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Bone mineral density in children from anthropological and clinical sciences: a review

Bernadette M. Manifold

The Mews, Darley Abbey, Derby, DE22 1AG, Derbyshire, United Kingdom

Abstract: Bone mineral density (BMD) is a frequent topic of discussion in the clinical literature in relation to the bone health of both adults and children. However, in archaeological and/ or anthropological studies the role of BMD is often cited as a possible factor in the poor skeletal preservation which can lead to an under-representation of juvenile skeletal remains. During skeletal development and growth throughout childhood and adolescence changes take place in both the size and shape of bones and these changes also result in the increasing of mineral content. BMD can be affected by many factors, which include, age, genetics, sexual maturation, amount of physical activity and dietary calcium. This paper aims to review the clinical and anthropological literature on BMD and discuss the numerous methods of measurement and how the availability of certain methods such as Dual-energy x-ray absorptiometry (DEXA) and quantitative computed tomography (QCT) can influence the study of bone density in archaeological skeletal collections and also the future potential for forensic anthropological studies.

Key words: Juvenile skeletal remains, bone health, bioarchaeology, DEXA, forensic anthropology

Introduction

Bone mineral density (BMD) is a frequent topic of discussion in the clinical literature in relation to the bone health of both adults and children. Bone health in children is a rapidly growing area of clinical concern; this interest is a response to the increase in childhood fractures as well as the concept that early bone development could be a major determinant of adult osteoporosis fragility fractures (Sawyer and Bachrach 2007; Rizzoli et al. 2010). However, in bioarchaeology the role of BMD is frequently discussed as a possible factor in the poor preservation or under-representation of children's skeletons in the burial environment. More recently, several studies have focused on the potential use of BMD in forensic anthropology in relation to the ageing of the adult skeleton (Fernández Castillo and López Ruiz 2011; Curate et al. 2013), also in paleopathological studies (Curate 2014).

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Throughout childhood and adolescence, the skeleton changes in both size and shape (Bianchi 2007), resulting in an increase in the length of bones via endochondral growth and an increase in width occurs through the oppositional growth of bone (Mays 1999). There will also be a gradual increase in the cortical thickness of bone, especially the lower limb and thus leading to an increase in both bone mass and density. The growth of the human skeleton is influenced by factors which control bone cell activity (i.e mechanical, genetic and environmental) (Smith and Wordsworth 2005).

Up to a quarter of peak bone mass (PBM) is acquired during the two years of peak height velocity. PBM can be defined as the total amount of bone tissue amassed by the end of skeletal maturation. Although it is estimated that 80-90% of PBM is acquired in the first two decades of life, studies have revealed a site specific phenomenon that varies with the unit of measurement, for example, BMD of the proximal femur peaks by the age of 20 years, whereas total body bone mineral content (BMC) peaks at around 30 years of age (Matkovic et al. 1994; Bonjour et al. 1991). At least ninety percentage is required by the age of 18 years, while the remaining 10% is added later during the skeletal consolidation phase (Bailey et al. 1999). Sex differences are thought not to become apparent until puberty. However, Specker et al. (1987) in a study of children aged between one and six years of age, observed that bone mineral content was lower in girls than in boys. The starting age of the pubertal spurt and growth process are earlier in girls, but the length of the growth spurt and the maximum peak of growth are greater in boys (Bianchi 2007). Height is thought to be the best indicator for bone mass in growing children. Different anthropometric variables appear to have differential effects on the skeletal development of children (Miller et al. 1991). Around 85% of the skeleton consists of cortical bone and 15% of trabecular bone. Trabecular bone density is influenced by hormonal and metabolic factors associated with sexual development. Peak bone mass (PBM) is reached in trabecular bone towards the end of the second year of life. Whereas the PBM is reached later in cortical bone. But overall growth is subject to great individual variability (Bianchi 2007).

Osteoporosis is a worldwide epidemic, affecting approximately 75 million people in the United States, Europe and Japan (EFFO and NOF 1997). Osteoporosis can be defined as a disease characterised by low bone mass, microarchitectural deterioration of bone tissue, and a consequent increase in fracture risk (EFFO and NOF 1997). In adults, a history of prior fractures is associated with around 86% increased risk of fracture at any site (Kanis et al. 2004), and as a result there has been much attention given to low bone mass in children and adolescents and especially with regard to the development of osteoporosis, which can be defined as low bone mineral density with microarchitectural alterations of bone, where there is increased bone fragility and greater risk of fractures (Bianchi 2007). Osteoporosis was redefined in 2000 as a skeletal disorder characterized by compromised bone strength, predisposing to an increased risk of fracture, there was emphasis on the importance of factors in addition to bone mass that contribute to bone strength (EFFO and NOF 1997). Dent (1973) described osteoporosis as a disease of adulthood with its roots in childhood. According to this model, bone mass achieved by early adulthood is a key determinant of

Table 1. Published data on the normative bone mineral density in healthy children from different geographical regions

Population examined	Sample size	Age	Sex	Ancestry	Body location	Method/technique	Reference
African American	130	1–139 days	59 F; 71 M	63 Caucasian/67 African American	Whole body density	DEXA	Koo et al. 1996
American	218	1–19 years	134 F; 84 M	Black /Caucasian	Lumbar Spine	DEXA	Southard et al. 1991
American	148	8–18 years		Caucasian	Whole body density	DEXA	Maynard et al. 1998
American	336	6–11 years	172 F; 164 M	Asian/ Black / Caucasian	Whole body density	DEXA	Horlick et al. 2000
American	468	4–12 years		Caucasian	Whole body density	DEXA	Janz et al. 2007
American	89	1–6 years	241 F; 227 M	Caucasian	Distal Radius	Direct photo absorti-omety/DEXA	Specker et al. 1987
Argentinan	778	2–10 years		Caucasian	Whole body density	DEXA	Zanchetta et al. 1995
Australian	183	8–16 years	433 F; 345 M		Spine, hip, whole body density	DEXA	Foley et al. 2009
			67 F; 116 M				
Australian	145	6–15 years		Caucasian	Femur	MRI/DEXA	Högler et al. 2003
Canadian	234	8–16 years	94 F; 51 M	Caucasian	Whole body density	DEXA	Faulkner et al. 1993; 1996
Danish	343	18 years	124 F; 110 M	Caucasian	Whole body density	DEXA	Mølgaard et al. 1997
Dutch	500	4–20 years		Caucasian	Whole body density/ lumbar spine	DEXA	Boots et al. 1997
			219 F; 155 M				
German	371	6–23 years		Caucasian	Whole body density	CT; DEXA	Neu et al. 2001
Japanese	155	23–41 weeks	295 F; 205 M		Right forearm	DEXA	Hayasti et al. 1996
Lebanese	363	10–17 years			Lumbar Spine, femoral neck	DEXA	Arabi et al. 2004
Polish and Spanish	108	Pre-term and full–term	186 F; 185 M		Whole body density	DEXA	Pludowski et al. 2010
			70 F; 85 M				
			179 F; 184 M				
			35 Polish; 74 Spanish				

the risk of developing osteoporosis and fragility later in life.

As a result many methods of measurement and reference samples are widely available within the clinical literature (Table 1). With regard to the measurement of BMD in past populations, several studies have been conducted on adult skeletons (Elenman et al. 1995; Holck 2007; Lynnerup and Von Wowern 1997; Mays 2001; Mays et al. 2006) and some juvenile samples (McEwan et al. 2005; Willey et al. 1997; Bennike et al. 2005; Manifold 2008; Kendell and Willey 2013). This paper aims to review the literature on bone density in children from clinical and anthropological contexts.

Bone biology

Juvenile bone is more porous and less dense and consists of a larger number of vascular channels than that of adult bone. It has a comparatively lower modulus of elasticity, lower bending strength and lower mineral content (Currey 2006). The skeleton is comprised of two tissue types: cortical bone and trabecular bone. The cortical bone forms the outer layer of the skeleton and forms the thick dense walls of the long bones, whereas the trabecular bone is formed as a honeycomb network of the bone tissues located as at the epiphyseal ends of the long bones and within the areas of the axial skeleton such as the vertebrae. Bone development arises from the differentiation of mesenchymal tissue in one of two ways (Scheuer and Black 2000). Bone can develop directly in mesenchyme via a process known as intramembranous formation. A second type, called endochrondral formation, is where a cartilaginous template for a future long bone is formed as an intermediary element and this is then destroyed and replaced by bone (Scheuer and Black 2000). Generally the bones of the calvarium and face are formed by intramembranous formation and it is thought that such bone formation occurs more rapidly in order to cover the developing brain (Scheuer and Black 2000). The remaining bones develop endochondrally (Fig. 1).

