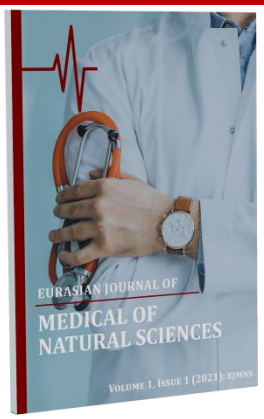
EURASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES
Innovative Academy Research Support Center
UIF = 8.3 | SJIF = 5.995 www.in-academy.uz



DIFFERENCES IN BONE COMPOSITION BETWEEN CHILDREN AND ADOLESCENTS

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https://www.doi.org/10.5281/zenodo.10430274

ARTICLE INFO

Received: 18th December 2023
Accepted: 24th December 2023
Online: 25th December 2023

KEY WORDS

Bone Composition, Skeletal Development, Growth Plate, Mineralization, Calcium and Phosphorus, Bone Remodeling, Osteoclasts and Osteoblasts, Puberty, Hormonal Influences, Peak Bone Mass, Nutrition, Physical Activity, Bone Density, Clinical Implications, Growth Spurts.

ABSTRACT

This article delves into the nuanced transformations in bone composition between children and adolescents, examining the dynamic processes that underlie skeletal development. It explores the pivotal role of the growth plate in childhood bone formation, the mineralization dynamics involving calcium and phosphorus, and the shift towards bone remodeling during adolescence. Hormonal influences, particularly during puberty, and the attainment of peak bone mass are discussed, shedding light on the critical milestones in bone development. Lifestyle factors, including nutrition and physical activity, are highlighted, and the clinical implications of understanding these differences are addressed, emphasizing the importance of monitoring and optimizing bone health during these formative stages.

The journey from childhood to adolescence is marked by profound changes in various aspects of human development, and bone composition is no exception. Bones, the scaffolding of the body, undergo dynamic transformations during these critical stages, shaping the foundation for future health and well-being. This article explores the key differences in bone composition between children and adolescents, shedding light on the intricate processes that contribute to skeletal development.

Introduction. Senile osteoporosis begins as a pediatric disease. This seeming paradox is rooted in the fact that there is intense skeletal growth and development during childhood and adolescence, and much more bone is formed than lost. Later in life, the loss of bone tissue exceeds the rate of bone replacement. It, therefore, follows that lifelong bone health is dependent on maximizing peak bone mass during the critical periods of growth and maturation. One commonly cited notion is that each individual possesses a “Bone Bank,” in which early deposits lay the foundation for skeletal health; later, during aging or in response to metabolic stresses, skeletal remodeling accelerates and withdrawals from the account exceed deposits, thereby compromising skeletal integrity. The natural process of bone remodeling makes youth the best time to “invest” in one's bone health.

Skeletal development and growth

Development and growth of the skeleton occur through the coordinated interaction of osteoblasts and osteoclasts. Osteoblasts are bone-forming cells, and are derived from pluripotent mesenchymal stem cells that can also differentiate into muscle, adipocytes, cartilage, or fibrous tissue. Bone is resorbed by osteoclasts, large, multinucleated cells that can dissolve mineral and release calcium and phosphorus into the extracellular fluid. Osteoclasts are related to monocyte/macrophage cells. During embryogenesis, and after birth as the child grows, the skeleton continues to undergo changes in architecture and size that are termed "modeling," with coordinate increases in bone mineral mass and density. These changes are achieved through sustained modeling through puberty. In addition to modeling, bones are continuously reshaped by removing and replacing skeletal structures already present, a process termed remodeling.

Exquisitely complex cross-talk between osteoblasts and osteoclasts is required to effect these changes through the remodeling of existing bone and modeling of new bone as bone growth proceeds. Linear growth during childhood and adolescence occurs by growth of cartilage at the end plates of long bones, followed by endochondral bone formation. The width of the bones increases by periosteal apposition. During puberty and early adult life, endosteal apposition and trabecular thickening provide maximum skeletal mass and strength (peak bone mass). Locally and systemically produced factors and mechanical forces influence these processes and control the coordinated functions of osteoblasts and osteoclasts to preserve structural strength. The adult skeleton continues to undergo remodeling throughout life, replacing approximately 15% of the mature skeleton each year to maintain mineral homeostasis, to repair damaged bone, and to respond to changes in skeletal stress.

Bone remodeling occurs most often in skeletal sites rich in cancellous (trabecular) bone, such as the vertebrae, proximal femur, calcaneus, and ultradistal radius. A second form of bone, termed cortical bone, is less metabolically active but provides great strength and integrity to the skeleton. Cortical bone comprises 80% of the skeleton, is dense and compact, and constitutes the outer part of all skeletal structures.

