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IP International Journal of Orthopaedic Rheumatology

Journal homepage: https://www.ijor.org/

Review Article

A review on bone health optimization during early childhood

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Abstract

Bone mineral density, content, and remodelling are all influenced by diet and exercise, and these factors can affect bone health in later life. The proper composition of nutrients can affect bone health and maximize peak bone mass; the nutrition that youngsters receive may have long-term effects. Since bones grow quickly during childhood and adolescence and are most susceptible to environmental factors, this is the greatest time to avoid bone disorders. Normal bone mineralization is influenced by a number of factors, such as low nutritional intake, glucocorticoid exposure, inflammation, delayed maturation, limited physical activity, and malabsorption.

Keywords: Bone health, Osteology, Bone mineralization, Remodelling, Therapies.

Received: 07-12-2024; **Accepted:** 04-06-2025; **Available Online**: 06-06-2025

1. Introduction

Nutritional advice, bone mechanical loading, optimizing pubertal status, and treating underlying chronic diseases are all part of the complicated therapy of bone disorders. For some cases, particularly in children with genetic disorders, chronic glucocorticoids, and cancers, pharmacological therapies are being investigated. Bone disorders are a leading cause of global morbidity and economic burden, so childhood and adolescence are critical for maximizing bone health. Recent studies states that early bone mass is the most important modifiable factor affecting skeletal health throughout life. Bone diseases can occur in children and adolescents.[1]

Proper calcium intake, vitamin D levels, and physical exercise can prevent bone disorders in children. However, lack of data on bone mineralization limits prevention strategies. Impaired bone accumulation in childhood and adolescence increases fracture risk. Modifiable determinants include dietary status, hormones, chronic disorders, and medications.

2. Discussion

A dynamic organ, the skeleton is always changing due to the creation and resorption of bones. Circulating hormones and local cytokines control this process. Cortical bone remodelling in early infants can occur at a rate of 50% annually. The equilibrium between resorption and production determines net bone mass. Net bone mass rises when formation surpasses resorption and falls when resorption surpasses production. Collagen, non-collagenous proteins, and hydroxyapatite crystals make up the living structures known as bones. Calcium and phosphate deposits cause the matrix to mineralize, giving the structure strength. Pregnancy is the starting point for bone mineral deposition, with the third trimester accounting for two-thirds of in utero accrual.[2]

Between 40% and 60% of adult bone mass is formed throughout adolescence, with 25% of peak bone mass occurring during the two years preceding peak height velocity. According to Bailey,[3] the average age at which bone mineral accretion rates peak after infancy is 12.5 years for girls and 14.0 years for boys. Roughly 90% of maximal bone mass has been accumulated by the age of 18.[4] For this reason, childhood and adolescence are crucial times for bone mineralization. For both boys and girls, the age at which peak bone mass accumulation occurs lags the age at which peak

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http://doi.org/10.18231/j.ijor.2025.003

height velocity occurs by roughly 6 to 12 months.[3] About 70% of the variation in bone mass can be attributed to genetic causes,[5] while no particular genes have been found to be at fault. Black women have more bone mass than Asian or white non-Hispanic women, while male subjects had more bone mass than female ones. The bone densities of Mexican-American women are comparable to those of Black and white non-Hispanic women. The nutritional intake of calcium, vitamin D, protein, sodium, and carbonated beverages (such as soda), as well as lifestyle choices including exercise, hormonal balance, and maintaining a healthy body weight, are all modifiable factors that affect bone mass. Both physical exercise and proper nutrition are essential and work in concert to enhance bone growth and maintenance.

3. Factors Affecting Bone Health

3.1. Everyday diet

According to a new meta-analysis of 88 studies, drinking soda is linked to consuming less milk and calcium. Longitudinal and experimental studies showed greater effect sizes than cross-sectional studies. Adolescents may not be able to obtain enough calcium and vitamin D if they substitute soda for milk in their diet. Since soda has no health benefits, it should be avoided.[6] People who eat a lot of salt or little protein are more likely to have less calcium retention.[7,8] A high-sodium diet encourages increased excretion of calcium in the urine and should be avoided since sodium and calcium share the same transport route in the proximal tubule.

