24

Nutrition and bone health

Mark L. Wahlqvist and Naiyana Wattanapenpaiboon

OBJECTIVES

- To find out about the epidemiology of osteoporosis and fractures.
- To understand bone physiology and how it contributes to bone health.
- To consider the causes of osteoporosis.
- To review the nutritional determinants of bone status.

WHAT IS OSTEOPOROSIS?

Osteoporosis is a disease characterised by an absolute decrease in bone mass that results in an increased susceptibility to fracture especially at the wrist, spine and hip. It is considered to be a major cause of fractures and debilitation in postmenopausal women and the elderly and constitutes an important public health problem (Melton 1988). In recent years, much attention has been directed toward the prevention of osteoporosis, since this disease has become a leading cause of morbidity and mortality in elderly women. Evidence suggests that osteoporosis is easier to prevent than to treat. The prevention of osteoporosis and osteoporosis-related fractures may best be achieved by initiating appropriate health behaviours early in life and continuing them throughout life. Healthy early life practices, including the adequate consumption of most nutrients, regular physical activities and other healthy behaviours, contribute to greater bone mineral measurements and optimal peak bone mass.

EPIDEMIOLOGY OF FRACTURE

The clinical significance of osteoporosis lies in the fractures which occur. Most occur at the spine, wrist and hip, but many fractures at other sites are also associated with a low bone mass independently of age, and should be considered to be osteoporotic. The frequency of osteoporotic fractures is certain to increase in both men and women as a result of the ageing of populations. There were an estimated 1.7 million hip fractures worldwide in 1990. Demographic changes alone could cause the annual number of hip fractures to more than double by the year 2040 (Cooper et al. 1992). The elderly population in developed countries such as the United States has been growing even faster than predicted by the most optimistic assumptions about improving life expectancy. If these trends continue the number of hip fractures could be more than tripled over this period. Cooper and colleagues (1992) have projected that the number of hip fractures occurring in the world each year will rise from 1.66 million in 1990 to 6.26 million by 2050. While Europe and North America account for about half of all hip fractures, this proportion will fall to around one-quarter in 2050, by which time steep increases will be observed throughout Asia and Latin America (Figure 24.1). With urbanisation, the incidence of hip fractures has increased dramatically in several Asian countries in recent years, particularly in elderly men and postmenopausal women. Moreover, the population in Asia is ageing rapidly, leading to a large increase in osteoporosis and hip fractures.

BONE PHYSIOLOGY

Bone consists of an organic matrix, primarily collagen fibres, in which are deposited salts of calcium and phosphate in combination with hydroxyl ions in crystals of hydroxyapatite. The tensile capacity of collagen and the non-compressibility of calcium salts combine to give bone its great strength. Bone is continually undergoing the process of remodelling in order to support a growing body, adapt to changes in lifestyle that impose different stresses and strains, maintain appropriate calcium levels in extracellular fluids, and repair microscopic fractures that occur over

