Bone development

In human subjects bone mass increases during childhood and adolescence, and from birth until the age of about 20 years Ca accumulates in the skeleton at an average rate of 150 mg/d. Acquisition rates are highest in infancy and during the pubertal growth spurt, and are lower during the other periods of childhood. Linear growth stops at the end of puberty, but bone mass continues to increase for some time afterwards, reaching a peak during young adulthood. The age at which peak bone mass is achieved varies between different regions of the body and different populations. After peak bone mass has been achieved there is a slow decline in bone mass. This decline is accelerated in women following the menopause. The rates of post-menopausal bone mineral loss average 1–2 %/year in cortical bone and 2–3 %/year in cancellous bone (Prentice et al. 2003).

Ca is one of the primary bone-forming minerals. At birth an infant has approximately 20–30 g Ca and by maturity the Ca mass is approximately 1200 g, of which approximately 99 % is in the bones and teeth (Prentice & Bates, 1993). Nutritionally, therefore, adequate Ca intake is critical to the achievement of optimal peak bone mass and modifies the rate of bone loss associated with ageing.

Osteoporosis

The internationally-agreed description of osteoporosis is a systemic skeletal disease characterised by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture (World Health Organization, 1994). The disorder is a major health problem through its relationship with these fractures, which typically occur at three skeletal sites, the hip, wrist and spine. It has been estimated from incidence rates derived in North America that a lifetime risk of a fragility fracture at one of these three sites among white women aged 50 years is approximately 40 %, with a comparable risk of 13 % among white men. Taking into account sites other than the hip, spine and distal forearm, the lifetime risk among women aged 50 years could be as high as 70 % (Melton et al. 1992). Hip fractures lead to an overall reduction in survival of approximately 15 %, and the majority of excess deaths occur within the first 6 months following the fracture. Fractures are also associated with considerable morbidity, necessitating hospitalisation in most cases, with an average length of hospital stay approaching 30 d. Hip fractures account for >90 % of the enormous health service budget spent on osteoporosis (Prentice et al. 2003).

An estimated 1.7×10^6 hip fractures occurred throughout the world in 1990. As a result of the increasing population and increased life expectancy, that number is expected to be $>6 \times 10^6$ by 2050. Currently, the majority of hip fractures occur in North America and Europe, but demographic shifts over the next 50 years will lead to large increases in the number of elderly in Asia, South America and Africa. Consequently, there will be a shift in the burden of the disease from the developed to the developing world. Approximately 75 % of hip fractures are expected to occur in the developing world by the year 2050 (World Health Organization, 1999).

The relationship between bone mineral density and fracture risk

The ultimate determinants of fragility fracture are bone strength and trauma. Bone strength is related to the quality of bone, its architecture and its mass. In older adults these characteristics correlate closely with bone mineral density (BMD), measured by dual-energy x-ray absorptiometry or related methods (Prentice et al. 2003). For this reason, the World Health Organization (1994) has a working definition of osteoporosis in terms of BMD relative to the young adult mean:

– osteoporosis, a value for BMD of ≥ 2.5 SD below the young adult mean (T-score ≤ -2.5);
– low bone mass (osteopenia), a value for BMD >1 SD below the young adult mean, but <2.5 SD below this value (T-score <-1 and >-2.5).

Several prospective studies have now demonstrated that for older adults BMD measurement predicts future fracture among women (World Health Organization, 1994). One large study (Cummings et al. 1993) reported a 2·6-fold increase in the age-adjusted risk of hip fracture with each 1 SD decrease in femoral neck density for women aged 65 years followed for a 2-year period. Women with a BMD in the lowest quartile therefore had an 8·5-fold greater risk of hip fracture than those in the highest quartile. A meta-analysis by Marshall et al. (1996) confirmed that the risk of fracture increases by approximately 2-fold for each 1 SD decrease in BMD. Although fracture risk is substantially lower among men than among women at any given age, the relationship between BMD and fracture risk lies on the same curve for each gender (De Laet et al. 1997). Fracture risk also increases with age, independently of BMD, to the same extent in both genders (De Laet et al. 1997), which may be related to changes in bone quality and bone turnover, or non-skeletal functions such as muscle strength or balance.