3.2. Bone calcium content

Bone development requires calcium, and the amount of calcium consumed through nutrition during infancy, youth, and adolescence influences the growth of bone mass. The skeleton contains about 99 percent of the body's calcium, which is absorbed through both passive and active transport—the latter of which is facilitated by vitamin D. Higher BMC and a lower incidence of fracture in adulthood are linked to milk consumption during childhood and adolescence.[9] Human milk, or infant formula in the event that human milk is unavailable, is the main source of sustenance for healthy term infants during the first year of life. The breastfed newborn shows no signs of a clinical mineral shortage, and there is no reason why the breastfed infant should not be the benchmark for bone mineral accretion, even though the BMC may be higher in formula-fed infants than in breastfed infants during the first year of life.

Milk and other dairy products make up 70% to 80% of dietary calcium intake after the first year of life, making them the main source of calcium. About 300 mg of calcium are included in each 8-oz (240 mL) serving of milk, and the calcium level of flavored or low-fat milk is comparable to that of whole milk. Green leafy vegetables, legumes, nuts, and morning cereals and fruit juices that have been fortified with calcium are additional dietary sources of calcium. Between 2000 and 2006, about 7% of the calcium in the food supply came from vegetables.[10] In spinach, collard greens, rhubarb, and beans, calcium binds with oxalates, reducing its bioavailability, which is normally high. It is challenging to achieve dietary calcium requirements with vegetables alone, even if they are a rich source of bioavailable calcium. This is because a significant amount of vegetables are needed to meet daily requirements. Phytates, which are present in some cereals (such as whole bran cereals), also lower bioavailability.

3.3. Level of vitamin D

A fat-soluble hormone called vitamin D (calciferol) is essential for the absorption and use of calcium. Only 10% to 15% of dietary calcium is absorbed in the absence of vitamin D.[11] Forms of vitamin D include ergocalciferol (vitamin D2) from plants, cholecalciferol (vitamin D3) that is consumed, and endogenous conversion of vitamin D2. Although the current study focuses on bone health, there is mounting evidence that vitamin D may also have implications for immunity, cancer prevention, and risk factors related to the metabolism and heart.

The skin converts 7-dehydrocholesterol into vitamin D3 when it is exposed to sunlight. After attaching itself to a protein called vitamin D-binding, it is transported to the liver, where it undergoes hydroxylation to produce 25-hydroxyvitamin D (25-OH-D). Serum 25-OH-D is a good indication of vitamin D storage and has a half-life of two to three weeks. In the kidney, circulating 25-OH-D undergoes a second cycle of hydroxylation to produce 1,25-OH2-D, the hormone's active form. Compared to 25-OH-D, 1,25-OH2-D has a 4-hour half-life.[12] Under PTH control, 1,25-OH2-D promotes intestinal absorption of calcium and phosphorus, mobilizes calcium from skeletal reserves, and enhances renal reabsorption of filtered calcium. Although there are few natural dietary sources of vitamin D, fortified foods, fatty fish (such as salmon, sardines, and tuna), and cod liver oil are good sources. Compared to fresh, wild-caught salmon, farm-raised salmon have lower vitamin D concentrations.[13]

3.4. Exercise and lifestyle

Walking, running, jumping, and dancing are more beneficial for bone health for the majority of kids and teenagers than swimming or riding a bicycle. However, excessive high-impact exercise can raise the risk of fractures. In comparison to less active girls, high school girls who engaged in more than 8 hours of weekly running, basketball, cheering, or gymnastics were twice as likely to have a fracture, according to a prospective longitudinal study of 6831 females. Field[14] recommended that females who play these sports cross-train in less strenuous activities as well. Lifestyle decisions may potentially increase the chance of BMD deficiencies. Alcohol, coffee, and smoking are all linked to lower BMD in

adults,[15] thus kids and teenagers should refrain from engaging in these activities.

3.5. Body weight

Two significant modifiable factors that affect bone mass are body composition and weight. Bone production is stimulated by mechanical loading during weight-bearing activities, and numerous studies have shown a clear correlation between BMD and BMI in both healthy adolescents[16] and adolescents with anorexia nervosa.[17] Although greater adiposity can potentially be linked to an increased risk of fracture, lean body mass is more significantly connected with BMD.[18,19] Therefore, it is advised to maintain a healthy body weight throughout childhood and adolescence in order to maximize bone health.