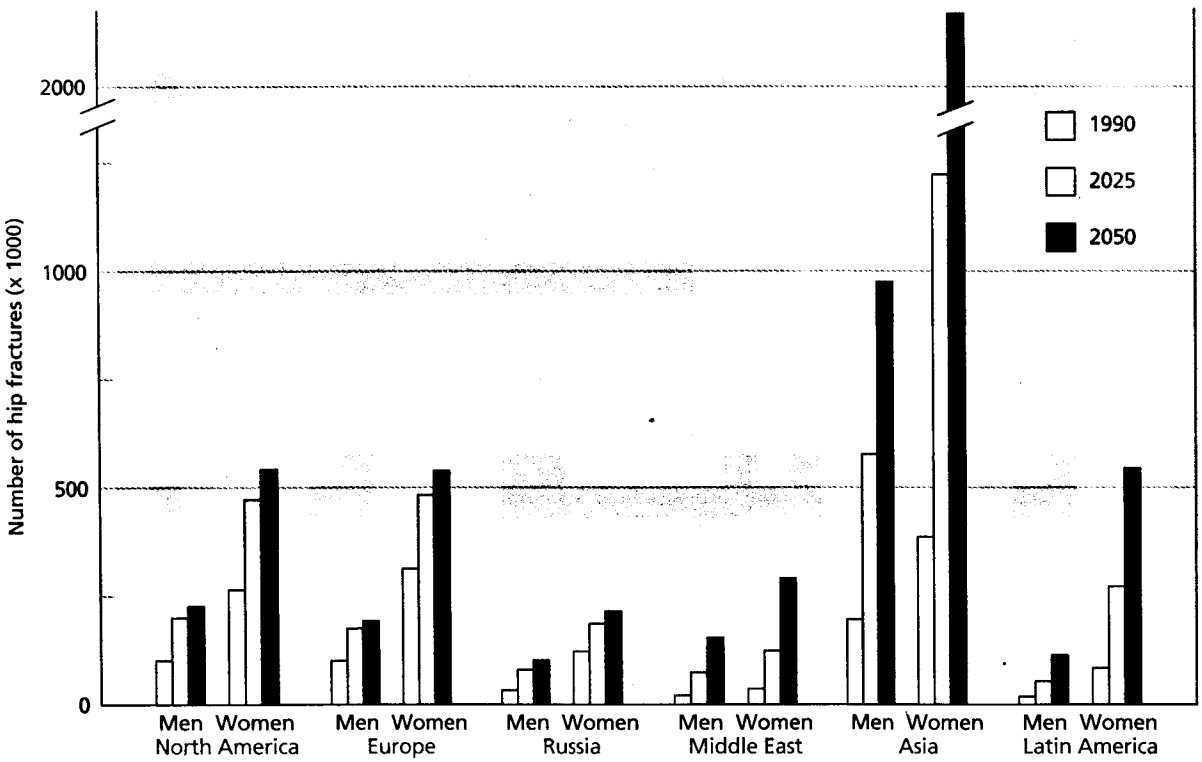

Figure 24.1 Projection of number of hip fractures in different regions of the world in the years 2025 and 2050 (Cooper et al. 1992)

time. Formation of new bone occurs continually in all living bone, with about 4% of surfaces involved at any given time.

Bone remodelling is a process in which bone is continually dismantled and reformed through the action of highly specialised cells, the osteoclasts and the osteoblasts. Osteoclasts resorb both the mineral and organic components of bone, forming small cavities on the inner and outer bone surfaces, which are then refilled with new bone by action of the osteoblasts. Resorption is accomplished in approximately two weeks. The osteoblasts replace resorbed bone and fill the resorption cavities over a period of two to three months. The action of parathyroid hormone (PTH) in promoting activity of the osteoclasts is countered by oestrogen, which reduces bone tissue response to PTH stimuli. Calcitonin inhibits osteoclast activity. In normal young adults, the resorption and formation phases are tightly coupled and bone mass is maintained. Bone loss involves an uncoupling of the phases of bone remodelling with an increase in resorption over formation.

Bone gain and bone loss

During the first year of life, the skeleton increases more in relation to body size than at any other time in the life cycle. In childhood, the skeleton increases in mass with the rest of the body. Under the influence of the gonadal hormones (oestrogen in women and testosterone in men), there is a rapid phase of skeletal growth during adolescence. About 45% of adult skeletal mass is formed at this time. A woman may gain 10% to 15% of skeletal mass in the reproductive stage of life cycle. This amount of bone is almost equivalent to the amount lost after menopause. During the growth periods of childhood and puberty, and beyond into young adulthood, deposition outstrips the resorption of bone. Peak bone mass is generally defined as the highest level of bone mass achieved as a result of normal growth. It is important because, together with age-related loss later on, it is one of the two principal factors determining bone mass later in life and hence one of the factors determining resistance or susceptibility to fracture. The timing of peak bone mass has been considered by various authors to occur from ages early as seventeen to eighteen to as late as 35 years. In each person, peak adult bone mass is determined by a combination of endogenous (genetic, hormonal) and environmental (nutrition, exercise) factors. Peak bone mass is greater in men than in women because of their larger frame size. Both bone mass and bone density are normally lower in women. Peak bone mass is also related to dietary calcium intake and the extent of weight-bearing exercise during the growth and development period. Around the ages of 35 to 40, a slow loss of bone mass begins in both men and women, with a continuous loss over adult life at a rate of 0.5% to 1.2% per year. Loss of bone mass is the result of changes in the mechanisms governing osteogenesis. The processes of resorption and deposition are uncoupled to a degree that interferes with the ability of osteoblast action to keep pace with osteoclast activity. Superimposed on this slower rate of bone loss, women experience a rapid rate of loss beginning at menopause. The accelerated rate of 2% to 3% per year continues in women for around five to ten years after menopause and then declines gradually to a rate leading to a postmaturity loss of 0.25% to 1% per year, so that, by age 70, women again lose bone mass at the same slow rate of men. There is a sub-group of postmenopausal women, however, who lose bone at an even faster rate. Throughout a lifetime, women lose up to 45% to 50% of bone mass and men 20% to 30%. The normal bone loss that occurs with ageing in both genders is related to deterioration of the collagen forming the organic matrix of bone as well as to gradual uncoupling of the remodelling process. Acceleration of the process that occurs in women after menopause is directly related to the lack of oestrogen. Bone loss in men also accelerates in later years, but about ten years later than in women, and it may be related to loss of androgen.

