

Review Article

Bone health from infancy to adolescence: a narrative review of nutritional and lifestyle determinants

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ABSTRACT

Lifelong bone health is largely determined by the foundation established during childhood and adolescence, when the skeleton undergoes rapid growth and mineralization. This paper explores the critical influence of early-life factors including nutrition, physical activity, hormonal balance and disease states on the development of peak bone mass, a key predictor of osteoporosis and fracture risk in later years. Emphasis is placed on the roles of calcium and vitamin D, the resurgence of nutritional rickets in both developing and developed regions and the decline in physical activity among youth worldwide. This narrative review integrates findings from global studies to highlight the importance of balanced diets, regular weight-bearing exercise and early screening in vulnerable populations. Ultimately, it underscores the need for a comprehensive public health strategy to promote optimal bone development from infancy through adolescence, ensuring skeletal resilience across the lifespan.

Keywords: Bone health, Importance, Lifespan, Paediatric nutrition, Peak bone mass

INTRODUCTION

Bone health is an important, yet often underappreciated, aspect of pediatric well-being. The skeletal system not only provides structural support and protects internal organs, but it also houses the marrow and serves as a reservoir for minerals such as calcium and phosphate. Childhood and adolescence are periods of dynamic bone modeling and remodeling, during which the skeleton grows significantly in size, density and strength. By late adolescence, individuals achieve their peak bone mass the maximum bone density and content attainable which is a critical determinant of lifelong skeletal health. Indeed, approximately 90% of peak bone mass is accrued by the end of the second decade of life, after which gains plateau and eventual losses begin in mid-adulthood.¹ Achieving a high peak bone mass provides a “bone reserve” that

protects against osteoporosis and fractures later in life, while suboptimal development in youth predisposes individuals to fractures and early-onset osteoporosis. Bone health in children is governed by both genetic and environmental factors.

Genetic heritage accounts for an estimated 60–80% of the variance in bone mass, with the remaining 20–40% influenced by modifiable factors such as nutrition and physical activity.² Adequate intake of bone-building nutrients and regular mechanical loading through exercise are essential, calcium and vitamin D are particularly vital, as deficiencies during childhood impair bone accrual and can lead to rickets. Weight-bearing and muscle-strengthening activities stimulate osteoblast activity in accordance with Wolff’s law, resulting in higher bone density in active children compared to sedentary peers.³

In recent years, global lifestyle trends have threatened pediatric bone health. Dietary patterns have shifted toward calorie-dense, nutrient-poor foods, increasing the risk of inadequate calcium and vitamin D intake. Simultaneously, sedentary behaviors driven by urbanization and increased screen time have reduced physical activity, with over 80% of adolescents failing to meet exercise guidelines.³ Consequently, both nutritional rickets in deficient populations and universally lower peak bone mass are emerging concerns, even in developed countries, highlighting the importance of prioritizing bone health from early life.

BONE DEVELOPMENT AND PEAK BONE MASS

Bone formation during childhood and adolescence involves a coordinated process of modeling and remodeling. During growth, modeling predominates: osteoblasts (bone-forming cells) and osteoclasts (bone-resorbing cells) work on different surfaces to shape the bones and increase their size and mass. In childhood, bone mass increases steadily, then accelerates with the pubertal growth spurt. Peak linear growth and peak bone mass acquisition occur during adolescence, roughly coinciding with puberty. Girls tend to reach peak bone mass earlier than boys—one reference reported peak bone mass is achieved at a mean age of ~12.5 years in girls and 14.1 years in boys for certain skeletal sites.⁴ By the late teens or early twenties, most individuals have achieved their maximal bone density, after which a plateau and eventual slow decline ensue. The bone accumulated in those first two decades forms the structural foundation for the rest of the lifespan.⁵

Attaining a high peak bone mass is widely regarded as a critical protective factor against osteoporosis. It is estimated that a 10% increase in peak bone mass could translate to 50% fewer fractures in older adulthood (owing to the higher starting point before age-related bone loss begins).⁶ Therefore, factors that positively influence bone accrual in youth have long-ranging benefits. Genetics set the baseline potential—as noted, 60–80% of variance in bone density is genetically determined.⁷