Bone mineralisation is the incorporation of minerals such as calcium and

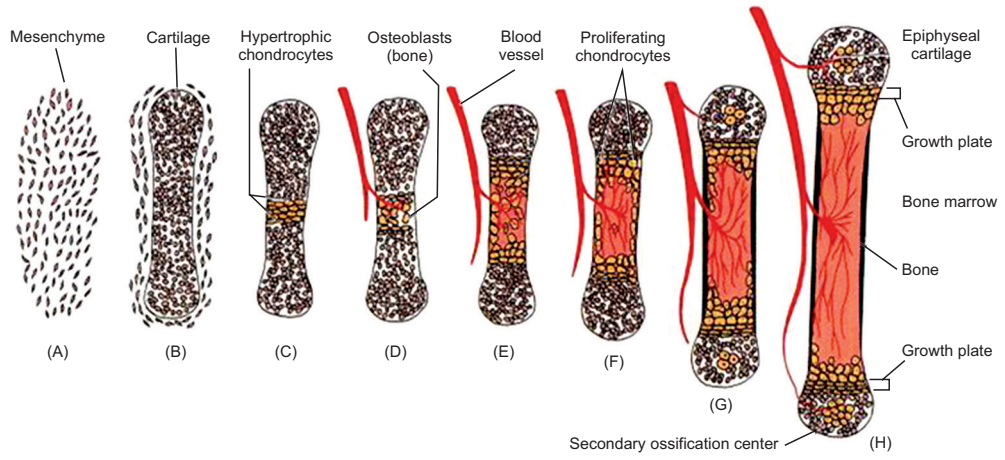

Fig. 1. Endochondral bone formation

phosphate into organic bone matrix, after it has been synthesised and deposited by osteoblasts. The term mineralisation can mean one of two processes, depending on the circumstances. Firstly, an increase in arenal bone mineral density or bone mineral content which occurs after the incorporation of mineral into pre-existing bone matrix, but can also result from an increase in bone size, thickening of the bone cortex, or the synthesis of new trabeculae bone. This latter process represents the formation of new bone tissue, where the incorporation of mineral into organic matrix is just one of several steps. The main point here is that mineralisation can only occur where organic matrix has previously been deposited. Therefore, what is commonly called 'decreased bone mineralisation' suggests that, either not enough organic matrix has been deposited, or not enough mineral is being incorporated into the matrix (Rauch and Schoenau 2002; Smith and Wordsworth 2005).

The amount of bone in the skeleton increases with growth. This growth reaches a plateau in young adulthood and subsequently falls with increasing age. When density is analysed in bone material, this is often called 'true bone density' as the material density in the volume occupied by bone matrix does not include marrow spaces or osteonal canals (Rauch and Schoenau 2001). It has been found that BMD decreases in the first month after birth, which is followed by a rapid increase during the next two years of life with slower changes thereafter (Rauch and Schoenau 2001). Guy and colleagues (1997) found that bone density and mineral content decreased after birth maintaining a minimum value during the first year of life. Trotter (1971) found an early and rapid increase in bone density from birth to around 5 years of age followed by a gradual decline, which continued into the early twenties. There may be a change in the growth pattern of bones with no epiphysis fused and those with epiphyses fused, and the densities of different parts of the same bone may be different due to ossification at the primary and secondary centres (Trotter 1971).

Bone mineral density reflects the degree of mineralisation of the organic bone matrix, and this varies in every bone. Material BMD is the average of a continuum of density values. The bone matrix has a density of zero when it is released from the osteoblasts and mineralisation starts two weeks later at a remodelling site. A few days after the start of mineralisation, inorganic material has filled 75% of the matrix volume which was previously occupied by water molecules (primary mineralisation). Within six months, mineral continues to be incorporated slowly into the matrix (secondary mineralisation). Because of this time-dependent increase, the recently deposited bone matrix has a lower mineral density than old matrix. Material BMD is inversely related to bone remodelling activity. When remodelling activity is high, there will be more unmineralised osteoid and 'young' bone matrix, which have not yet completed secondary mineralisation (Rauch and Schoenau 2001).

Age

Age is thought to have a major influence on BMD. Trotter and Hixton (1974) carried out a survey of 427 human skeletons ranging from 16 weeks' gestation to 100 years of age. They recorded an increase in bone density between 30 weeks before birth and 20 years of age. This showed a steady increase with a sudden drop fol-

lowing birth and a gradual increase until adulthood. Trotter and Hixton (1974) have suggested that this is due to bone development as a result of a rapid growth of bone size, without a concordant increase in bone weight. This may be due to the temporary drop in calcium levels and calcium: nitrogen ratio in very young bone (Dickerson 1962). Similar findings have been made for the bones of dogs (Burns and Henderson 1936), pigs (Dickerson 1962), and cats (Burns and Henderson 1936).

Bone mass in infancy

Both birth weight and infancy have been shown to have an effect on bone mass during childhood and adulthood (Godfrey et al. 2001). Fall and colleagues (1998) stated that the growth hormone insulin-like growth factor-1 (IGF-1) axis, might be affected by an adverse intrauterine environment, and that alteration in these axes may result in different rates of skeletal growth and loss. Alternatively, these may be the consequences of altered skeletal development *in utero*. In their study of neonatal bone mass in a population-based cohort of healthy term deliveries, Godfrey and colleagues (2001) found that low parental birthweight and maternal cigarette smoking during late pregnancy were associated with a low neonatal bone mass. Factors such as calcium metabolism of the mother and fetus can influence the normal and accretion of bone *in utero* or after, may affect the amount of BMD present at birth. The majority of bone is gained during the last trimester, and another factor affecting BMD at birth is gestational age (Specker et al. 2001). Conditions that affect fetal growth can lead to changes in the metabolism of type I collagen; Namgung and colleagues (1996) examined if alterations type I collagen metabolism could cause low BMC in small-for-gestational-age-infants (SGA). They found that cord serum markers of type I collagen synthesis and degradation did not differ between SGA and appropriate-for-gestational-age (AGA) infants, concluding that low BMC in SGA and AGA infants reflected low mineral supply rather than defective collagen metabolism. Infants born prematurely have lower BMC than term infants. Several studies have reported normative data for preterm and term infants (Koo et al. 1996; Brunton et al. 1993; Braillon et al. 1992; Brunton et al. 1997). Minton and colleagues (1979) reported differences in BMC in both term and preterm infants using photon absorptiometry. It was found that BMC at birth was correlated significantly with gestational age and birth weight. However, subsequent measurements during the first three months showed that the postnatal increase in BMC was significantly less than the BMC expected *in utero*. It was speculated that the decrease intake of calcium and phosphate affects post-natal bone mineralisation in premature infants (Minton et al. 1979).

Postnatal development of total body mineralisation and regional bone mineralisation during infancy has been investigated by Koo and colleagues (1998) and related to anthropometric measurements and other physiological variables in infancy. It was found that in infancy, average body BMD increased by 389%, and total body BMD increased by 157%. The best determinant of bone mineral is body weight accounting for 97% of total body BMD, 98% of total body areas and 86% of total body BMD variation (Koo et al. 1996; 1998). There is a different relationship between bone mineral and body

mass for infants and abnormal versus normal bone mineralisation. Total body bone mineral content (TBBMC) was found to be normal in small preterm infants, and significantly higher in infants with congenital osteopetrosis when compared with healthy infants with similar body mass (Key et al. 1984; Koo et al. 1995). Normative data for BMC in preterm and term infants are limited due to problems in comparing results.

Bone mass in puberty and post-puberty

Before puberty, no sex differences in bone mass of both the axial and appendicular skeleton has been reported (Bonjour and Rizzoli 1998). These authors also report no sex differences in bone mass at birth and volumetric BMD which appears similar between female and male newborns. This absence remains until the onset of puberty. During puberty differences do occur. These differences are thought to be due to a more prolonged bone maturation period in males than females, with a larger increase in bone size and cortical thickness. Puberty affects the bone size rather than the volumetric mineral density. There is no significant sex difference in the volumetric trabecular density at the end of pubertal maturation. During puberty, the accumulation rate in arenal BMD at both the lumbar spine and femoral neck levels increase four to sixfold in males and females (Bonjour and Rizzoli 1998).

Horlick et al. found a small but significant pre-pubertal sex difference in total body BMC. Males have a greater total bone BMC than females, and have relatively leaner and less fat mass. A puberty related increase in BMD is frequently reported at the lumbar spine. Between 20–34% was observed in both sexes (Rauch and Schoenau 1998).Whereas Gilsanz and colleagues (1988; 1991; 1994) noted a 15–20% increase in lumbar spine BMD in both sexes, but this figure may be less than 20% for Caucasians. The reason for this increase remains undetermined, but hormonal influences and muscle strength appear a likely cause. The timing of puberty has also been investigated in relation to BMD in later life. Some studies in adult women found an inverse relationship between the age at menarche and BMD, especially at sites with a predominance of trabecular bone BMD, however cortical bone BMD does not appear to be affected (Rauch and Schoenau 1998).

De Ridder and colleagues (1998) looked at bone markers and the increase of bone density in pubertal girls using dual-energy x-ray absorptiometry (DEXA). Pubertal development was measured by breast stages using categories: B1 – 9.8 years, B2 – 11.1 years, B3 – 11.3 years, B4 – 12.2 years and B4+ – 12.4 years. A difference was found in BMD (g/cm^2) in the lumbar spine, arm, femur and total body. An increase from B1 – 9.8 years until B3 – 11.3 years, and decrease occurred from B4 – 12.2 years. There was also an increase in bone density and bone turnover during the first half of puberty is thought to be related to the rapid rise in circulating sex steroids and IGF-1 levels. Sex steroids directly affect bone growth by sex steroids receptors on the osteoblast cells (De Ridder et al. 1998).