POSSIBLE CAUSES OF OSTEOPOROSIS —NUTRITIONAL FACTORS

Calcium

Bone contains 99% of the body's supply of calcium. The other 1% circulates in the blood and is essential for neural transmission, cardiac function, muscular contraction, and blood clotting. The bones serve as a storehouse to release necessary calcium if the serum concentration required for these vital functions is not maintained. The density of bone mass attained at the time growth is complete determines to some degree what will be left after years of gradual loss. Although peak bone mass is determined by a number of factors, calcium intake from birth through adolescence is a major contributor. The influence of calcium intake

during adulthood is not known, but available evidence indicates that those with a lifetime history of adequate calcium intake are less susceptible to osteoporosis at advanced ages. The level of calcium intake has little effect on the age-related bone loss in either men or women, but it has an impressive effect on hip fractures.

Heaney (1987) states that bone mass is the result of the interplay of three factors: heredity, exercise, and nutrition. Nutrition, particularly calcium intake, may be permissive relative to the other two factors, because it is necessary for the achievement of peak bone mass, but it is not in itself a limiting factor. There is a threshold with calcium. If intake is sufficient, above threshold it will not be significantly related to bone density. Intake below threshold is significantly related. Because dietary calcium clearly is related to bone health, it is important to examine the recommended daily allowance for calcium intake. It is set at 800 mg per day for non-pregnant, non-lactating women. It is necessary also to look at the bioavailability of calcium. It is regulated by intestinal absorption, bone resorption and renal excretion (Figure 24.2). The absorption of calcium at the small intestine is primarily through passive diffusion. Calcium bioavailability can be enhanced with an increase in calcium absorption. It is mediated by a low serum balance leading to an elevated PTH and an elevated renal 1α-hydroxylation of 25(OH)D to produce 1,25-dihydroxycholecalciferol, which enhances calcium absorption. Bioavailability can be improved also by a decrease in intestinal secretion of calcium and in glomerular filtration of calcium, an increase in tubular reabsorption of calcium, or changes in bone resorption. Most women do not get enough calcium.

The best sources of calcium both qualitatively and quantitatively are milk and other dairy products. The high bioavailability of the calcium in dairy products is