For example, family history and ethnic background influence bone density (with African ancestry often associated with higher bone mass compared to Caucasian or Asian ancestry and thus lower osteoporosis risk in later life).⁸ However, the environmental component (20–40%) is substantial enough that optimizing those factors can make a meaningful difference. Nutrition and physical activity are the chief modifiable influences in this regard and are sometimes called “nutritional and exercise hormones” for bone due to their powerful effects. Hormonal milieu is another important determinant of bone development. Sufficient levels of growth hormone, IGF-1 and sex steroids (estrogen and testosterone) during puberty are necessary for the pubertal bone growth surge.⁹ Estrogen in particular, in both sexes, is pivotal for epiphyseal growth plate fusion and skeletal maturation.¹⁰

Conditions that disrupt normal puberty or cause hormonal deficiencies can lead to reduced bone mass. For instance, in adolescents with delayed puberty or amenorrhea (such as those with anorexia nervosa or the female athlete triad), bone accrual is often impaired due to low estrogen levels, resulting in lower peak bone mass and even osteoporosis at a young age. Chronic illnesses (like juvenile inflammatory diseases or endocrine disorders) and prolonged use of corticosteroids can also interfere with the bone-building processes, underscoring the need to monitor bone health in these high-risk groups.¹¹ The different peak bone mass determinants are illustrated in Table 1.

NUTRITIONAL FACTORS AFFECTING BONE HEALTH

Calcium intake

Calcium is the primary mineral constituent of bone and adequate intake during childhood is essential for proper mineralization of the collagen matrix in growing bones. Children have high calcium needs to support rapid skeletal growth the Recommended Dietary Allowances (RDA) for calcium are 200–260 mg/day in infants (0–12 months, as Adequate Intake), 700 mg/day for toddlers (1–3 years), 1,000 mg/day for children 4–8 years and 1,300 mg/day for those 9–18 years.¹²

These recommendations aim to cover the calcium required for bone accretion and maintenance of positive calcium balance during growth. Unfortunately, many children do not meet these targets. Dietary surveys in the United States, for example, show that mean calcium intakes among 2–19 years old range around 950–1,000 mg/day and nearly half of 4–18 years old children have calcium intakes below the Estimated Average Requirement (EAR) for their age.¹³

Globally, calcium intake varies widely. In some low- and middle-income countries where dairy consumption is low, calcium intakes can be well below recommendations contributing to persistent cases of nutritional rickets that are driven primarily by calcium deficiency (sometimes termed “calcipenic rickets”). Diets high in phytate or oxalate (from certain grains and vegetables) can also reduce calcium absorption, though typical mixed diets dilute these effects. Clinical and epidemiological studies generally support the importance of calcium for pediatric bone health.¹⁴

Vitamin D status

Vitamin D is indispensable for bone health because it facilitates intestinal absorption of calcium and phosphate. In children, vitamin D deficiency results in rickets, the classic bone disease of impaired mineralization. Vitamin D can be obtained from dietary sources (fatty fish, egg yolks, fortified foods and supplements) or synthesized in the skin upon exposure to ultraviolet B (UVB) sunlight. Cutaneous production is typically the major source;

however, factors like latitude, season, skin pigmentation, clothing and sun avoidance for cultural or health reasons (e.g., to prevent skin cancer) can all limit UVB-mediated synthesis. The serum concentration of 25-hydroxyvitamin D (25(OH)D) is the accepted indicator of vitamin D status. There is some debate on optimal levels for bone health, but deficiency is often defined as 25(OH)D below 30 nmol/L (which is associated with rickets/osteomalacia) and insufficiency as 30–50 nmol/L. Notably, consensus guidelines agree that levels below 25–30 nmol/L should be strictly avoided in all age groups.¹⁵

Vitamin D deficiency is common worldwide, even in sunny climates and affects children as well as adults. A recent global pooled analysis spanning 308 studies and over 7.9 million participants reported that 15.7% of the world's population had 25(OH)D <30 nmol/L and 47.9% had levels <50 nmol/L.¹⁶ Regions in the Middle East and South Asia showed particularly high prevalence for example, in the Eastern Mediterranean region, an estimated 35% of people have 25(OH)D <30 nmol/L and over 70% are below 50 nmol/L.¹⁷ Contributing factors include limited sun exposure (due to cultural dress, indoor lifestyles or high pollution blocking UVB) and low dietary intake in these areas.