The bone mineral mass that accumulates at the end of the growth period in human subjects, the peak bone mass, is considered to be a major determinant of the risk of osteoporotic fractures in later life (World Health Organization, 1994). The reason is that for adults >50 years bone mass at the time of measurement reflects the combined effects of peak bone mass achieved in young adult life and subsequent bone loss. Although BMD at any age is a predictor of bone strength and fracture risk at that age (Goulding et al. 2001; Oleson et al. 2002), the indication that BMD predicts osteoporotic fracture risk in old age is based on studies conducted in older adults. The evidence that this relationship can be extrapolated to young subjects is weak, and the extent to which a change in BMD in children and young adults, caused by a change in diet or lifestyle, can predict a change in fracture risk in later life remains undetermined (Prentice et al. 2003).

Part of the variation in peak bone mass between individuals is related to inherited characteristics (60–70 %) and part to environmental factors acting during foetal life, childhood and/or adolescence (30–40 %; Prentice, 2001).

Bone turnover, bone mass and fracture risk

The rate of bone formation and bone resorption, the balance of which over time ultimately determines bone mass, may be assessed by measurement of biochemical markers (Eastell *et al*. 1993). Biochemical markers for monitoring bone turnover all rely on the measurement, in serum or urine, of enzymes or matrix proteins synthesised by osteoblasts or osteoclasts that spill over into body fluids, or of osteoclast-generated degradation products of the bone matrix itself. Serum levels of skeletal alkaline phosphatase and osteocalcin are currently the most convincing markers of bone formation, while the most useful markers of bone resorption are all products of collagen degradation (e.g. pyridinium cross-links or peptides associated with cross-linking at either the N-terminal or C-terminal ends) that may be measured in urine; Cashman & Flynn, 1999).

It is now recognised that an increased rate of bone turnover in adults may be a risk factor for fracture (Riggs *et al*. 1996), partly because it exacerbates bone loss (Hansen *et al*. 1991). Conversely, a reduced rate of bone turnover may lower risk of fracture by slowing the rate of bone loss in later life (Riggs *et al*. 1996). Bone turnover is an important determinant of BMD after menopause and low BMD is associated with high bone turnover (Garnero *et al*. 1996; Scariano *et al*. 1998). High rates of bone turnover are also associated with a disruption of the trabecular network, leading to a loss of connectivity that is not necessarily reflected in a decrease in bone mass (Parfitt, 1984).

Prospective studies have shown an increased risk of osteoporotic fracture with increased rate of bone turnover, independent of bone density, in women at the time of the menopause (Hansen *et al*. 1991; Riis *et al*. 1995) and in elderly women (Garnero *et al*. 1996). The risk of hip fracture is increased approximately two-fold, independently of BMD, in elderly women with values for resorption markers exceeding the reference range for premenopausal women.

In children and adolescents a reduced rate of bone turnover is associated with increased BMD (Johnston *et al*. 1992; Slemenda *et al*. 1997; Dibba *et al*. 2000).

Calcium interventions in older adults

There have been more than twenty randomised controlled trials of the effect of Ca supplementation on bone (density) in the decades following the menopause. These trials have been reviewed by a number of authors (Dawson-Hughes, 1991; Institute of Medicine, 1997; Nordin, 1997; Prentice, 1997; Department of Health, 1998).

Almost all these studies showed a small beneficial trend in BMD for the Ca-treated subjects. In the great majority of these studies this trend was significant at one or more skeletal sites (e.g. the forearm, spine, proximal femur or total body). For example, supplementation studies of post-menopausal women showed that an increase in Ca intake reduces bone loss at the hip (Dawson-Hughes *et al*. 1990; Nelson *et al*. 1991; Reid *et al*. 1993; Chevalley *et al*. 1994; Prince *et al*. 1995), especially for subjects with a low habitual Ca intake (Dawson-Hughes *et al*. 1990). However, a number of studies indicated that increases in Ca intake have little effect on spinal bone mineral in older women (Nelson *et al*. 1991; Chevalley *et al*. 1994; Prince *et al*. 1995).

The benefits of Ca intervention appear to be more marked in late post-menopausal life than at the peri-menopause (Dawson-Hughes *et al*. 1990), although some studies have also found beneficial effects in this latter group (Elders *et al*. 1991). It may be that the greater variation in rates of bone loss in peri-menopausal women obscures the relatively small effect of Ca supplementation. However, Ca contributes to the effectiveness of hormone-replacement therapy and Nieves *et al*. (1998), in a review of clinical trials of hormone-replacement therapy, concluded that a high Ca intake potentiates the positive effect of oestrogen on bone mass at all skeletal sites, and perhaps that of calcitonin on bone mass of the spine.