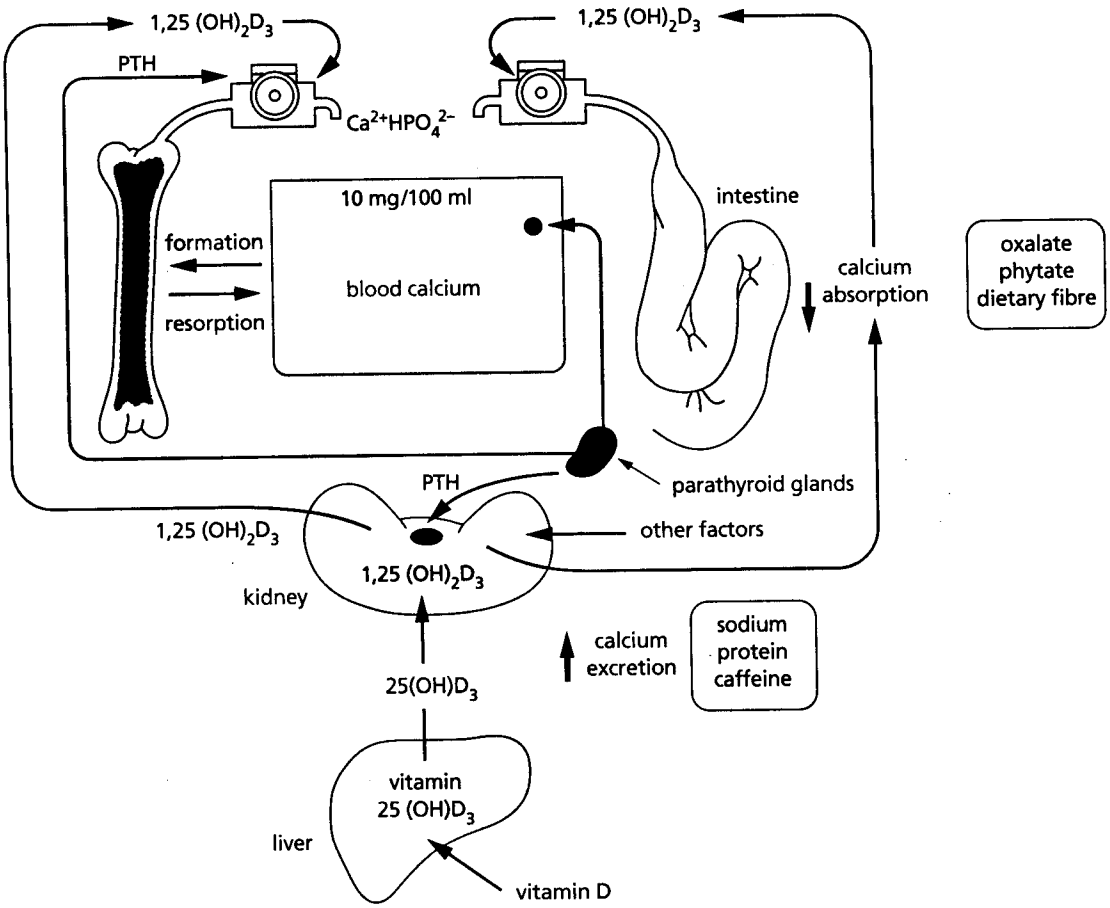

Figure 24.2 Calcium homeostasis and factors affecting plasma calcium concentration

probably related in part to the vitamin D content and the presence of lactose, both of which enhance intestinal absorption. But not all dairy products are good calcium sources. Butter, cream and cream cheese are high in fat and low in calcium. The increased bioavailability of the calcium in lactase-deficient people probably represents an adaptation of high vitamin D to compensate for the presumed low dairy intake. This has important implications for treating lactase-deficient osteoporotic patients because it appears the low calcium levels are a function of a low dietary intake of calcium rather than a defect in calcium absorption from the intestine. There are many non-dairy foods that are good sources of calcium. These include certain leafy green vegetables, such as collard greens and kale, and canned fish with bones such as sardines and salmon. Tofu made with a calcium coagulant, custards, and other soy bean products are all good sources. However, many nutritional factors including oxalates, phytates and large amounts of dietary fibre which interfere with calcium absorption can adversely affect the bioavailability of calcium. Moreover, any calcium loss has a disproportionally high effect on calcium balance compared with absorption. This is because only a fraction of the calcium ingested is absorbed. Dietary factors which increase urinary excretion of calcium include sodium, protein, caffeine and phosphates found in soft drinks.

A low calcium content and the non-dairy sources of calcium are unique features of Asian diets. The major sources of calcium include vegetables, fish and soy bean products. In China and Japan, dairy products account for only about 20% to 25% of the calcium intake. Calcium absorption from vegetables (with the exception of spinach due to its high oxalate level) and from soy bean products is as good as from milk. However, their calcium content is lower than that of dairy products. Eating calcium rich foods is still considered the best way to meet calcium needs, but many women find it difficult to consume adequate dietary calcium. Consequently supplements have become popular. A number of new foods and beverages claim to be calcium fortified. Such fortification may be problematic because not everyone needs additional calcium, although it is difficult to obtain excessive calcium from food.

Skeletal benefits from long-term calcium supplementation have been reported for females at practically every period of the life cycle. The requirement of calcium to maintain the optimal bone mineral content (BMC) or density (BMD) varies at the different stages of the life cycle beyond infancy. The adequate consumption of calcium, in conjunction with vitamin D, in early life will optimise peak bone mass, and adequate intakes of these two nutrients should continue through the remainder of life to help maintain bone mass.

The demand for calcium during the peripubertal period is probably greater than at any other period of the life cycle. A significant amount of bone mass is typically accumulated during this time in females, as well as in males at a similar developmental stage. The incremental amount of BMC is highly associated with calcium intakes (Johnston et al. 1992). Low calcium intakes may compromise the accrual of bone mass. During the adolescent (postpubertal) period, the major impact of sufficient calcium intakes on bone development in females occurs before menarche. However, accrual of bone mass continues at a very high rate for about four more years (Bonjour et al. 1991). The onset of sex hormones related to post-menarche growth apparently dominates the growth pattern so long as nutrients are supplied in amounts sufficient to support growth. Unfortunately, many females during the teenage years avoid milk and other dietary products because of a concern about dietary fat consumption. The low calcium intake, coupled with high consumption of soft drinks containing phosphoric acid, may affect the skeletal accumulation of calcium.