In contrast, North America (Americas region) had a lower prevalence of severe deficiency (~5% <30 nmol/L), likely reflecting both fortification practices and supplement use. Even in high-income countries, vitamin D insufficiency is not rare for instance, national data from the United States indicate around 9% of children and adolescents have 25(OH)D <30 nmol/L and ~61% have <75 nmol/L.¹⁸

Subclinical vitamin D insufficiency can lead to decreased calcium absorption, secondary hyperparathyroidism and reduced bone mass accrual. Over time this may contribute to osteopenia and a higher fracture risk.¹⁹ Therefore, pediatric guidelines universally emphasize vitamin D sufficiency. A landmark policy statement by the American Academy of Pediatrics in 2008 (updated guidance) recommends that all infants, children and adolescents receive at least 400 IU of vitamin D daily, starting soon after birth.²⁰ This was an increase from earlier recommendations of 200 IU, reflecting new evidence of safety and benefit. For infants who are exclusively or partially breastfed (breast milk is low in vitamin D), this means providing oral vitamin D drops (400 IU per day).

Formula-fed infants generally receive vitamin D in fortified formula, but if intake is <1 liter of formula per day, supplementation is advised to reach 400 IU. Children over 1 year are advised to get 600 IU daily (as per Institute of Medicine recommendations), though the AAP stated 400 IU as a minimum baseline. Many other countries have similar policies, particularly for infants. In Canada and much of Europe, infant vitamin D supplementation (400–800 IU depending on locale and risk factors) is standard in the first year of life. Such preventative policies have been

effective in virtually eliminating infantile rickets where compliance is high.²¹

For older children and adolescents, ensuring adequate vitamin D often relies on a combination of diet (fortified milk and foods, oily fish intake) and sensible sun exposure. Approximately 5–15 minutes of mid-day sun on face, arms and legs a few times a week can produce substantial vitamin D in light-skinned individuals, though darker-skinned individuals require longer exposure. However, balancing sun exposure with skin cancer prevention is tricky; thus, oral intake remains a safe strategy. In practice, many adolescents may need vitamin D supplements (e.g., 600–1,000 IU daily) to maintain optimal 25(OH)D levels, especially in winter or if they live in higher latitudes. In regions with widespread deficiency, fortification of staple foods (like wheat flour or cooking oil with vitamin D) is being increasingly adopted as a population-level intervention.²²

Diet quality and other nutrients

Beyond calcium and vitamin D, overall diet quality in childhood has been linked to bone health. A systematic review examining diet quality indices and bone outcomes in youth found that children with higher adherence to balanced, nutrient-rich diets (for example, diets high in fruits, vegetables, whole grains, dairy and lean protein and low in sugars and processed foods) had better bone mineral density or content in the majority of studies.²³ These “healthy eating” patterns tend to provide not only calcium and vitamin D (via dairy and fish), but also protein (important for collagen matrix), magnesium, potassium, vitamin K and other micronutrients that support bone metabolism.

In contrast, Western-style diets high in soft drinks, refined carbohydrates and sodium may indirectly harm bone health by displacing more nutritious options. Some observational studies have raised concerns about high soda intake correlating with lower BMD in teenagers, possibly because cola drinks (rich in phosphoric acid) might alter calcium/phosphate balance or because they replace milk in the diet. However, the evidence is mixed and such effects appear more related to what soda displaces (milk) rather than a direct deleterious effect of phosphorus.²⁴ Protein is often under-appreciated in discussions of pediatric bone health. Ample protein is required for bone matrix production; extreme protein malnutrition (as seen in cases of kwashiorkor or anorexia nervosa) is detrimental to bone accrual. On the other hand, very high protein intake, especially without adequate calcium, can increase calcium excretion; but in children, high-protein diets usually also come with high dairy intake (hence high calcium), so net effects are usually positive for bone.²⁵ Other dietary factors include phosphate (generally sufficient in diets), vitamin K (found in greens, supports bone via its role in osteocalcin carboxylation) and trace minerals like zinc and copper (involved in collagen cross-linking). While each of these nutrients is rarely limiting to the point of clinical bone