Mechanical factors

It is widely known that physical activity levels especially in childhood will increase the size and density of the skeleton (Smith and Wordsworth 2005). In cases of reduced physical activity, low bone

mass can result (Bennike et al. 2005). Some studies have shown that physical activity in childhood and adolescence has a positive effect on bone density (Van den Bergh et al. 1995; Nordstrom et al. 1995; Gunnes et al. 1996; Davis et al. 1999; Viña et al. 1999). However, the effect of exercise only influences bone mass up to a certain level (Nordstrom et al. 1996). This increase in bone is expected to be site specific. Ruff (2003) argued the importance of mechanical factors such as body size, muscle size and bone structure in the development of the non-adult skeleton. 'Infancy peaks' were observed in femoral and humeral bone velocities (Ruff, 2003, 326). An increase in femoral strength was reported in the second year of life, which appears to correspond to the beginning of walking, representing the response of previously 'underbuilt' bone to its new mechanical environment. In the third year, a decline in velocity is seen, after the femur has reached equilibrium with its environment (Ruff, 2003). Humeral strength velocity declines in the second year of life, corresponding to the end of crawling and beginning of walking. This is the only part of the growth period when a change in bone strength is not greater than those of body size, if strength is mechanically dependent (Ruff, 2003, 326).

Peck and Stout (2007) showed that bone mass of any skeletal element is intricately linked to its specific mechanical loading environment. They observed intraskeletal variability in bone mass between the bones of the upper limbs and the lower limbs. The femur and tibia were similar, while the fibula was compatible to the ulna. The upper limbs show a different pattern, with the radius having a higher bone density when compared to all other bones, except the ulna. Bone mass in the tibia and femur is correlated because of their role as the primary weight bearing bones. The fibula shares the same loading as that of the upper limb. Greater variability is seen in the upper limb as a result of its functions. The high bone mass observed in the radius and ulna may be attributed to its increased loading frequency imposed on bone by normal muscle contraction. The humerus may experience a decrease in magnitude and frequency of loading after infancy, as it is neither a weight bearing bone, nor under the same rate of use as the radius and ulna (Peck and Stout 2007).

Genetics

An estimated 60–80% of the variability in PBM between individuals has been attributed to heritable factors, demonstrated in adult and adolescent twin studies (Eisman 1999; Pocock et al. 1987; Albagha and Ralston 2003; Dequeker et al. 1987; Young et al. 1995). In a study by Jouanny et al 1995, who observed a 3.8 fold increase in a son's risk of low bone density if his father presented with low bone density. The daughter's risk was higher at 5.1 fold increase if the mother had low BMD. Although the genes responsible for determining bone size, mineral accrual and resorption have not been established with certainty, several candidate genes have been implicated including the vitamin C receptor polymorphisms, estrogen receptor gene, collagen Ial gene, transforming growth factor gene and apolipoprotein E gene (Jouanny et al. 1995).

Genetic factors may play a role in explaining the variation in BMD at different bone sites during adolescence and young adulthood (Smith and Wordsmith

Table 2. Disorders associated with low bone mass and/or fragility fractures in children and adolescents (Adapted from Bachrach et al. 2007)

Genetic Disorders	Chronic Diseases	Endocrine disorders	Immobilization
Ehlers-Donlos Syndrome	Anorexia nervosa	Glucocorticoid excess	Cerebral palsy
Fibrous dysplasia	Asthma	Growth hormone deficiency	Muscular dystrophy
Gaucher's disease	Celiac disease	Hyperthyroidism	Paraplegic
Galactosemia	Cystic fibrosis	Hyperparathyroidism	Idiopathic juvenile
Glycogen storage disease	Hematological disorders	Sex steroid deficiency	osteoporosis
Homocystinuria	Inflammatory bowel diseases	Type 1 diabetes	Disorders causing osteomalacia
Hypophosphatasic			
Marfan's syndrome	Malignancy (Leukemia)		Hypophosphatemic rickets
Osteogenesis imprefecta	Posttransplantation		Vitamin D deficiency
	Renal failure		Vitamin D resistance
	Rheumatological disorders		

2005). Several disorders can lead to this (Table 2); however, some conditions are extremely rare, especially in the archaeological record, for example osteogenesis imperfecta (OI) and osteopetrosis (Ortner 2003). The extent to which genetics plays a role is debatable. It has been found that the bone mass of the daughters of osteoporotic women is reduced when compared to non-osteoporotic women. This may suggest it was more likely to be due to an effect on peak bone mass rather than its subsequent loss (Smith and Wordsmith 2005).

Diet and environment: their role in bone mineral density

Diet

Nutrition can have an effect on bone mineral density. Undernutrition can cause growth problems and both osteoarchaeological and clinical studies usually relate cortical thinning directly to nutritional stress (Hummert 1983). Calcium and protein appear to be directly related to the development of bone mass. Both high and low protein diets seem to result in a lower BMD. This appears to be the case with Eskimos, partly due to high protein diet (Lynnerup 1997 and Von Wowern). At Wharram Percy, despite short periods of malnourishment BMD proceeded normally with growth (McEwan et al. 2005). Calcium is the key nutrient for skeletal health throughout life allowing for optional gains in bone mass during the growing years and reducing bone loss in later life (Heaney et al. 2000). Calcium intake must be sufficient to meet the demands of bone mineral accrual and to compensate for losses. Calcium is often described as a threshold nutrient: skeletal mass increases with increasing calcium until intake reaches the level at which increasing gains are constant. The definition of the calcium threshold for children of varying ages remains in dispute (Wosje and Specker 2000). Phorphorous is another essential

nutrient for bone health, although it receives less attention due to its abundance in daily diet.

Vitamin D is essential for the absorption of calcium not readily available. Studies of bone acquisition in relation to vitamin D are few; Jones and Dwyer (2000) did note a positive effect of winter solar exposure on the bone density of 8 year old Tasmanian children. Severe vitamin D deficiency in children can result in nutritional rickets with marked physical abnormality and osteomalacia. Protein on bone health has been reviewed both deficiencies and excesses and may have adverse effects on the human skeleton (Rizzoli and Bonjour). In a cohort of 200 adolescents, a positive association of bone mass gain and protein intake was noted in both sexes and was most notable from prepuberty through to midpuberty. Children with inadequate protein and caloric intake exhibited growth retardation and decreased formation of cortical bone (Garn 1970).

Metabolic bone disorders

Metabolic bone disorders cause disruptions in the formation of normal bone remodelling and mineralisation (Mays 2008). It would be expected that any disease which has an effect on the bone mineralisation process during childhood would result in diminished bone mineral density in that individual. Oliver et al. (1991) found that in children with x-linked hypophosphatemic rickets there was severely reduced density in the radius diaphysis. There are few studies outlining the effects of malnutrition in children and the subsequent effect on bone density. Handan and colleagues (2006) found that bone mineralisation, when correlated with chronological growth, was delayed in children who were malnourished. Bone mineral density changes have been studied in children with vitamin D deficiency; however, there appears to be contradictory results. El-Desouki and Al-Jurayyan (1997) reported severe mineral loss in children with osteomalacia; whereas Ergür and Erselcan (2000) observed no statistical differences in BMD in children with rickets.

The effects of breastfeeding

There are few studies available which demonstrate any major differences between breastfeeding in infancy and later bone density in a child's life and several have reported no difference (Fewtrell et al. 2009; Specker et al. 1987). However in a study of 330 children from southern Tasmania, Jones and colleagues (2000) found that breastfeeding had an effect on bone mineral density. Children who were breastfed for more than three months had a higher BMD at the femoral neck, lumbar spine and total body compared with those who were bottle-fed. The association with breastfeeding was present in children born at term, but not in those born pre-term (Jones et al. 2000).

There appears to be a difference in the outcome on breastfeeding and BMD in pre-term infants, a few studies have found that those pre-term infants who were breastfed have lower bone mineral density than those infants who were bottle/formula fed (Schanler et al. 1992; Bishop et al. 1996; Chan, 1993). These studies found that a catch-up phase for bone density existed after 12 months of age with earlier deficits being reversed by about 2 year of age; and there was evidence of beneficial effect at 5 years of age, although breastmilk supplementa-

tion was used. Overall, studies do suggest that the nutritional environment of pre-term infants could play an important role in determining skeletal mineralisation and growth later in life.

Climate

The season of birth may influence bone mass due to differences in the levels of ultraviolet light. Studies report seasonal differences in vitamin D concentrations; and it is likely that low maternal vitamin D status during pregnancy affects fetal bone mass (Namgung et al. 1994; 1992). Studies have found that the bone mass of infants born in the summer is lower than that of those born in the winter (Namgung et al. 1992; Oliveri et al. 1991). However, the opposite has been reported in Korea, where infants born in winter have lower bone mineral density than the infants born in summer (Namgung and Tsang 2003).

Childhood Fractures

Fractures can occur in otherwise normal healthy children, with the distal forearm the most common site. The incidence of fractures peaks between 9 and 12 years of age in females and between 12 and 14 years of age in males, coinciding with the pubertal growth spurt. Because peak bone growth precedes peak bone mineral accrual by 6 to 12 months, the skeleton in early adolescence may be relatively undermineralised and more susceptible to fracture with trauma. Some studies have found mean BMD to be significantly lower in children with forearm fractures than in controls. Vertebral compression fractures are far less common than of extremities in childhood. Spine fractures may indicate a marked deficit in bone quality and/or quantity (Chan et al. 1984; Goulding et al. 2000; 2001; Ma and Jones 2003). More recently, links have been made between obesity and prior fractures in children, which may lead to a reduction in total bone density (Dimitri et al. 2010).