Bone continues to consolidate in females after growth in height ceases at about sixteen to eighteen years of age. Therefore, an adequate supply of dietary calcium is necessary for calcium accumulation in the skeleton. Calcium acts independently on bone to enhance peak bone mass at least up to age 30, particularly in the forearm bones and in the total body BMC, but probably not in the lumbar vertebrae or proximal femur after adolescence ends (Bonjour et al. 1991). Women who exercise regularly are more likely to maintain their bone mass as they proceed toward menopause. In the case of radial bone, they may even improve their BMC and BMD. Avoidance of cigarette smoking and minimal or no alcohol consumption help to maintain peak bone mass. Several studies have reported that calcium supplementation during early menopause has little or no effect on BMC or BMD. It has been demonstrated that an adequate vitamin D status in perimenopausal women is protective against bone loss by reducing the serum concentration of parathyroid hormone. Calcium supplementation of women for five years after menopause results in

significant gains of BMD compared to placebo-treated subjects. A number of studies suggest that calcium alone is not sufficient to slow the rate of bone mass loss late in life. Inadequate intakes of other nutrients, such as vitamin D, may have skewed or confounded the results.

Vitamin D

Vitamin D plays a central and lifelong role in bone health because its active form, 1,25-dihydroxy cholecalciferol, stimulates intestinal absorption of calcium. Age-associated changes in several aspects of vitamin D synthesis, absorption and metabolism may be deleterious to bone health. There are a number of explanations as to why the elderly may have reduced circulating levels of 25-hydroxy cholecalciferol which may be indicative of suboptimal vitamin D status. One is that the elderly absorb less vitamin D from diets; lower dietary intakes may exacerbate this. Another explanation is that the concentration in the epidermis of the vitamin D precursor, 7-dehydro-cholesterol, declines with age, so elderly people synthesise less vitamin D in their skin. In addition, the elderly, voluntarily or involuntarily, generally spend less time in the sun than younger people.

There is increasing evidence that vitamin D supplementation, with or without calcium, may be effective in preventing fractures in the elderly. In communities with a high prevalence of vitamin D deficiency in the elderly population, routine vitamin D supplementation should be encouraged in high-risk subjects (e.g. those who are institutionalised or housebound).

Vitamin K

Compared with the well-established role of vitamin D in bone health, an appreciation that vitamin K also plays a role in skeletal metabolism came comparatively recently. It was discovered in the mid-1970s that mineralised tissue contained a large reservoir of γ-carboxyglutamic acid (Gla), which is known as osteocalcin or bone Gla protein (BGP) and accounts for up to 15% of the non-collagenous bone. The formation of Gla from glutamic residue in the protein precursor requires a vitamin K-dependent enzyme. Two other proteins isolated from bone tissue, namely matrix Gla protein and protein S, neither of which is exclusive to bone tissue and whose roles in bone metabolism are uncertain, are also vitamin K-dependent proteins.

Relatively little is known about the adequacy of vitamin K status in relation to bone health. However, at the nutritional and biochemical level there is good evidence that adequate dietary intakes of vitamin K are needed to sustain maximal formation of BGP. Circulating levels of the under-carboxylated fraction of BGP are found to be responsive to changes in dietary vitamin K, known as phylloquinone.

Other nutritional factors

There are numerous nutritional factors that affect the bioavailability of calcium. Excreted sodium is particularly important as it increases urinary excretion of calcium by means of a sodium–calcium exchange in the renal tubules. Sodium intake is indicated to be a significant determinant of calcium requirement and therefore calcium balance. It has been proposed that a renal calcium leak is important in the development of postmenopausal osteoporosis (Nordin et al. 1991). There is evidence suggesting that a high protein intake is associated with a negative calcium balance. There is a strong negative association between protein intake and bone density in cross-sectional studies. When diets containing a similar amount of protein in the form of normal foods are consumed, however, no calciuria (calcium in the urine) is observed. The difference in response is due to the phosphate naturally associated with protein in the diet which increases the synthesis of PTH by depressing serum calcium, thereby increasing the parathyroid hormone-dependent re-absorption of calcium by the renal tubules (and mobilisation of calcium from bone). The dietary protein–phosphorus relationship is critical to the maintenance of calcium homeostasis on a high protein diet. Most investigators believe that the absolute level of calcium is the most critical factor, largely because phosphorus exacerbates bone loss when dietary calcium is limited. Thus, concern about the effects of a high phosphorus diet is particularly justified when the calcium intake is low.