disease in isolation, they contribute as parts of a holistic diet. Therefore, promoting an overall balanced diet rather than just calcium and vitamin D in isolation is advisable for robust bone development.²⁶ It is worth noting that nutritional needs and challenges can differ by age. In case of infants, they have unique nutritional considerations. They roughly double their bone mineral content in the first year of life.²⁷ Human breast milk, while the gold standard for infant feeding, contains relatively low levels of vitamin D and only modest calcium (though calcium absorption from breast milk is high).²⁸ Thus, exclusively breastfed infants depend on maternal vitamin D status and supplementation to avoid deficiency. Formula-fed infants receive added vitamin D and sufficient calcium/phosphate by design, reducing rickets risk in that group.

Preterm infants are at particular risk for bone mineralization problems (metabolic bone disease of prematurity) because they miss out on the third-trimester mineral accretion in utero; they often require higher calcium and phosphate intakes after birth and vitamin D supplementation of 400–800 IU/day is recommended for them.²⁹ In toddlers, young children may be at risk of inadequate calcium if they transition off breast milk or formula to diets low in dairy. “Milk refusal” toddlers or those on vegan diets need alternative calcium sources (fortified plant milks, tofu, etc.) or supplements.

Vitamin D supplements of 400–600 IU daily is commonly advised up to age 3–5 in many guidelines.³⁰ For the adolescents, Puberty brings a surge in skeletal growth and calcium needs peak at 1,300 mg/day. However, this is a time when many youths (especially girls) may reduce dairy intake due to dietary preferences or lactose intolerance. Calcium-rich alternatives and supplementation should be considered if intake is low. Additionally, gonadal steroid production (especially estrogen in girls) during mid-late puberty aids calcium incorporation into bone; undernutrition or extreme exercise can disrupt this, reinforcing how intertwined nutrition is with hormonal factors.³¹

Physical activity and bone health

Physical activity is the other critical pillar, alongside nutrition, supporting bone health in the young. Bones adapt to the loads placed upon them: when subjected to regular mechanical stress from muscle contractions and gravity (as occurs during exercise), bones respond by increasing formation, improving geometry and enhancing strength. This concept, rooted in Frost’s mechanostat theory, is well demonstrated in both animal models and human studies. In children, habitual weight-bearing physical activities correlate with greater bone density and cross-sectional area compared to sedentary lifestyles.³² Additionally, muscle development and bone development are interdependent muscles tug on bones, stimulating bone growth and bones provide attachment and leverage for muscle forces. This muscle-bone unit is sometimes referred to as the “muscle-bone axis,” and it includes

biochemical crosstalk (myokines and osteokines) in addition to mechanical interactions.³³

Cross-sectional and longitudinal evidence

Studies have consistently found that active children (those who participate in sports, play outdoors, etc.) have better bone outcomes. For example, cross-sectional studies using DXA scans show higher BMD in youth engaged in high-impact sports (gymnastics, basketball, soccer) or high muscle-strength activities (resistance training) compared to non-athletic controls.³⁴ Longitudinal studies also indicate that changes in activity level can modulate bone gain. One famous longitudinal study (the Iowa Bone Development Study) found that bone accrual during puberty was significantly associated with the amount of moderate-to-vigorous physical activity the children reported—those in the highest activity tertile accumulated bone at a faster rate than those in the lowest activity group, even after adjusting for maturation and body size.³⁵

Exercise intervention trials

More convincingly, intervention trials provide evidence of cause-effect. Numerous randomized controlled trials (RCTs) have introduced exercise programs to children and measured bone changes. A recent systematic review identified 12 RCTs of school-based physical activity interventions (many incorporating jumping, running and strength exercises) in children from early childhood to puberty. The findings were largely positive: interventions that added regular weight-bearing exercise (e.g., 10–20 minutes of jumping activities 3 times a week or daily physical education classes with impact activities) led to significant improvements in bone mineral content and bone structural properties at loaded sites (like the hip and lumbar spine) compared to usual curriculum.³⁶