3.6. Hormonal status

Bone mass is influenced by several hormones. Women's BMD is maintained in large part by estrogen, and a lack of it is linked to greater bone resorption and a higher risk of fracture. While excess glucocorticoids both enhance bone resorption and hinder bone production, testosterone, growth hormone, and IGF-1 all aid in the development of new bone.

3.7. Bone health assessment

There are few longitudinal studies on the factors influencing children's bone health based on fracture incidence, even though bone health assessment is essential for assessing fracture risk in children. About 70% of bone strength is made up of skeletal fragility, which varies according to age, body weight, fracture history, and the force of an accident. Since bone mass makes up around 70% of bone strength, it can be used as a stand-in for bone health, acknowledging that a child's low bone mass does not always indicate a higher risk of fracture.

Because of its accessibility, accuracy, speed, and low radiation dosage, dual-energy x-ray absorptiometry (DXA) is the recommended technique for determining bone mass.[20] By dividing BMC by the area of the scanned region, DXA determines areal BMD and assesses bone mass.

For paediatric patients, the entire body and the lumbar spine are the suggested measurement locations. Bone disease is defined as a BMD that is 2.5 SDs or more below the young adult mean, while osteopenia is defined as a BMD that is ≥1 SD below the young adult mean. Since youngsters have not yet reached maximal bone mass, care should be exercised when interpreting DXA results. Children and adolescents with poor bone density continue to have low bone density throughout time, according to longitudinal studies that show a high degree of tracking over a three-year period.[21] Although there is little evidence to support this statement, paediatricians should perform a DXA to identify children and adolescents at risk for skeletal fragility fractures and to guide treatment options.[22] Although it has a high radiation exposure dosage, quantitative computed tomography measures real volumetric BMD. Because there is a dearth of paediatric reference data and low precision in the paediatric population, quantitative ultrasonography—a non-invasive technique for evaluating bone health—is challenging to interpret. It measures the speed of an ultrasonic wave as it travels over the surface of bone.

4. Disorders Linked to Children's and Adolescents' Decreased Bone Mass

Rare disorders with increased bone fragility, such as Turner syndrome, idiopathic juvenile bone disorder, and osteogenesis imperfecta, are best treated by paediatric endocrinologists, geneticists, and paediatric bone health specialists. However, general paediatricians are often the ones who treat children with chronic conditions. Reduced bone mass can be linked to several conditions, including cerebral palsy, childhood malignancies, inflammatory bowel disease, celiac disease, juvenile idiopathic arthritis, systemic lupus erythematosus, cystic fibrosis, and chronic renal failure.[23] Malnutrition, elevated metabolic needs, intestinal malabsorption, low body weight, hypogonadism, immobility, chronic inflammation with elevated cytokine production, and the consequences of long-term glucocorticoid medication are risk factors. Children with cerebral palsy are particularly at risk. According to one study, 26% of kids over ten years of age had a fracture, and 77% of kids with cerebral palsy had a femoral neck BMD z score below -2.0.[23]

Adolescents frequently suffer from eating disorders, and anorexia nervosa has been connected to lower bone mineral density and a higher risk of fractures.[24] Poor diet, low body weight, oestrogen insufficiency, and hypercortisolism are etiologic variables. The degree of BMD loss is closely related to the duration of amenorrhea and malnutrition. Boys with anorexia nervosa also have reduced BMD, which is linked to low testosterone levels.[25] Reduced bone mass can also be seen in patients with bulimia nervosa or partial eating disorders.[26] According to Nattiv,[27] the "female athlete triad" consists of decreased BMD, menstruation dysfunction, and low energy availability in female athletes.

Endocrine disorders such as excess glucocorticoids or PTH, hypogonadism, hyperthyroidism, or growth hormone or IGF-1 insufficiency are associated with low bone mass. Bone mass can also be adversely affected by several drugs, including proton pump inhibitors, chemotherapeutic medicines, and anticonvulsants. In the US, adolescent pregnancy rates have decreased due to the effectiveness of depot medroxyprogesterone acetate (DMPA). Long-term use, however, may result in decreased bone mass and hypothalamic suppression.[28] In 2004, a black box warning on its adverse effects was issued by the US Food and Drug Administration. While most adolescents have a minimal risk of fracture, stopping DMPA is linked to quick increases in bone mass. Despite warning about the potential effects on bone mineral density, the Society for Adolescent Health and Medicine advises continuing to use DMPA.[29]