Oxalates, which are present in high concentration in spinach, bind calcium in an insoluble form and decrease absorption. Phytate (unless broken down by food phytase as with bread leavened with yeast) also reduces calcium availability. Large amounts of dietary fibre can interfere with calcium bioavailability. Considering the beneficial effects of dietary fibre in the prevention and treatment of coronary heart disease and

cancer, however, it is still worthwhile to keep wholegrain products in the diet in moderation (Heaney et al. 1995). Diets high in fat will decrease the bioavailability of calcium. A number of studies indicate that there is no difference in the bone mineral content of postmenopausal vegetarians and omnivores when calcium intakes are similar and when protein intake in the omnivores is within the usual range. Large doses of zinc supplementation or megadoses of vitamin A can also lower calcium bioavailability. Caffeine and phosphates found in soft drinks are known to increase urinary calcium excretion as related to prostaglandin synthesis.

There are a number of compounds in food that are not regarded as nutrients, and yet are of biological relevance in terms of osteoporosis. Boron at levels obtainable from fruits and vegetables interacts with magnesium intake to change endogenous oestrogen status (Nielsen 1994). It also decreases urinary calcium excretion, particularly at low magnesium intakes. Phytoestrogens or exogenous oestrogens found principally in plant derived foods might exert oestrogenic effect on bone in a similar manner to endogenous oestrogens. In addition, genistein and daidzein have been shown *in vitro* to reduce bone resorption. An increase of bone mineral content has been reported in postmenopausal women who were supplemented with a soy diet for 12 weeks (Dalais et al. 1998). Emerging evidence also suggests that consumption of fruit and vegetables may have a positive link with bone health (New et al. 2000).

Nutrition and fracture healing

Fracture healing is a specialised form of wound repair distinguished by the ability to deposit calcium salts and thereby create a structure sufficiently strong to bear mechanical loads. Vitamin D metabolites have been intensively investigated for their roles in controlling bone and mineral homeostasis in various states of metabolic bone diseases. Any specific vitamin deficiency can be assumed to have an impact on fracture healing. For example, vitamin C or ascorbic acid deficiency can affect the integrity of the collagen molecule.

Osteocalcin or BGP has been shown to be chemotactic for osteoclast precursors and monocytes, enhancing the attachment of bone resorbing cells to bone, and playing a role in bone remodelling. Its potential role in fracture healing becomes apparent when osteoporotic patients with crush or femoral fractures are shown to have depressed levels of circulating vitamin K when compared to age matched controls. Many questions still remain regarding the role of nutrition in fracture healing. While the nutrients and vitamin metabolites discussed earlier have been partially investigated, the long-term effects of deficiencies have not been evaluated. Such studies are necessary to clarify the observations made in acute short-term studies and to predict the effects of nutrient alterations on skeletal remodelling around orthopaedic implants.

POSSIBLE CAUSES OF OSTEOPOROSIS —NON-NUTRITIONAL FACTORS

Loss of bone mass to a degree that results in fractures can result from:

1 an excessive acceleration of loss;
2 a peak bone mass so low that with the passage of time, the bones eventually become fragile and susceptible to fracture.

Non-nutritional risk factors for osteoporosis include age, ethnicity, gender, body build, family history, premature menopause, limited exercise, use of cigarettes, alcohol consumption and prolonged use of excess exogenous thyroid hormone.

Genetic make-up

The contribution of heredity to bone mass or density by early adulthood in all ethnic groups has been estimated to be 70% to 80%. This estimation has been accepted by most bone biologists, but a satisfactory explanation for differences in bone mass across races is less clear. The genetic component in bone density is well documented in several twin and mother-daughter studies (Pocock et al. 1987). Premenopausal daughters of osteoporotic mothers have demonstrated reduced bone mass in the spine and femoral neck compared with daughters of non-osteoporotic mothers. It is suggested that there is a strong genetic component in peak bone mass and possibly also in postmenopausal bone status.

Menstrual status

Menstrual status is a major determinant of osteoporosis risk in women. Acceleration of bone loss coincides with

the menopause, either natural or surgical, at which time the ovaries stop producing oestrogen. Any interruption of menstruation for an extended period results in bone loss. The amenorrhoea that accompanies excessive weight loss as seen in anorexia nervosa or as a consequence of excessive exercise has the same effect on bones as menopause. Bone mass in amenorrhoeic athletes has been measured at levels of 25% to 40% below control levels. When menstruation was resumed in these athletes, bone mass increased, but eventually plateaued at a level lower than that of sedentary women. Early menarche is believed to be associated with a higher peak bone mass in young women, but this has not been confirmed and little is known about its effect on later bone status. On the other hand, it is very widely accepted that early menopause predisposes to osteoporosis. This is based on the undeniable fact that loss of bone in women starts at or about the time of ovarian failure, with the consequence that, at any given age, early menopause means more years since menopause in which to lose bone. Age and years since menopause are simply indications of the length of time that the bone has been exposed to negative risk factors. In any given set of postmenopausal women, age and years since menopause are both determinants of bone density, and their effect becomes more apparent with the passage of time.