Effects were most pronounced in pubertal children, presumably because of the growth spurt amplifying the response.³⁷ One illustrative study is the Bounce at the Bell trial in New Zealand, where elementary school children did 10 minutes of supervised jumping exercises twice daily; after one year, those children had greater increases in hip and spine BMD than children in control schools.³⁸ Another example is an intervention in Hong Kong teenage girls combining skipping (jump rope) and circuit training, which resulted in higher leg bone mass and improved bone geometry relative to controls after 8 months. These interventions are often low-cost and easily implementable through school physical education, suggesting a practical avenue to bolster bone health on a population level.³⁹

Type and timing of exercise

Not all exercises are equal for bone benefits. Impact (weight-bearing) activities and high-force, high-rate muscle contractions are key stimuli. Ground reaction forces from jumping, hopping, running and sports like basketball or volleyball or the strain from gymnastics,

provide the kind of dynamic loading that bone cells respond to.⁴⁰ In contrast, non-weight-bearing activities like swimming or cycling, while excellent for cardiovascular fitness, have less osteogenic impact (swimmers often have BMD similar to non-athletes, all else equal). Resistance training (lifting weights or body-weight resistance exercises) can also induce bone gains by increasing muscle forces on bone. A common recommendation is for children and teens to engage in multidirectional activities that

involve jumping and cutting movements, as these cause bone to experience forces from various angles, which may optimize bone shape and strength (for instance, playing soccer or basketball, which involve running, jumping and quick changes in direction, is beneficial). As children enter adolescence, incorporating some resistance training is also encouraged by pediatric and sports medicine guidelines, both for bone and overall musculoskeletal health (with proper supervision to ensure safe technique).⁴¹

Table 1: Determinants impact on peak bone mass.

Category	Determinants	Key mechanisms	Impact on peak bone mass
Genetic factors	Hereditary traits, ethnic background, family history of osteoporosis	Regulates bone geometry, size and remodelling rate	Accounts for ~60–80% of variance in bone mass; sets upper potential
Nutritional factors	Calcium, vitamin D, protein, phosphorus, magnesium, vitamin K	Supports mineralization, collagen synthesis and bone metabolism	Adequate intake promotes optimal bone accrual; deficiencies impair peak mass
Hormonal factors	Growth hormone, IGF-1, estrogen, testosterone, thyroid hormones	Stimulate bone formation, regulate growth plate closure and maintain bone turnover balance	Hormonal deficiencies during growth reduce peak bone mass
Physical activity	Weight-bearing and high-impact exercise, resistance training	Mechanical loading stimulates osteoblast activity and bone formation	Enhances bone density, geometry and strength, especially during peri-puberty
Body composition	Lean muscle mass, healthy body weight	Muscle forces provide mechanical stimulus to bone; adipose tissue affects hormonal milieu	Higher lean mass generally linked to greater peak bone mass
Lifestyle factors	Adequate sleep, avoidance of smoking and excessive alcohol	Supports metabolic and hormonal balance	Unhealthy lifestyle choices can hinder bone development
Health status	Chronic illnesses, endocrine disorders, malabsorption syndromes	Affect nutrient absorption, hormonal regulation and activity levels	Conditions such as juvenile arthritis or prolonged corticosteroid use reduce peak mass
Environmental factors	Sunlight exposure, socio-economic status, access to healthcare	Sunlight promotes vitamin D synthesis; SES influences diet and activity	Limited sunlight or low SES may reduce bone accrual potential

The timing of exercise relative to growth is noteworthy. The peri-pubertal years (approximately ages 10–14 in girls, 12–16 in boys) are when exercise yields the greatest bone gains sometimes described as a “use it or lose it” window. During this period, the rate of bone formation is high and evidence suggests that bones are extra responsive to mechanical challenges. Interventions initiated during early puberty have shown larger percentage improvements in bone outcomes than those in pre-pubertal or late-post pubertal subjects. Nevertheless, benefits from exercise are seen at all ages of childhood. Starting good activity habits early (in preschool and early childhood) not only directly benefits bone and muscle development, but also tracks into continued activity in later childhood, compounding benefits.^{42,43} Some studies indicate that even short-term exercise interventions in preschoolers (ages 3–5) can improve bone mass by a small amount, though the magnitude is less than interventions at puberty. Thus, a life-course approach is ideal: encourage physical play in