During the first two decades of life, the skeleton grows in both size and density, and it is estimated that more than half of peak bone mass is acquired during the teen years. The process of bone growth is not uniform, and the axial and appendicular skeleton increase in size at different rates. Specifically, there is proportionately greater growth in the limbs than the trunk prior to puberty. In early and mid-puberty, the relative rate of growth of the spine increases and growth slows at all sites in late puberty. The rapid growth of the skeleton during puberty exceeds the rate of mineralization, and bone mineral accrual lags behind growth in height by 8 months

Bone mass continues to accumulate until around age 30. At that point, bones reach their maximum strength and density, known as peak bone mass. Women tend to experience minimal change in total bone mass between age 30 and menopause. But, in the first few years after menopause, many women experience a period of rapid bone loss, which then slows but continues throughout the post-menopausal years. This loss of bone mass can lead to osteoporosis, a condition of weakened bones and increased risk of fragility fracture. In men, age-related bone loss occurs later and proceeds at a steady rate. In both men and women, declining bone mass is the primary cause of weak bones and fractures. It is estimated that 10

million Americans over age 50 have osteoporosis, which represents significant health and economic costs to society. If osteoporosis has origins in childhood, then understanding the factors affecting bone accretion in childhood may be the key to early prevention of this common, disabling condition.

Why Do Kids' Bones Need to Be Treated Differently?

An adult's bones are harder, more brittle and more likely to break than bend. A child's bones are more flexible because their chemical composition is different from that of adult bones. This means a kid's bone might bend or "bow" instead of breaking. Bent bones can actually be harder to treat than broken bones, but pediatric orthopedic specialists are trained to treat this type of bone injury, too.

Pediatric orthopedic specialists can often **treat kids' fractures** with a cast and avoid surgery. Because kids' bones heal more rapidly and many of the orthopedic devices and braces used to treat broken bones don't fit kids very well, the pediatric orthopedic specialists at Children's use techniques and devices designed for growing bones. This often means less time in a cast or splint and fewer surgeries than for an adult.

After your child breaks a bone, her emotional care can be just as important as the physical care she receives. A pediatric orthopedic specialist understands the whole kid—like how scary it can be to put on or remove a cast or how sad it can feel to not be able to play a favorite sport for a short time—and can help your child feel at ease as her broken bone heals.

How Do Kids' Bones Change Over Time?

When babies are first born, some of their "bones" are actually made up of a flexible cartilage (a firm tissue softer than bone). As the child grows, some of the cartilage hardens and turns to bone, and some bones fuse together.

Your child's bones won't stop growing until her late teens or early 20s. This is both an advantage and a disadvantage. As a child's bone grows, it is likely to remodel and realign itself. This means if a broken bone is crooked, it can straighten itself out over time. On the other hand, if a break occurs in the growth region at the end of a bone, also called the growth plate, or physis, it may affect normal growth and cause significant deformity of the limb. A pediatric orthopedic specialist is trained to recognize and care for these types of injuries.

THE PEAK BONE MASS

THE FOUNDATION FOR LIFELONG skeletal health is established during childhood and adolescence. Although there is controversy regarding the exact timing of peak bone mass, bone size and strength reach a maximum by early adulthood . Failure to accrue optimal peak bone mass has been linked to an increased risk of osteoporosis. The variables that contribute to optimal bone health have been delineated in studies of healthy youth .

Approximately 90% of adult bone mass is gained in the first two decades of life. Optimizing peak bone mass and bone strength early in life and stabilizing it during young adulthood is believed to play a significant role in preventing osteoporosis and fractures later in life. Adequate weight-bearing physical activity, nutrition, body mass, and hormonal balance are essential in achieving optimal skeletal health. A growing list of chronic diseases has also been linked to low bone mass or fragility fractures. Disorders causing rickets and osteomalacia are reviewed by Durval elsewhere in this issue. In some chronic conditions, a single factor (e.g., immobilization or hypogonadism) accounts for the increased risk of low

bone mass. In most of these disorders, however, skeletal health is threatened by a combination of risk factors including malnutrition, vitamin D insufficiency, malabsorption, deficiency or resistance to sex steroids or growth hormone, immobilization, and increased cytokine production. Medications that are used to treat these disorders, such as glucocorticoids, calcineurin inhibitors, and chemotherapeutic agents, may also contribute to bone loss . The magnitude of effect that these disorders or medications will have on an individual patient varies, depending upon genetic factors, disease severity, activity, and other variables. For this reason, clinicians seek diagnostic tools to identify patients at greatest risk for bone fragility.