Body weight

Traditionally it has been stated that osteoporosis is one condition where being overweight acts as a protective factor. Data from cross-sectional studies gave this impression. Longitudinal studies suggest, however, that increasing weight is of benefit to bone but that the maximum benefit is reached by about 110% of ideal body weight. In essence, it appears that thinness is a risk factor for osteoporosis, and not that obesity is protective. A relation between body weight and forearm bone density in postmenopausal women is more due to an effect of weight on the rate of bone loss than an effect on peak bone density. It is interesting to note that vertebral fractures are associated with reduced body weight, but peripheral fractures are not.

Lack of exercise

There is no doubt that exercise, particularly of a vigorous weight-bearing nature, promotes bone formation, and that immobilisation leads to bone loss. Stress on a particular bone or bones probably inhibits post-menopausal bone loss, but the variations in exercise within the normal range of such activity are probably unimportant. At the other end of the scale, people who are immobilised acutely will lose a massive amount of skeletal mineral, particularly over the first six months after immobilisation. Maintenance of healthy bone requires exposure to weight-bearing pressures. Stresses from muscle contraction and maintaining the body in an upright position against the pull of gravity stimulate osteoblast function. It is well known that astronauts under zero or microgravity conditions are subject to rapid loss of bone mass, so much that appropriate exercise is a feature of their daily routines. The loss is faster than in postmenopausal women, and it is unknown whether it occurs via the same mechanism. The increase in the prevalence of osteoporosis and in the incidence of hip fracture in several developed countries in Asia may be due to urbanisation and a reduction in weight-bearing activity. In the past, the effects of a low calcium intake may have been offset by a high level of weight-bearing activity. With urbanisation, physical labour becomes less necessary in everyday life. The effect of a low calcium intake may eventually be manifested as an epidemic of osteoporosis in urbanised parts in Asia (Lau & Woo 1994).

Medications

A number of medications contribute to osteoporosis, either by interfering with calcium absorption or by actively promoting calcium loss from bone. Steroids, for example, affect vitamin D metabolism and can lead to bone loss. Excessive amounts of exogenous thyroid hormone, even in very low amounts, can promote loss of bone mass over a period of time.

Alcohol and tobacco consumption

Cigarette smoking and alcohol consumption are risk factors for developing osteoporosis, probably because of toxic effects on osteoblasts. Excessive alcohol directly impairs bone formation or the replacement of resorbed cavities with new bone, but social drinking is reported actually to improve bone density. Smoking has also been implicated in osteoporosis, but most of the evidence is rather marginal. Smokers go through menopause at an earlier age than non-smokers, so they

are oestrogen deficient for a larger proportion of their lives. Also, smokers as a group have lower body weight, and therefore are more subject to bone loss because of thinness.

RECOMMENDATIONS FOR PREVENTION OF OSTEOPOROSIS

1 Consume a wide variety of foods including those that are calcium dense.
2 Restrict intake of sodium, protein, caffeine and phosphates.
3 Engage in regular physical activity.
4 Quit cigarette smoking.

SUMMARY

- Inadequate nutrition is one of the risk factors of developing osteoporosis. Other risk factors are genetic factors, menstrual status, low physical activity, certain medications, cigarette smoking and excessive alcohol consumption.
- Research on the influence of dietary factors in bone health has concentrated on calcium and vitamin D. It is now appreciated that vitamin K also plays a role in skeletal metabolism.
- There are a number of other nutrients such as protein and dietary fibre, and non-nutrients such as oxalate and boron, that are of relevance in terms of osteoporosis, either by affecting calcium bioavailability or any other mechanisms.

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FOOD AND NUTRITION

Australasia, Asia and the Pacific

Second Edition

Edited by
Mark L. Wahlqvist

Contributors
Madeleine Ball
David R. Briggs
Patricia A. Crotty
Gwyn P. Jones
Antigone Kouris-Blazos
Louise B. Lennard
Richard S.D. Read
Iain Robertson
Ingrid H.E. Rutishauser
Mark L. Wahlqvist
Naiyana Wattanapenpaiboon

Thanks to Antigone Kouris-Blazos for her editorial and technical assistance.

ALLEN&UNWIN

First published in 1997
Second edition published in 2002 by
Allen & Unwin Pty Ltd
83 Alexander Street, Crows Nest, NSW 1590 Australia
Phone: (61 2) 8425 0100
Fax: (61 2) 9906 2218
E-mail: info@allenandunwin.com
Web: www.allenandunwin.com

National Library of Australia
Cataloguing-in-Publication entry:

Food and nutrition: Australasia, Asia and the Pacific.