Ethnic Difference

Ethnic differences affect bone acquisition during childhood (Lee et al. 2007). Horlick et al. (2000) found the effects of ethnicity on total body BMC significant. BMC was greater in black children than in non-black children. Bachrach et al. (1999) also observed a greater BMD content in Blacks at all skeletal sites. Also significant differences were observed in bone density values between Asian, Hispanic and White individuals. Gilsanz et al. (1997) suggested that the differences in spine BMD between American black and white females occur during puberty; this is also reported by Wang et al. (1997). Prentice et al. (1990) reported greater radial BMD in white British infants compared with black Gambian infants. Asians have been reported as having lower BMD than white children, this may be due to smaller bone size. Nyati et al. (2006) found an ethnic and sex differences in the growth of axial and appendicular skeleton in South African prepubertal black and white children. Lee et al. (2007) reported an increase in bone mineral density at 11 years of age in Korean children and this increase occurred earlier in females than males. When compared to Canadian children, BMD of the total proximal femur was found to be higher than that of the Canadian children, whereas; the BMD of the lumbar spine was found to be less in the Korean children (Lee et al. 2007). This shows that there is a difference

between the bone mineral density of certain bones and ethnic groups. It has been reported that most studies show that bone mineral density is greater in African Americans during childhood than any other group (Agarwal and Stout 2003).

Bone mineral density studies in archaeological human remains

There are many bioarchaeological studies of bone mineral density in adult skeletal remains. However, there are a limited number of publications on bone mineral density studies on non-adult archaeological remains. The majority of bone mineral density studies concentrate on the older skeleton with preference to osteoporosis (Farquharson et al. 1997; Lees et al. 1993; Mays 2000, 2001; Mays et al. 2006; Curate 2014). A number of papers have explored bone mineral density in relation to child health and growth in past populations (Bennike et al. 2005; McEwan et al. 2005). Previous studies on bone mineral density in relation to preservation include Boaz and Behrensmeyer (1976) who measured the bone density of 35 portions of human bone, but the data was based on one bone element and one portion of bone to address the importance of shape and density in relation to fluvial transport. Galloway et al. (1997) measured a total of six long bones (i.e humerus, radius, ulna, femur, tibia and fibula) from adult male and female remains in order to create a standard and accurate measure of bone mineral density.

The measurements were carried out using single photon absorptiometer. There were variations in bone mineral density between the types of bones, scan areas and the morphology. The results suggested that the mid-shaft regions of the long bones have substantially higher bone density than the proximal and distal regions; this appears to support the theory that the mid-shafts of long bones tend to be preserved in large numbers. Other studies include Willey et al. (1997), who examined bone mineral density in two skeletal collections, namely, the Crow Creek Massacre site and a contemporary sample using single photon absorptiometry. They found that certain limb bones have segments of higher density such as which may show greater representation in the archaeological record. In a more recent study on the Crow Creek assemblage Kendell and Willey (2013), observed that BMD was at its greatest in the mid-shaft of the long bones of the non-adult remains. However, in studies carried out by Manifold (forthcoming) on non-adult skeletons from England, there appears to be varying density at the proximal, mid-shaft and distal portions of the long bones in relation to age.

Diagenetic considerations

It must be remembered when studying the BMD of archaeological bone that post-mortem bone loss occurs. As many of the changes of post-mortem bone loss are only visible at microscopic level, the degree of diagenesis has to be more advanced to produce changes that are macroscopically observable. Many diagenetic changes are interpreted as pathological lesions or pseudopathology (Schultz 2003). In some cases soil covers bone surfaces and fills the medullary cavity; which can appear as radiographic artefacts, of which sharp, irregular areas of radiodensity (Mays 2008). Damage can be caused by humid acids that can dis-

solve bony tissues (Schultz 2003). Also, plant roots can cause bone loss by tunnelling through the bony tissue, causing holes. Very small hair-like roots are able to destroy the compact bone substance; the area most often affected is at and along the external bone surface. Fungi and algae can destroy bone, as previously described (Hackett 1981). These tunnels can flow together and produce large destructive holes, which can be misinterpreted as osteoporosis or a tumour. Tunnels produced by algae are smaller than fungi. Protozoa also destroy bony tissue during diagenesis (Schultz 2003). Finally, insects and larvae are mainly found in spongy bone and not in cortical bone substance (Schultz 2003).

Methods of BMD measurement

In order to assess BMC and BMD in children, several techniques have been employed in bioarchaeology: such as dual-energy absorptiometry (DEXA), computed tomography (QCT), quantitative ultrasound (QUS) and radiography. Several clinical studies have used dual-energy x-ray absorptiometry (DEXA) to measure normal values of BMD and BMC in children of different ages (Maynard et al. 1998; Zanchetta et al. 1995; Mølgaard et al. 1997; Southard et al. 1991). These techniques can be used to some extent in comparing archaeological samples. Other researchers have used digital photodensitometry to measure bone density in animals (Symmons 2004) and child remains (Manifold, forthcoming). In addition, energy dispersive low angle x-ray scattering techniques have been employed to measure BMD in archaeological bone (Farquharson et al. 1997). Porosimetry techniques may also be used to measure the total volume and shape of pore spaces within the bone, as bone density is the macroscopic expression of porosity (Robinson et al. 2003).

When considering techniques, it must be remembered that any non-invasive density determination technique that is applied to archaeological bone, whether adult or non-adult, which does not examine the mineral make-up of the sample analysed may produce errors. Bones which appear to be well preserved on the outside may have undergone considerable change internally and microscopically (Bell et al. 1996; Farquharson et al. 1997).

Dual-energy x-ray absorptiometry (DEXA)

Dual-energy x-ray absorptiometry (DEXA) is one of the most common techniques used in the measurement of bone mineral density. DEXA determines the mineral in a given region by the differential absorption of x-rays of two different energies in a higher photon flux, better edge detection, measured by a computer and, with the use of calibration materials, the attenuations value is converted into determination of BMC (in g). BMC values are divided by the projected area of the body analysed and referred to as BMD (g/cm^2) (Gilsanz 1998). BMD is not true bone density, but a ratio of the amount of bone and area scanned (Fewtrell 2003). The use of DEXA in the study of bioarchaeological material is becoming more widespread (Curate 2014; Curate et al. 2013; Fernández Castillo and López Ruiz 2011; McEwan et al. 2005; Manifold 2008). One of the first studies conducted on archaeological bone was to investigate osteoporosis (Hummerl et al. 1990) and this has become a popular area of study (Curate 2014). Manifold (2008) carried

Table 3. An example of BMD (g/cm²) readings using DEXA on a selection of children's bones from the Monastic site of Hulton Abbey, Staffordshire, UK (Manifold, 2008)

Skeleton Number	Age	Bone type	BMD (g/cm²)
HA75	0–3 months	Humerus	0.318
		Proximal Femur	0.401
HA 78	12–18 months	Humerus	0.292
CS01	2–3 years	Distal femur	0.642
		Distal humerus	0.666
CS03	3–5 years	Proximal tibia	0.559
		Fibula	0.342
		Proximal femur	0.829
		Distal femur	0.732
HA18	10–12 years	Proximal femur	0.879
		Midshaft femur	1.147
		Proximal humerus	0.718
		Distal humerus	0.687
		Radius	0.610
		Proximal ulna	0.658
		Distal ulna	0.658
HA35	17–18 years	Proximal femur	1.279
		Midshaft femur (a)	1.790
		Midshaft femur (b)	1.606
		Midshaft femur (c)	1.409
		Distal femur	0.893
		Midshaft tibia	1.490
		Midshaft fibula	0.943
		Lumbar spine	
		L1	0.888
		L2	1.123
		L3	1.570
		L4	1.070
		Total	1.055

out a short pilot study to establish a simple scanning technique using DEXA on six child skeletons from the Monastic site of Hulton Abbey, Staffordshire in the UK (Table 3).

There appeared to be a steady increase from the age of 2 years onwards,with the proximal femur more dense than the distal femur, and the proximal humerus more dense than the distal humerus (Table 3). McEwan et al. (2005) investigated the health of medieval children from the English site of Wharram Percy. There are a number of considerations to take into account when employing DEXA in bioarchaeology. Firstly, making comparisons between different studies and machines is not straight forward due to the differences in software and hardware of DEXA machines. Secondly, you cannot directly compare absolute BMD data from different machines (Genant et al. 1994; Hui et al. 1997). There are differences between scans taken during clinical assessments and

Fig. 2. Bone position in rice bag

those used for bioarchaeological research, and in order to carry out cross-calibration between machines, a bone phantom of different densities is normally applied in clinical settings; with archaeological specimens there may be a need for repeat scanning of objects in different machines (Mays 2008). The biggest difference between the clinical use and the archaeological use is the absence of soft tissue, and this need to be accounted for. Normally specimens (i.e human bone) is placed alongside a material whose density is similar to that of soft tissue, such as water or rice (Fig. 2).The use of water can be prohibited if the specimens under study are valuable and from museum collections. But despite the soft tissue substitute, absolute mineral density cannot be directly compared with modern subjects (Chappard et al. 2004). However, once diagenetic changes have been accounted for then a valid comparison can be made (Mays 2008).