Obese and less-active children also have been shown to have decreased BMD or bone mass compared with non-obese children of similar weight . It is not clear whether this decreased BMD among obese children is a direct effect of fat on bone or due to decreased muscle mass or reduced activity levels, or a combination of both of these factors. However, the epidemic of childhood obesity may in part directly or indirectly explain the increase in childhood fracture incidence that has recently been reported . Identifying children with low bone mass early in life could be an important strategy for preventative or therapeutic efforts to optimize bone accrual and, consequently, bone strength

ASSESSING PEDIATRIC BONE HEALTH

DXA is the most widely used densitometric method for diagnosing osteoporosis in adults. DXA was developed in the late 1980s for use primarily in postmenopausal women. Pediatric software became available in the early 1990s after improvements in algorithms for detecting bone edges in children with low bone density. The advantages of DXA are its wide availability, short scanning times, and relatively low radiation exposure. The radiation exposure is comparable to that received during a round trip transcontinental airplane flight. DXA has several important limitations, however . The technique does not provide a measure of volumetric bone mineral density or of bone geometry nor does it distinguish between cortical and trabecular bone. Although bone size and geometry can be adjusted for mathematically, these are only estimates of these parameters. Because this is a 2-dimensional measurement and not a true volumetric density, measurements using DXA are often referred to as *areal BMD* (aBMD). Measurements of aBMD are influenced by bone size, with larger bones having artificially inflated aBMD measurements . This is an important problem in pediatric bone assessment because of the large differences in body size and bone size within and across different ages. Studies show that aBMD by DXA increases with age, but computed tomography evaluations indicate that true volumetric BMD (vBMD) is relatively constant during childhood until puberty, at which time there is a large increase in vBMD .

BMC increases with age, and the increase in aBMD that is observed is likely the result of greater bone size.

1. The Blueprint of Growth: Childhood Bone Formation

In childhood, bones are in a state of active growth and development. The primary driver of this process is the growth plate, a region of cartilage at the ends of long bones. Here, new bone tissue is generated, contributing to the lengthening and development of the skeletal system. Children experience rapid bone growth, especially during growth spurts, as the body prepares for the challenges of adolescence.

2. Mineralization Matters: Calcium and Phosphorus Dynamics

Mineralization is a critical aspect of bone health, and during childhood, the focus is on building a robust framework. Calcium and phosphorus are the primary minerals involved in this process. Adequate intake of these minerals through a balanced diet is crucial for ensuring the optimal mineralization of bone tissue. Proper mineralization during childhood sets the stage for the increased bone density required in adolescence and adulthood.

3. Transition to Adolescence: Bone Remodeling Takes Center Stage

As children enter adolescence, there is a shift in the dynamics of bone development. Bone remodeling becomes a prominent process, involving the continuous breakdown and rebuilding of bone tissue. This dynamic equilibrium is orchestrated by specialized cells called osteoclasts and osteoblasts. Osteoclasts break down old or damaged bone, while osteoblasts form new bone in its place. This remodeling process enhances bone strength and adaptability.

4. Hormonal Influences: The Role of Puberty

The onset of puberty brings about significant hormonal changes, notably an increase in sex hormones like estrogen and testosterone. These hormones play a pivotal role in bone development by influencing the activity of osteoclasts and osteoblasts. During adolescence, estrogen promotes the closure of growth plates, signaling the end of linear bone growth. The intricate dance of hormones ensures the proper shaping and strengthening of the skeletal structure.

5. Peak Bone Mass: A Crucial Milestone

Adolescence is a critical period for achieving peak bone mass—the maximum amount of bone tissue an individual can attain. Building a strong foundation during this time is essential for preventing osteoporosis and fractures later in life. Factors such as nutrition, physical activity, and hormonal balance contribute significantly to the attainment of peak bone mass.

6. Lifestyle Factors: Nutrition and Physical Activity

Nutrition plays a pivotal role in bone health throughout childhood and adolescence. Adeuate intake of calcium, vitamin D, and other essential nutrients is crucial for supporting bone development. Engaging in weight-bearing physical activities, such as running or jumping, is equally important, as it stimulates bone remodeling and strengthens the overall skeletal structure.

7. Adapting to Change: Bone Density and Structure

Bone density and structure undergo notable changes during the transition from childhood to adolescence. While childhood bones are characterized by rapid growth and mineralization, adolescent bones exhibit a more intricate and denser structure. The increased bone density is a result of the ongoing remodeling process and the accumulation of mineral deposits within the bone matrix.

8. Clinical Implications: Addressing Challenges

Understanding the differences in bone composition between children and adolescents has clinical implications. Healthcare providers may monitor bone health during these developmental stages, especially in cases where nutritional deficiencies, hormonal imbalances, or growth-related concerns are present. Early intervention and lifestyle modifications can help address potential challenges and optimize bone health.

Conclusion: The journey from childhood to adolescence represents a pivotal chapter in skeletal development. Bones, ever-adaptable and responsive, undergo a series of orchestrated changes to meet the evolving needs of the growing body. Navigating these differences in bone composition provides valuable insights for parents, healthcare professionals, and educators, highlighting the importance of fostering optimal bone health through nutrition, physical activity, and a supportive environment. As children and adolescents embark on their unique paths of growth, the intricacies of bone development lay the foundation for a future of strength, resilience, and overall well-being.

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