2nd ed.
Includes index.
ISBN 1 86508 692 4

1. Food. 2. Food analysis. 3. Food—Australian.
4. Food—New Zealand. 5. Nutrition.
6. Nutrition—Requirements. 7. Nutrition
Australia. 8. Nutrition—New Zealand.
I. Wahlqvist, Mark L.

641.3

Set in 10/12 Bembo by Asset Typesetting Pty Ltd
Index compiled by Russell Brooks
Printed by South Wind Productions, Singapore

10 9 8 7 6 5 4 3 2

Contents

Contributors vii

Part I HUMAN NUTRITION: THE CONCEPT AND CONTEXT

1 Introduction to human nutrition *Mark L. Wahlqvist* 3
2 Evaluating the reliability of nutrition information *Antigone Kouris-Blazos* 11
3 Anthropological and sociological approaches to understanding food, eating and nutrition *Patricia A. Crotty* 20

Part II CONTEMPORARY FOOD USE AND SAFETY

4 The food supply *Richard S.D. Read* and *Gwyn P. Jones* 37
5 Food composition and processing *Gwyn P. Jones* 49
6 Food microbiology and food poisoning *David R. Briggs* and *Louise B. Lennard* 70
7 Risks, additives, contaminants and natural toxicants *David R. Briggs* and *Louise B. Lennard* 90
8 New and emerging developments in food production *David R. Briggs* and *Louise B. Lennard* 115
9 Food law *David R. Briggs* and *Louise B. Lennard* 137
10 Contemporary food use: Food supply and food intake *Ingrid H.E. Rutishauser* 152

Part III THE BIOLOGY OF FOOD COMPONENTS

11 Food energy and energy expenditure *Richard S.D. Read* 171
12 Carbohydrates *Gwyn P. Jones* 183
13 Fats *Gwyn P. Jones* 199

14 Protein *Richard S.D. Read* 210
15 Digestion of food *Richard S.D. Read* 227
16 Vitamins and vitamin-like compounds *Mark L. Wahlqvist* and *Naiyana Wattanapenpaiboon* 243
17 Minerals *Gwyn P. Jones* 271
18 Water *Gwyn P. Jones* 283

Part IV LIFESPAN NUTRITION

19 Pregnancy and lactation *Ingrid H.E. Rutishauser* 291
20 Infant nutrition *Ingrid H.E. Rutishauser* 302
21 Childhood and adolescence *Ingrid H.E. Rutishauser* 312
22 Nutrition for activity, sport and survival *Richard S.D. Read* and *Antigone Kouris-Blazos* 322
23 Requirements in maturity and ageing *Mark L. Wahlqvist* and *Antigone Kouris-Blazos* 344

Part V FOOD AND DISEASE

24 Nutrition and bone health *Mark L. Wahlqvist* and *Naiyana Wattanapenpaiboon* 367
25 Genetic individuality, diet and disease *Mark L. Wahlqvist* and *Antigone Kouris-Blazos* 377
26 Overweight, obesity and eating disorders *Richard S.D. Read* and *Antigone Kouris-Blazos* 384
27 Atherosclerosis and coronary heart disease *Madeleine Ball* 415
28 Diabetes *Madeleine Ball* 425
29 Alcohol and diseases related to alcohol *Madeleine Ball* 435
30 Protein energy malnutrition *Madeleine Ball* 443
31 Immune function, infection and diseases of affluence *Mark L. Wahlqvist* and *Antigone Kouris-Blazos* 454
32 Nutrition and cancer *Mark L. Wahlqvist* and *Antigone Kouris-Blazos* 464
33 Food sensitivities *David R. Briggs* and *Louise B. Lennard* 478
34 Nutrition and mental health *Naiyana Wattanapenpaiboon* and *Mark L. Wahlqvist* 487

Part VI FOOD, INDIVIDUALS, ENVIRONMENT AND POLICY

35 Nutrition assessment and monitoring *Ingrid H.E. Rutishauser* 495
36 Nutritional standards of reference *Ingrid H.E. Rutishauser* 508
37 Health promotion and nutrition *Patricia A. Crotty* 522
38 Dietary advice and food guidance systems *Antigone Kouris-Blazos* 532
39 Food, population and sustainable environments *Richard S.D. Read* 558
40 Food and nutrition policies in the Asia–Pacific region: Nutrition in transition *Mark L. Wahlqvist* and *Antigone Kouris-Blazos* 575

Abbreviations 599
Acknowledgments 601
Index 602