Quantitative computed tomography (QCT)

Quantitative computed tomography (QCT) scanners generate slice images as an array of pixels, with each pixel having a value depending on the attenuation of the X-rays as they pass through the object been scanned (Carlton and Adler 2001). The attenuation is represented by Hounsfield Units (HU), which are scaled and calibrated according to the attenuation of water, so that water has an HU of 1000 and air will have an HU of-1000. The Hounsfield units are then converted to a grey scale covering around 256 shades of grey; this is remapped to about 20 grey-scale shades, which is the reconstructed image. By adjusting window width and level enables viewing of structure of bone, tissue or artefact at the best possible resolution (Fleckenstein and Tranum-Jensen 1993; Lynnerup 2008). Quantitative computed tomography is frequently employed in bioarchaeology in the study of mummies and bog bodies (Lynnerup 2008). QCT allows the measurement of bone mineral density in clinical research, and there are many advantages to its use in archaeological research, such as the study of trabecular bone in greater detail. QCT also allows the calculation of volumetric bone mineral and also gives greater detail with regard to any diagenetic changes which have taken place. Quantitative computed tomography can be obtained at any skeletal site with a standard clinical CT scanner using an external bone mineral reference phantom for calibration and specially developed software. Five different bone measurements can be obtained using QCT to study skeletal development in children: the density of cancellous bone, the density of cortical bone, the size of the axial skeleton, the size of the appendicular skeleton, and the volume of cortical bone in the appendicular skeleton (Gilsanz 1998). This method is the only widely available method to di-

rectly measure BMD in vivo (Rauch and Schoenau 1998). However like DEXA, a soft tissue substitute is required such as water; and again, like DEXA a direct comparison of absolute BMD between ancient skeletal remains and their modern equivalent is not possible. Gonzalez-Reimers and colleagues (2007) carried out a study on prehistoric remains using QCT and found that QCT has the potential to allow study into the trabecular bone mass of ancient remains. There is a lot of research potential with regards to the use of QCT in archaeology, however, it is expensive and gaining access to such equipment can be extremely difficult.

Quantitative ultrasound (QUS)

Ultrasound can be used to assess appendicular bone by measuring the changes that occur in the velocity and in the energy of ultrasound waves as they go through bone. The ultrasound transmission velocity (speed of sound, or SOS) is obtained by dividing the width of the region of interest by the transmit time, and is expressed in metres per second (m/s). The loss of acoustic energy that occurs when the ultrasound wave is absorbed or scattering by the medium through which it is being propagated, results in a reduction in the amplitude of the wave and is referred to as broadband ultrasound attenuation (BUA). BUA is defined as the slope of attenuation versus the frequency in the range of 200–600 kHz and is expressed in decibels per megahertz (dB/MHz) (Gilsanz 1998). This method would also require some form of tissue substitute such as rice or water, in order to allow the sound waves to penetrate the bone. This method is not routinely employed in the study of bioarchaeological remains, but there is the potential for further study.

Photodensitometry (PD)

The simplest way to view the internal structure of a bone element is through the employment of radiographs. Radiographs (X-rays) are electromagnetic waves, which are generated by the energy of electrons as they accelerate through an electric field (Lynnerup 2008). To produce a radiograph, the passing of X-rays is required through a specimen and the capture of the negative image that results from the attenuation of the X-rays (Mays 2008). One of the oldest techniques for measuring bone density from radiographs is photodensitometry (Mack et al. 1939). Density is measured using a standard step-wedge, which is exposed along with the bone on the radiograph, and the standard used to estimate bone density using an optical densitometer (Mays 2008). Online software computer packages such as Image J and scion imaging allow the calculation of optical density.

Limitations of techniques in studying archaeological bone

There are many limitations in studying bone mineral density in archaeological samples. These include the post-mortem alterations to human bones such as breakage and damage caused by soil, which can alter results. The use of modern medical data for comparison with archaeological data cannot be exact. There will always be limitations to the amount of information gleaned from the skeletal collection, due to health and disease issues, which cannot be directly compared. But valid comparisons between skeletal

assemblages and modern clinical data can be made (Mays, 2008) However, practical issues can also severely affect such studies, such as gaining access to specialised equipment like DEXA and QCT, allocation of radiographic supervisors and cost of taking such images

Conclusions

Bone growth and mineralisation during childhood and adolescence is important for bone health in adult life. Factors such as adequate nutrition and physical activity benefit the growing skeleton and increase the levels of bone mineral density. This increase in BMD may play a role in what skeletal elements are being preserved. The study of bone density in archaeological remains is continuing to develop with the use of medical imaging techniques, such as DEXA and QCT, but whilst there are advantages to using these techniques, there is a disadvantage in their use on skeletal material, because absolute BMD values are not directly comparable to those of living subjects. The many factors which affect bone mineral density are unknown variables in archaeological populations, so there is a hidden effect on results. Using bone mineral density in the investigation of bone preservation and representation of non-adult remains has a lot of potential and what is needed is the development of some form of 'archaeological standard values' for BMD which would allow for comparison of sites allowing for more detailed information to be gathered on possible site differences which can lead to poor preservation and also diet and health of such individuals.

Conflict of interest

Author certifies that there is no conflict of interest with any financial organization regarding the material discussed in the paper.

Corresponding author

Bernadette Manifold, The Mews, Darley Abbey, Derby, DE22 1AG, Derbyshire, United Kingdom
e-mail address:
bmmanifold@hotmail.co.uk

References

Agarwal SC, Stout SD. 2003. Bone Loss and Osteoporosis: An Anthropological Perspective. Kluwer Academic: New York.

Albagha OME, Ralston SH. 2003. Genetic determinants of susceptibility to osteoporosis. Endocrinol Metab Clin N Am 32:65–81.

Arabi A, Nabulsi M, Maalouf J, Choucair M, Khalifé H, Vieth R et al. 2004. Bone mineral density by age, gender, pubertal stages, and socioeconomic status in healthy Lebanese children and adolescents. Bone 35(5):1169–79.

Arikcoski P, Komulainen J, Voutilainen R, Kröger L, Kröger H. 2002. Lumbar bone mineral density in normal subjects aged 3–6 yrs: a prospective study. Acta Paediatr 91(2):287–91.

Bachrach LK, Levine MA, Cowell CT, Show NJ. 2007. Clinical indicators for the use of DXA in paediatrics. In: Sawyer AJ, Bachrach LK, Fung FB, editors. Bone Densitometry in Growing Patients: Guidelines for Clinical Practice. Humana Press: Totowa New Jersey.

Bachrach LK, Hastie T, Wang MC, Narasimham B, Marcus R, 1999. Bone mineral acquisition in healthy Asian, Hispanic, Black and Caucasian Youth: A longitudinal study. J Clin Endocr Metab 84(12):4702–12.

Bailey DA, McKay HA, Mirwald RL. 1999. A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children. J Bone Miner Res 14(10):1672–78.

Bell LS, Skinner MF, Jones SJ. 1996. The speed of post mortem changes to the human skeleton and its taphonomic significant. Forensic Sci Int 82:129–40.

Bennike P, Lewis M, Schutkowski H, Valentin F. 2005. Comparisons of child mortality in two contrasting medieval cemeteries from Denmark. Am J Phys Anthropol 127:734–46.

Bianchi ML. 2007. Osteoporosis in children and adolescents. Bone 41(2):486–95.

Bishop NJ, Dahlenburg SL, Fewtrell MS, Morley R, Lucus A. 1996. Early diet of preterm infants and bone mineralization at age five years. Acta Paediatr 85(2):230–36.

Boaz NT, Behrensmeyer AK. 1976. Hominid taphonomy: transport of human skeletal parts in an artificial fluviatile environment. Am J Phys Anthropol 45(1):53–60.

Bonjour JP, Theintz G, Buchs B, Slossman D, Rizzoli R. 1991. Critical years and stages of puberty for spine and femoral bone mass accumulation during adolescence. J Clin Endocrinol Metab 73:555–63.

Bonjour JP, Rizzoli R .1998. Peak bone mass acquisition In: Schoenau E and Matkovic V. Editors. Paediatric Osteology: Prevention of Osteoporosis – A Paediatric Task? Proceedings of the 2nd international workshop on paediatric osteology. Cologne: Elsevier science. 61–81.

Boots AM, De Rodder MAJ, Pols HAP, Krenning EP, Muinck Keizer-Schrama SMPF. 1997. Bone mineral density in children and adolescents: relation to puberty, calcium intake and physical activity. Clin Endo Metab 82(1):57–62.

Braillon PM, Salle BL, Brunet J, Glorieux FH, Delmas PD, Meunier PJ. 1997. Dual-energy X-ray absorptionmetry measurements of bone mineral content in newborns; validation of a technique. Pediatr Res 32:77–80.

Brunton JA, Bayler HS, Atkinson SA. 1993. Validation and application of dual-energy X-ray absorptionmetry to measure bone mass and body composition in small infants. Am J Clin Nutr 58:839–45.