A number of studies have suggested that the beneficial effect of Ca is most marked in the first year of treatment, particularly at sites that consist predominantly of cancellous bone (Reid *et al*. 1995). The mechanism of this effect is probably that it causes a fall in circulating parathyroid hormone concentrations, which decreases the number of bone remodelling units on the surface of cancellous bone. However, there is also evidence of a smaller residual positive effect on BMD of about 0·25 %/year after the first year (Reid *et al*. 1995), which, if it were to continue over a number of years, would result in a marked cumulative benefit on BMD and a potentially reduced fracture risk.

There is only limited evidence for an effect of Ca intervention on fracture risk in older adults. Three studies have reported a significant effect of Ca on fracture incidence (Chevalley *et al*. 1994; Reid *et al*. 1995; Recker *et al*. 1996). When all randomised clinical trials reporting fracture events are combined, the use of Ca supplements was associated with a 35 % decrease in vertebral fracture risk and a somewhat smaller effect on non-vertebral fractures (Kanis, 1999). However, these studies should be interpreted with caution, since they were relatively small, with limited power to detect the effects of Ca supplementation on fractures.

Three studies have been reported in which Ca was co-administered with vitamin D to elderly subjects. Chapuy *et al*. (1994) demonstrated a reduction of more than one-quarter in non-vertebral and hip fracture rates in a cohort of 3000 elderly women given 20 μg vitamin D and 1200 mg Ca daily studied over a period of 3 years, a finding which was confirmed recently (Chapuy *et al*. 2002). Dawson-Hughes *et al*. (1997) demonstrated a reduction of more than half in non-vertebral fracture rates in 400 older men and women randomised to receive Ca 500 mg plus 17·5 μg vitamin D daily, or to receive a placebo. It is not possible to determine whether the Ca, the vitamin D or the combination were responsible for the outcome of these studies. The effect on fracture rates of vitamin D supplementation alone is unclear, since two large studies of vitamin D intervention in older adults have reported conflicting results (Heikinheimo *et al*. 1992; Lips *et al*. 1996). Overall, the findings indicate a major reduction in morbidity in the elderly when combined Ca and vitamin D therapy is used.

Calcium intervention studies in children and adolescents

A number of published studies of Ca supplementation in children and adolescents have observed increases in bone mineral content and BMD of 2–6 % in response to supplementation with Ca salts, depending on the skeletal site (Johnston *et al.* 1992; Lloyd *et al.* 1993, 1996; Andon *et al.* 1994; Lee *et al.* 1994, 1995, 1996, 1997; Bonjour *et al.* 1997, 2001; Nowson *et al.* 1997; Slemenda *et al.* 1997; Specker *et al.* 1997; Dibba *et al.* 2000, 2002; Stear *et al.* 2003). In these trials Ca supplementation was provided in various salt forms such as citrate malate (Johnston *et al.* 1992; Lloyd *et al.* 1993), carbonate alone (Lee *et al.* 1994, 1995; Dibba *et al.* 2000), carbonate combined with gluconolactate (Nowson *et al.* 1997), or phosphate from milk extract (Bonjour *et al.* 1997, 2001). In another prospective controlled study a positive effect on bone mineral mass was seen after milk supplementation (Cadogan *et al.* 1997). In general, this effect of Ca supplements on bone mineral content and BMD has not been associated with alterations in skeletal size, although in some studies increases at the lumbar spine have been reported, indicating anabolic effects on the skeleton (Lee *et al.* 1995; Bonjour *et al.* 1997, 2001; Dibba *et al.* 2000).

There is no obvious relationship between the magnitude of the increase in bone mineral achieved in different studies and the customary Ca intake of the study group or the level of Ca supplements used (Prentice, 1995). It is possible that the response may be greater in, or limited to, children with lower Ca intake (Bonjour *et al.* 1997) or who are at specific stages of development (Johnston *et al.* 1992; Lloyd *et al.* 1996; Nowson *et al.* 1997), but the data are not consistent (Lee *et al.* 1994, 1995; Lloyd *et al.* 1996; Dibba *et al.* 2000).