toddlers and children, structured and unstructured physical activities through childhood and robust exercise and sports participation during adolescence.⁴⁴ On the other side, physical inactivity is detrimental to bone. Extended sedentariness, such as immobilization or very low activity, leads to bone loss. This is starkly illustrated in clinical situations: children on prolonged bed rest or casting (e.g., for complex fractures or illness) lose significant bone mass in weeks. Astronaut studies (in adults) show the skeletal unloading of microgravity causes rapid bone loss; similarly, if children do not bear weight (as in conditions like cerebral palsy where mobility is limited), they have much lower bone densities and a higher fracture risk.⁴⁴ While most children thankfully are mobile, the concern is more about relative sedentariness high screen time, little vigorous play which can result in suboptimal bone gains. Public health data are worrisome: as mentioned, around 8 in 10 adolescents globally do not meet the recommended ≥60 minutes/day of moderate-to-vigorous physical

activity. Contributing factors include increased academic and electronic entertainment time, urban environments not conducive to play and in some cases, safety concerns limiting outdoor activity.⁴⁵

DISCUSSION/POLICY AND PRACTICE RECOMMENDATIONS

Based on this review, several actionable recommendations can be made ensure vitamin D supplementation of 400 IU/day for all infants worldwide and continue supplementation (400–600 IU) through childhood unless a child has regular safe sun exposure and dietary intake that clearly meets requirements. This could be implemented via maternal-child health programs, pediatric visits or school health services.⁴⁶ Emphasize dietary calcium intake through culturally appropriate foods. Where dairy is accepted, three servings of milk/yogurt per day for older children can meet needs. Monitor at-risk children (e.g., those on dairy-free diets). Develop and support school-based nutrition and physical activity programs: e.g., school lunches that provide a good source of calcium (cheese, milk or fortified alternatives) and vitamin D (fortified foods) and daily exercise opportunities during school hours. Schools can be bone health champions by building these into the curriculum.

Increase public and caregiver awareness about bone health

Many parents know about iron or general nutrition but may be less aware of vitamin D or the importance of exercise for bones. Public health messaging (through pediatricians, social media, parenting classes) should reinforce that giving the vitamin D drops or letting kids play outside (with sun protection as needed) are important for strong bones.

Surveillance and screening

Regions with known high rickets rates might consider routine vitamin D level screening or at least targeting screening in high-risk groups (like toddlers with delayed walking or children with developmental delays). Community surveys on vitamin D status and dietary intake can help identify problem areas. For children with conditions like cerebral palsy or leukemia (that predispose to low BMD), proactive monitoring via DXA might be indicated along with early interventions.

Encourage research and evaluation

Ongoing evaluation of current prevention programs (are they effectively reducing rickets? Is compliance with supplements high? etc.) is important. Also, more RCTs in diverse populations on supplementation (like trials of vitamin D in African or Asian children on fracture outcomes) would strengthen the evidence base, as most trials have been in Western populations with relatively low numbers of endpoints.

CONCLUSION

Bone health during childhood and adolescence forms the bedrock for skeletal well-being throughout life. This review elucidated that ensuring optimal bone health in the pediatric population hinges on a multipronged approach addressing nutrition, physical activity and early identification of bone-affecting conditions. Sufficient intake of calcium and vitamin D, from infancy onward, is fundamental for proper bone mineralization and the prevention of rickets and osteomalacia. Regular weight-bearing physical activity acts in synergy with nutrition to maximize bone density and strength gains during the growth years. When these modifiable factors are favorable, children are more likely to achieve a high peak bone mass, which confers protection against fractures and osteoporosis later in life.

Conversely, deficiencies in diet or sedentariness can lead to compromised bone development. The reappearance of nutritional rickets in both developing and developed regions is a cautionary example of how quickly pediatric bone health can be jeopardized if vitamin D or calcium needs are not met. Similarly, the widespread prevalence of low vitamin D status among youth and the under-exercise pandemic in adolescents, present urgent public health challenges that can be addressed through concerted strategies in healthcare, schools and communities. The review also highlights that bone health is not a siloed issue; it intersects with overall child health and nutrition. Efforts to improve childhood diet quality will support bone health while also combating obesity and micronutrient deficiencies. Encouraging an active lifestyle in children yields cognitive, cardiovascular and emotional benefits in addition to strengthening bones. Therefore, interventions targeting these areas have multi-faceted payoffs.

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