Brunton JA, Wilner HA, Atkinson SA. 1997. Improvement in the accuracy of dual-energy X-ray absorptionmetry for the whole body and regional analysis of body composition: validation using piglets and methodological considerations in infants. Pediatr Res 41:590–96.

Burns C, Henderson N. 1936. The influence of age on the mineral constituents of bones of kittens and pups. Biochem J 30:1207–14.

Carlton RR, Adler AM. 2001. Principles of Radiographic Imaging. An Art and a Science. Delmar: Albany, NY.

Chan GM. 1993. Growth and bone mineral status of discharged very low birth weight infants fed different formulas or human milk. J Pediatr 123:439–43.

Chan GM, Hess M, Hollis J, Book LS. 1984. Bone mineral status in childhood accidental fractures. Am J Dis Child 138:569–70.

Chappard D, Moquereau M, Mercier P, Gallois Y, Legrand E Baslé MF et al. 2004. Ex vivo bone mineral density of the wrist: influence of medullar fat. Bone 34:1023–28.

Cheng JCY, Mahmood A, Hui PW. 1993. Bone mineral content in Chinese children. Hong Kong Medical J. 45(3):209–14.

Cooper C, Fall C, Egger P, Hobbs R, Eastell R, Barker D. 1997. Growth in fancy and bone mass in later life. Ann Rheum Dis 56:17–21.

Curate F. 2014. Osteoporosis and paleopathology: a review. J Anthrop Sci 92:1–28.

Curate F, Albuquerque A, Cunha EM. 2013: Age at death estimation using bone densitometry: testing the Fernández Casillo and López Ruiz method in two documented skeletal samples from Portugal. Forensic Sci Int 226(1):296.e1–296.e6.

Currey JD. 2006. Bones: Structure and Mechanics. Princeton University Press.

Currey J, Butler G. 1975. The mechanical properties of bone tissue in children. J Bone Joint Surg 57-A:810–14.

Dent CE. 1973. Keynote address: Problems in metabolic bone disease. In: Frame B, Parfitt AM, Duncan H. Editors. Clinical Aspects of metabolic bone disease. Excerpta Medica, Amsterdam, The Netherlands. 1–7.

Del Rio L, Carrascosa A, Pons F, Gusinyé M, Yests D, Domenech FM. 1994. Bone mineral density of the lumbar spine in white Mediterranean Spanish children and adolescents: changes related to age, sex and puberty. Pediatr Res 35(3):362–66.

Dequeker J, Nijs J, Verstaeten A, Geudens P, Gevers G. 1987. Genetic determinates of bone mineral content at the spine and the radius: A twin study. Bone 8:207-09.

De Ridder CM. 1998. Bone markers and the increase of bone density in pubertal girls. In: Schoenau E and Matkovic V. Editors. Paediatric Osteology: Prevention of Osteoporosis-a paediatric task? Proceedings of the 2nd international workshop on paediatric osteology. Cologne: Elsevier Science. 81–85.

Dickerson J. 1962. The effects of development on the composition of a long bone of the pig, rat, and fowl. Biochem J 82:47–55.

Dimitri P, Wales JK, Bishop N. 2010. Fat and bone in children: differential effects of obesity on bone size and mass according to fracture history. J Bone Miner Res 25 (2):527–36.

EFFO and NOF. 1997. Who are candidates for prevention and treatment for osteoporosis? Osteoporos Int 7:1.

Ekenman I, Eriksson SAV, Lindgren JU. 1995. Bone density in Medieval skeletons. Calcified Tissue Int 56:355–58.

Eisman JA. 1999. Genetics of osteoporosis. Endocrine Rev 20:788–804.

El –Desouki M, Al-Jurayyan N. 1997. Bone mineral density and bone scintigraphy in children and adolescents with osteomalacia. Eur J Nucl Med 24"202-05.

Ergür AT, Erselcan T. 2000. Diagnostic value of bone mineral density measurements in infants with rickets. J Trop Pediatrics 46:124–26.

Fall C, Hindermarsh P, Dennison E, Kellingray S, Baker D, Cooper C. 1998. Programming of growth hormone secretion and bone mineral density in elderly men: a hypothesis. Clin Endo Metab 83:135–39.

Farquharson M, Speller R, Brickley M. 1997. Measuring bone density in archaeological bone using energy dispersive low-angle X-ray scattering techniques. J Archaeol Sci 24:765–72.

Faulkner RA, Bailey DA, Drinkwater DT, Wilkinson AA, Houston CS, McKay HA. 1993. Regional and total body bone mineral content, bone mineral density and total body tissue composition in children 8–16 years of age. Calcified Tissue Int 53:7–12.

Faulkner RA, Bailey DA, Drinkwater DT, Mckay HA, Arnold C, Wilkinson AA. 1996. Bone densitometry in Canadian children 8–17 years of age. Calcified Tissue Int 59:344–51.

Fernández Castillo RF, López Ruiz M. 2011. Assessment of age and sex by means of DXA bone densitometry: application in forensic anthropology. Forensic Sci Int 209:53–58.

Fewtrell MS. 2003. Bone densitometry in children assessed by dual X-ray absorptionmetry: uses and pitfalls. Arch Dis Child 88:795–98.

Fewtrell MS, Bishop NJ, Edmonds CJ, Isaacs EB, Lucas A. 2009. Aluminium exposure from intravenous feeding solutions and later bone health: 15 year follow up of a randomised trial in preterm infants. Pediatrics 24(5): 1372–79.

Fleckenstein P, Tranum-Jensen J. 1993. Anatomy in Diagnostic Imaging. Copenhagen: Munksgaard.

Foley S, Quinn S, Jones G. 2009. Tracking of bone mass from childhood to adolescence and factors that predict deviation from tracking. Bone 44(5):752–57.

Galloway A, Willey P, Snyder L. 1997. Human bone mineral densities and survival of bone elements: a contemporary sample.

In: Haglund WD and Sorg MH. Editors. Forensic Taphonomy: The Post Mortem Fate of Human Remains. Florida: CRC Press. 295–317.

Garn SM. 1970. The Earlier Gain and Later Loss of Cortical Bone. Charles C Thomas: Springfield, IL.

Genant HK, Grampp S, Glüer CC, Faulkner KG, Jergas M, Engelke K et al. 1994. Universal standardization for dual X-ray absorptionmetry: patient and phantom cross-calibration results. J Bone Miner Res 9:1503–14.

Gilsanz V, Gibben DT, Roe TF, Carlson M, Senac MO, Boechat MI, et al. 1988. Vertebral bone density in children: Effect of puberty. Radiology 166:847–50.

Gilsanz V, Roe TF, Mora S, Costin G, Goodman WG. 1991. Changes in bone vertebral bone density in black girls and white girls during childhood and puberty. N Engl J Med 32:1597–1600.

Gilsanz V, Boechat MI, Roe TF, Loro ML, Sayer JW, Goodman WG. 1994. Gender differences in vertebral body sizes in children and adolescents. Radiology 190:673–77.

Gilsanz V, Konvanlikaya A, Costin G, Roe TF, Sayer J, Kaufman F. 1997. Differential effects of gender on the size of bones in the axial and appendicular skeleton. Clin Endo Metab 82:1603-07.

Gilsanz V. 1998. Bone density in children: a review of the available techniques and indications. Eur J Radiol 26(2):177–82.

Godfrey K, Walker-Bone K, Robinson S, Taylor P, Shores S, Wheeler Y et al. 2001. Neonatal bone mass: influences of parental birthweight, material smoking, body composition, and activity during pregnancy. J Bone Miner Res 16: 1694–1703.

Goksen D, Darcan S, Coker M, Kose T. 2006. Bone mineral density of healthy Turkish children and adolescence. J Clin Densitom 9(1):84–90.

Gonzalez-Reimers E, Velasco-Vázquez J, Arnay-De-La-Rosa M, Machado-Calvo M. 2007. Quantitative computerized tomography for the diagnosis of osteopenia in prehistoric skeletal remains. J Archaeol Sci 34:554–61.

Goulding A, Jones IE, Manning PJ. 2000. More broken bones: a 4 year double cohort study of young girls with and without distal forearm fractures. J Bone Miner Res 15:2011–18.

Goulding A, Jones IE, Taylor RW, Williams SM, Manning PJ. 2001. Bone mineral density and body composition in boys with distal forearm fractures: A dual energy x-ray absorphmetry study. J Pediatr 39(1):509–15.

Gunnes M, Lehmann EH. 1996. Physical activity and dietary constituents as predictors of forearm cortical and trabecular bone gain in healthy children and adolescents: a prospective study. Acta paediatr 85:19–25.

Guy H, Masset C, Baud CA. 1997. Infant Taphonomy. Int J Osteoarchaeol 7:221–29.

Hackett CJ. 1981. Microscopic focal destruction (tunnels) in exhumed bone. Med Sci Law 21:241–65.

Hammerl J, Portsch R, Happ J, Frohn J, Hor G. 1990. Osteodensitometric des femurhalses an historischen skeletten. In: Werner R and Mattiass HH. Editors. Osteologie-Interdisziplinar. Berlin: Springer. 139–42.

Handan ALP, Zerrin O, Tahir K, Hatice U. 2006. Bone mineral density in malnourished children without rachitic manifestations. Pediatr Int 48:128–31.