An important consideration in the interpretation of longitudinal Ca intervention studies is the phenomenon of the 'bone remodelling transient', a term used to describe the suppression of bone remodelling that is seen with Ca supplementation and that results in a one-time initial gain in bone mass over the first 3–12 months after increasing Ca intake (Frost, 1973). It is thought to reflect completion of osteoblast activity after the inhibition of osteoclastic activity by the increased Ca intake (Slemenda *et al.* 1997). It is transient in so far as much of, if not all, the gain in bone mass appears to be sustained only for the duration of Ca supplementation.

This outcome has been observed on follow up of some Ca supplementation trials in children, which showed that differences in bone mass between subjects and controls disappeared after supplementation ceased (Fehily *et al.* 1992; Lee *et al.* 1996, 1997; Slemenda *et al.* 1997). However, not all studies observed this outcome and some studies showed that at least some of the differences persist for between 1 and 3·5 years after withdrawal of supplement (Bonjour *et al.* 1997, 2001; Dibba *et al.* 2002). For example, results of a follow-up study of 8-year-old prepubertal girls done 3·5 years after the end of a 48-week randomised double-blind placebo-controlled intervention with milk-extracted calcium phosphate (850 mg Ca/d) incorporated into various foods showed that the increase in the overall BMD of the six skeletal sites observed during the intervention was still highly significant ($P = 0.012$; Bonjour *et al.* 2001). The authors concluded that supplementation with this form of Ca can lead to a sustained increase in bone mass accrual that lasts beyond the end of supplementation. However, it is not clear whether the effect is just speeding up maturation with earlier attainment of peak bone mass, or whether the accrual of bone mass is shifted onto a different trajectory resulting in attainment of a greater peak bone mass at maturity. More research is required to determine the long-term effect on bone mineral status of Ca intervention in growing individuals.

Adequacy of calcium intakes

In the absence of reliable indicators of nutritional adequacy for Ca, estimates of Ca deficiency are based largely on adequacy of dietary intake relative to estimated requirements (Flynn & Cashman, 1999). This approach is complicated by the lack of agreement between expert groups on requirements for Ca. However, even taking the lower end of the range of estimated requirements as the yardstick, there is considerable evidence that there is a significant prevalence of dietary inadequacy for Ca in some population groups in many countries (Cleveland *et al.* 1996; Department of Health, 1998; Gregory *et al.* 2000; Hannon *et al.* 2001). For example, in UK women 13–18 % of 14–34 year olds and 8–15 % of those >65 years have habitual Ca intakes less than the lower reference nutrient intake, a level below which intake is almost certainly deficient (Department of Health, 1998). Using the method of Carriquiry (1999) to estimate prevalence of inadequacy, 45 % of 11–18-year-old girls in the UK fail to consume the average requirement for Ca (Gregory *et al.* 2000). Data from the North/South Food Consumption Survey in Ireland show that 23 % of 18–64-year-old women fail to achieve the average requirement for Ca (Hannon *et al.* 2001).

Conclusions

While inadequate dietary Ca intake in childhood and adolescence can impair bone development, it is unclear whether the commonly observed increase in bone mass resulting from dietary Ca supplementation is of lasting benefit, leading to the optimisation of peak bone mass in early adulthood. Resolution of this uncertainty will require longitudinal studies on the relationship between Ca intake throughout the early years of childhood and attainment of peak bone mass in early adulthood.

In older adults inadequate dietary Ca accelerates bone loss and may contribute to osteoporosis. Ca supplementation of the usual diet in post-menopausal women and older men has been shown to reduce the rate of loss of BMD at a number of sites over periods of 1–2 years. However, the extent to which increasing Ca intake reduces fracture risk needs to be determined in studies of adequate size with bone fracture as an outcome. There is good evidence to indicate that a marked reduction in fracture risk can be achieved in the elderly using combined Ca and vitamin D therapy.

There is considerable disagreement between expert groups on the daily Ca intake levels that should be recommended, reflecting the uncertainty in the data used for establishing Ca requirements. Even allowing for disagreements on recommended intakes, evidence indicates that dietary Ca intake is inadequate for the maintenance of bone health in a substantial proportion of some population groups, particularly adolescent girls and older women.

Acknowledgement

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