5. Approaches for Increasing Bone Mass in Fracture-Risky Populations

Supplements are recommended for kids and teens who do not obtain enough calcium from their diet, even from foods that have been fortified. Common types of supplemental calcium are calcium carbonate (40 percent elemental calcium) and calcium citrate (21% elemental calcium). Calcium supplements come in liquid, tablet, and chewable formats. Calcium carbonate, which contains forty percent elemental calcium, and calcium citrate, which has twenty-one percent elemental calcium, constitute typical forms of supplementary calcium. There are palatable, pill, and liquid varieties of supplements that contain calcium.

Moreover, to treat vitamin D deficiency, a blood 25-OH-D concentration is measured; this is advised for patients who have a higher risk of bone fragility and those who frequently sustain low-impact fractures. For individuals with higher risk of fracture, some specialists strive for a blood 25-OH-D concentration greater than 30 ng/mL. Furthermore, bisphosphonates have been used to increase bone mineral density (BMD) and reduce fracture risk in children undergoing corticosteroid treatment,[30] connective tissue disorders,[31] as well as cerebral palsy.[32] However, there is ongoing debate regarding the use of bisphosphonates in children due to their long half-lives and potential side effects. Preventive measures to encourage adequate bone mineral accrual and restrict variables that encourage greater resorption are the focus of paediatric bone disease linked to chronic illness. Normalizing bone mineralization can be aided by early treatment of the underlying condition. Complete recovery of reduced BMD after the first 12 months of treatment has been linked to following a gluten-free diet. The reason why 30% of adult CD patients get bone disorders or do not recover from dietary changes and diagnosis is yet unknown. Any bone deficiencies in T1DM patients should be partially reversed by optimizing insulin dosages to enhance glycaemic management and reduce microvascular and inflammatory illness. All patients are urged to engage in regular weight-bearing exercise. Vitamin D supplementation is advised for all patients at risk for bone deterioration, and nutrition, particularly vitamin K intake, is maximized. Adults with T1DM, CF, or CD have been evaluated for bisphosphonate therapy.

A paediatrician's role in optimizing bone health is extremely important. During health maintenance visits, they should ask about the type and quantity of exercise, calcium and vitamin D supplements, non-dairy sources of calcium and vitamin D, soda use, and dairy intake. Adolescents need four servings of dairy products per day, while children ages three to eight should have two to three servings or the equivalent. Regular calcium supplementation for healthy kids and teenagers is not supported by the available data. Children one year of age and up should consume 600 IU of vitamin D every day. For the best bone health, weight-bearing exercises are suggested over swimming or cycling. Serum 25-OH-D concentration should be tested in individuals with decreased bone mass or recurrent low-impact fractures, but routine screening for vitamin D deficiency is not advised. Adolescent female subjects and medical disorders linked to decreased bone mass and increased fragility should be evaluated using a Direct Ultrasound Assessment (DXA). To promote bone mass in individuals with anorexia nervosa or the female athlete triad, adolescent athletes should be informed about the female athlete triad and refrain from administering oral contraceptives. Significant skeletal impacts of childhood chronic disease have been identified, and our understanding of the pathogenesis of compromised bone health has advanced. With a focus on bone health care in multidisciplinary care, the clinical approach is changing. Alternative treatments such as anti-RANKL antibody and anti-sclerostin antibody should be assessed, and research is required to determine the best indication, dosage, and duration for bisphosphonate therapy.

6. Conclusion

A comprehensive medical history is essential for children with bone problems or suspected ones, including factors like fractures, comorbidities, physical activity, and family history. Physical examinations should include anthropometry, joint laxity, scoliosis, limb abnormalities, and spine pain. Laboratory examinations of bone mineralization should be conducted. Chronic illnesses can lead to poor bone health, with reduced strength and potential increased fracture risk. Anti-bone disorder drugs should be considered when a child experiences a first fracture or series of low-trauma fractures. Nitrogenous bisphosphonates are commonly used to improve bone strength and BMD, but their effectiveness in fracture reduction is less clear.

7. Source of Funding

None.

8. Conflict of Interest

None.

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Cite this article: Dubey S. A review on bone health optimization during early childhood. *IP Int J Orthop Rheumatol.* 2025;11(1):17-21.