Hayashi T, Satoh H, Soga T, Tanaka D, Habashi K, Okuyama K. 1996. Evaluation of bone density in newborn infants by computed x-ray densitometry. J Pediatr Gastr Nutr 23(2):130–134.

Hartikainen H, Maleta K, Kulmala T, Ashorn P. 2005. Seasonality of gestational weight gain and foetal growth in rural Malawi. East Afr Med J 82(6):294–99.

Heaney RP, Abrams S, Dawson-Hughes B, Looker A, Marcus R, Matkovic V et al. 2000. Peak bone mass. Osteoporos Int 11(12):985–1009.

Hölger W, Blimkie CJR, Cowell Ct, Kemp AF, Briody J, Wiebe P et al. 2003. A comparison of bone geometry and cortical density at the mid-femur between prepuberty and young adulthood using magnetic resonance imaging. Bone 33:771–78.

Holck P. 2007. Bone mineral densities in the prehistoric, Viking-age and medieval population of Norway. In J Osteoarchaeol 17:199–206.

Horlick M, Thornton J, Wang J, Levine LS, Fedun B, Pierson RN. 2000. Bone mineral in prepubertal children; gender and ethnicity. J Bone Miner Res 15:1393–97.

Hummert JR. 1983. Bone growth and dietary stress among subadults from Nubia's Batn el Hajar. Am J Phys Anthropol 62:167–76.

Hui SL, Gao S, Zhou X-H, Johnson CC, Lu Y, Glüer CC, et al. 1997. Universal standardization of bone density measurements: a method with optimal properties for calibration among several instruments. J Bone Miner Res 12:1463–70.

Janz KF, Eichenberger-Gilmore JM, Levy SM, Letuchy FM, Burns TL, Beck TJ. 2007. Physical activity and femoral neck bone strength during childhood: the Iowa bone development study. Bone 14 (2):216–22.

Jones G, Riley M, Dwyer T. 2000. Breastfeeding in early life and bone mass in prepubertal children: a longitudinal study. Osteoporosis Inter 11:146–52.

Jones G and Dwyer T. 1998. Bone mass in prepubertal children: gender difference and the role of physical activity and sunlight exposure. J Clin Endocrinol Metab 83:4274–79

Jouanny P, Guillemin F, Kuntz C, Jeandel C, Pureel J. 1995. Environmental and genetic factors affecting bone mass similarity of bone density among members of healthy families. Arthritis Rheum 38:61–67.

Kanis JA, Johnell O, De Laet C, Johansson H, Oden A, Delmas P et al. 2004. A meta-analysis of previous fracture and subsequent fracture risk. Bone 35:375–82.

Kendell A and Willey P. 2013. Crow Creek bone bed commingling: relationship between bone mineral density and minimum number of individuals and its effect on paleodemographic analyses. In: Osterholtz AJ, Baustian KM and Martin DL. Editors. Commingled and Disarticulated Human Remains: Working Towards Improved Theory, Methods and Data. New York: Springer. 85–104

Key L, Carnes D, Cole S, Holtrop M, Bar-Shavit Z, Shapiro F, Arceci R, Steinberg J, Gundberg C, Kahn A, Teitelbaum S, Anast C. 1984. Treatment of congenital osteopetrosis with high dose calcitriol. N Eng J Med 310:409–15.

Koo WWK, Walters J, Carlson SE. 1995. Postnatal delay in bone mineralization of preterm (PT) infants. J Bone Miner Res 10:296.

Koo WWK, Walter J, Bush AJ, Chesney RW, Carlson SE. 1996. Dual energy X-ray absorptionmetry studies of bone mineral status in newborn infants. J Bone Miner Res 11:997–1002.

Koo WWK, Bush AJ, Walters J, Carlson SE. 1998. Postnatal developments of bone mineral status during infancy. J Am Coll Nutr 17(1):65–70.

Kurl S, Heinonem K, Länsimies E, Launiala K. 1998. Determinants of bone mineral density in prematurely born children aged 6–7 years. Acta Paediatr 87:650–53.

Landin l, Nilsson BOE. 2008. Forearm bone mineral content in children: Normative data. Acta Paediatr 70(6):919–23.

Lee SN, Desai S, Shetty G, Song HR, Lee SH, Hur CY et al. 2007. Bone mineral density of proximal femur and spine in Korean children between 2 and 18 years of age. J Bone Miner Metab 25(6):423–30.

Lees B, Molleson T, Arnett TR, Stevenson JC. 1993. Differences in proximal femur bone density over two centuries. The Lancet 341:673–75.

Lynnerup N, Von Wowern N. 1997. Bone mineral content in medieval Greenland Norse. Int J Osteoarchaeol 7:235–40.

Lynnerup N. 2008. Computed tomography scanning and three-dimensional of mummies and bog bodies. In: Pinhasi R and Mays S. Editors. Advances in Palaeopathology. Chichester: John Wiley and Sons Ltd. 101–119.

Ma DQ, Jones G. 2003. The association between bone mineral density metacarpal morphomety and upper limb fracture in

children: a population based case-control study. J Clin Endocrinol Metab 88:1486–91.

Mack PB, O'Brien AT; Smith JM, Bauman AW. 1939. A method for estimating the degree of mineralization of bones from tracing of roentgenograms. Science 89:467.

Manifold BM. 2008. Little people, little bones: bone mineral density in non-adult skeletal remains. Poster presented at the World Archaeological Congress, University College Dublin, Dublin.

Manifold BM (Forthcoming) Estimating bone mineral density in non-adult skeletal remains using photodensitometry.

Matkovic V, Jelic T, Wardlow GH, Llich JZ, Goel PK, Wright JK et al. 1994. Timing of peak bone mass in Caucasian females and its implications for the prevention of osteoporosis. Inference from a crossectional model. J Clin Invest 93:799–808.

Maynard LM, Guo SS, Chumlea WC, Roche AF, Wisemandle WA, Zeller CM et al. 1998. Total body and regional bone mineral content and areal bone mineral density in children aged 8–18y: fels longitudinal study. Am J Clin Nutr 68:1111–17.

Mays S. 1999. Linear and appositional long bone growth in earlier human populations: a case study from medieval England. In: Hoppa RD and Fitzgerald CM. Editors. Human Growth in the Past: Studies from Bones and Teeth. Cambridge: Cambridge University Press. 290–312.

Mays S. 2000. Age-dependent cortical bone loss in women from 18th and early 19th century London. Am J Phys Anthropol 112:349–61.

Mays S. 2001. Effects of age and occupation on cortical bone in a group of 18th–19th century men. Am J Phys Anthropol 116: 34–44.

Mays S. 2008. Radiography and allied techniques in the palaeopathology of skeletal remains. In: Pinhasi R and Mays S. Editors. Advances in Palaeopathology. Chichester: John Wiley and Sons Ltd. 77–100.

Mays S, Turner-Walker G, Syversen U. 2006. Osteoporosis in a population from medieval Norway. Am J Phys Anthropol 131: 343–51.

McEwan JM, Mays S, Blake GM. 2005. The relationship of bone mineral density and growth parameters to stress indicators in a medieval juvenile population. In J Osteoarchaeol 15:155–163.

Miller JZ, Slemenda CW, Meany FJ, Reister TK, Hui S, Johnstone CC. 1991. The relationship of bone mineral density and anthropomorphic variables in healthy male and female children. Bone Miner 14:137–52.

Minton SD, Steichen JJ Tsang RC. 1979. Bone mineral content in term and pre-term appropriate-for-gestational-age-infants. J Pediatr 49(6):1037–42.

Mølgaard C, Thomsen BL, Prentice A, Cole TJ, Fleischer Michaelsen K. 1997. Whole body bone mineral content in healthy children and adolescent. Arch Dis Child 79:9–15.

Namgung R, Mimouni F, Campougan BN, Ho ML, Tsang RC. 1992. Low bone mineral content in summer compared with winter-born infants. J Pediatr Gastr Nutr 15:285–88.

Namgung R, Tsang RC, Specker BL, Sierra RL, Ho ML. 1994. Low bone mineral content and high serum osteocalain and 1,25-dihydryvitamin D in summer versus winter born newborn infants: an early fetal effects? J Pediatr Gastr Nutr 19:220–27.

Namgung R, Tsang RC, Sierra RI, Ho ML. 1996. Normal serum indices of bone collagen biosynthesis and degradation in small for gestational age infants. J Pediatr Gastr Nutr 23:224–28

Namgung R, Tsang RC. 2003. Bone in the pregnant mother and newborn at birth. Clin Chim Acta 333(11):1–11.

Neu CM, Manz F, Ranch F, Merkel A, Schoenau E. 2001. Bone densities and bone size at the distal radius in healthy children and adolescents: a study using peripheral quantitative computed tomography. Bone 28:227–32.

Nordstrom P, Thorden P, Nordstrom G, Bergstrom E, Lorentzon R. 1995. Bone mass,

muscle strength and different body constitutional parameters in adolescent boys with a low or moderate exercise levels. Bone 17:351–56.

Nordstrom P, Nordstrom G, Thorsem K, Lorentzon P. 1996. Local bone mineral density, muscle strength, and exercise in adolescent boys: a comparative study of two groups with different muscle strength, and exercise levels. Calcified Tissue Inter 58:402-08.

Nyati LH, Norris SA, Cameron N, Pettifor JM. 2006. Effects of ethnicity and sex on the growth of the axial and appendicular skeleton of children living in a developing country. Am J Phys Anthropol 130:135–41.

Oliveri MB, Ladizesky M, Martinez L, Alonso A, Somoza J, Mautalen CA. 1991. Mineral metabolism of children in vitamin D deficient area of Argentina. Proceedings of the workshop on vitamin D.

Oliveri MB, Cassinelli H, Bergadá C, Mautalen CA. 1991a. Bone mineral density of the spine and radius shaft in children with X-linked hypophosphalemic rickets (XLH) Bone Miner 12(2):91–100

Ortner DJ. 2003. Identification of Pathological Conditions in Human Skeletal Remains. New York: Academic Press.

Park JN, Kim KH, Lee SS. 2004. A study of factors affecting bone mineral density in children: anthropometric measurements, socioeconomic factors, family history and environmental factors. Korean Journal of Nutrition 37(1):52–60.

Peck JJ, Stout SD. 2007. Intraskeletal variability in bone mass. Am J Phys Anthropol 132:89–97.

Płudowski P, Jaworski M, Matusik H, Kobylińska M, Klimek P, Lorenc RS. 2010. The evaluation of consistency between body composition assessments in pediatric population using pencil beam and fan beam dual-energy x-ray absorptiometers. J Clin Densitom 13(1): 84–95.

Pocock NA, Eisman JA, Hopper JL, Yeates MG, Sambrook PN, Eberl S. 1987. Genetic determinates of bone mass in adults. A twin study. J Clin Invest 80:706–10.

Prentice A, Laskey MA, Show J, Cole TJ, Fraser OK. 1990. Bone mineral content of Gambian and British children aged 0–36 months. Bone Miner 10:221–24.

Proesmans W, Goos G, Emma F, Geusens P, Nijs J, Dequeker J. 1994. Total bone mineral mass measured with dual photon absorptionmetry in healthy children. Eur J Pediatr 153(11): 807–12.

Rauch F, Schoenau E. 1998. Timing of puberty and skeletal development. In: Schoenau E and Matkovic V. Editors. Paediatric Osteology: Prevention of Osteoporosis-a Paediatric Task? Proceedings of the 2nd international workshop on paediatric osteology. Cologne: Elsevier Science. 87–94.

Rauch F, Schoenau E. 2001. Changes in bone mineral density during childhood and adolescence: an approach based on bone's biological organisation. J Bone Miner Res 16:597–604.

Rauch F, Schoenau E. 2002. Skeletal development in premature infants: a review of bone physiology beyond nutritional aspects. Arch Dis Child Fetal Neonate Ed 86:F82-F85.

Ribero RR, Santos-Ribeiro KD, Guerra-Junior G, de. A. Bairos-Filho A. 2010. Comparison of bone quantity by ultrasound measurements of phalanges between White and Black children living in Paraná, Brazil with Europeans. Braz J Med Biol Res 43(10):976–81.

Rigo J, De Curtis M, Pieltain C, Nyamugabo K, Senterre J. 1996. Bone mineral density index (BMDI) determined by whole body dual-energy X-ray absorptiometry (DEXA) in IDM, IUGR, and preterm infants: comparison to intrauterine references values. Paediatr Res 40: A548.

Rizzoli R, Bonjour JR. 2004. Dietary protein and bone health. J Bone Miner Res 19: 527–31.

Rizzoli R, Bianchi ML, Garabédion M, McKay HA, Moreno LA. 2010. Maximizing bone mineral mass gain during growth for the prevention of fractures in the adolescents and elderly. Bone 46(2):294–305.

Robinson S, Nicholson RA, Pollard AM, O'Connor TP. 2003. An evaluation of nitrogen porosimetry as a technique for predicting taphonomic durability in animal bone. J Archaeol Sci 30:391–403.

Ruff C. 2003. Growth in bone strength, body size, and muscle size in a juvenile longitudinal sample. Bone 33:317–29.

Sawyer AJ, Bachrach LK. 2007. Rationale for bone densitometry in childhood and adolescence. In: Sawyer AJ, Bachrach LK and Fung FB. Editors. Bone Densitometry in growing Patients: Guidelines for Clinical Practice. Totowa NJ: Humana Press In. 1–13.

Schanler RJ, Burns PA, Abrams SA, Garza C. 1992. Bone mineralization outcomes in human milk fed preterm infants. Pediatr Res 31(6):583–86.

Scheuer L, Black S. 2000. Developmental Juvenile Osteology. Academic Press: London.

Schnitzler CM, Mesquita JM, Pettifor JM. 2009. Cortical bone development in black and white South African children: iliac crest histomorphometry. Bone 44.603–11.

Schoenau F, Fricke O. 2008. Mechanical influences on bone development in children. Eur J Endocrinol 159:S27-S31.

Schultz M. 2003. Differential diagnoses of intravitam and postmortem bone loss at the micro-level. In: Agarwal SC and Stout SD. Editors. Bone Loss and Osteoporosis: An Anthropological Perspective. New York: Kluwer Academic. 172–187.

Smith R, Wordsworth P. 2005. Clinical and Biochemical Disorders of the Skeleton. Oxford: Oxford University Press.

Southard RN, Morris JD, Mahan JD, Hayes JR, Torch MA, Sommer A et al. 1991. Bone mass in healthy children: measurements with quantitative DXA. Radiology 179:735–38.

Specker BL, Brazero LW, Tsang RC, Levin R, Searcy J, Steichen J. 1987. Bone mineral content in children 1–6 years of age –detectable sex differences after 4 years of age. Am J Dis Child 141:343–44.

Specker BL, Namgung R, Tsang RC. 2001. Bone mineral acquisition in utero, during infancy, and throughout childhood. In: R Marcus, Feldman D and Kelsey J, editors. Osteoporosis volume 1: second edition: Academic Press: 599–620.

Stiner MC. 2004. A comparison of photon densitometry and computed tomography parameters of bone density in ungulate body part profiles. Journal of Taphonomy 2(3):117–45.

Symmons R. 2004. Digital photodensitometry: a reliable and accessible method for measuring bone density. J Archaeol Sci 31:711–19.

Trotter M. 1971. The density of bones in the young skeleton. Growth 35:221–31.

Trotter M, Hixton B. 1974. Sequential changes in weight, density and percentage ash weight of human skeletons from an early fetal period through to old age. Anat Rec 179:1–18.

Tsukahara H, Sudo M, Umezaki M, Hiraoka M, Yamamoto K, Ishii Y et al. 1992. Dual-energy X-ray absorptiometry in the lumbar spine, proximal femur and distal radius in children. Pediatr Radiol 22(8):560–62.

Van den Bergh MF, De Man SA, Witteman JC, Hofman A, Tauerbach WT, Grabbee DE. 1995. Physical activity, calcium intake and bone mineral content in children in the Netherlands. J Epidemiol Commun H 19:299–304.

Van Gerven D, Hummert J, Burr J. 1985. Cortical bone maintenance and the geometry of the tibia in prehistoric children from Nubia's Batn El Hajar. Am J Phys Anthropol 66:275–80.

Viña SE, Bueno LG, Armandá MMI, Hernández PC, Lozano TC, Ruibal FJL et al. 1999. Forearm bone mineral density in healthy children. An Esp Pediatríc 51(6):657–63.

Wang HC, Aguirre M, Bhudikanok GS, Kendall CG, Kirsch S, Marcus R et al. 1997. Bone mass and hip axis length in healthy Asians, black, Hispanic and White Americans Youths. J Bone Miner Res 12:1922–35.

Webber CE, Beaumont LF, Morrison J, Sala A, Barr RD. 2007. Age predicted value for lumbar spine, proximal femur, and whole

bone mineral density: results from a population of normal children aged 8–18yrs. Can Assoc Radiol J 58(1):37–45.

Wetzsteon RJ, Hughes JM, Kaufman BC, Vazquez G, Stoffregen TA, Stovitz SD et al. 2009. Ethnic differences in bone geometry and strength are apparent childhood. Bone 44:970–75.

Willey P, Galloway A, Snyder L. 1997. Bone mineral density and survival of elements and element portions in the bones of the crow creek massacre victims. Am J Phys Anthropol 104:513–28.

Wosje KS, Specker BL. 2000. Role of calcium in bone health during childhood. Nutrition Rev 58:253–68.

Yeste D, Del Río L, Gussinyé M, Carrascose A. 1998. Bone mineral density in nursing infants and young children (0–4 yrs old) at the level of the lumbar spine: the normal pattern. An Esp Pediatric 49(3):248–52.

Young P, Hopper JL, Nowson CA, Green RM, Sherwin JA, Kaymakci B et al. 1995. Determinants of bone mass in 10 to 26 year old females: A twin study. J Bone Min Res 10(4):558–67.

Zanchetta JR, Plotkin H, Alvarez Filgueira ML. 1995. Bone mass in children: normative values for the 2–20 year old population. Bone 16(4):393S–99S.

Zhai F, Zhag L, Wang C, Pan H. 2004. Study of normal reference values for bone mineral contents in children and adolescents in Beijing. Journal of Hygiene Research 33(2):172–75.