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

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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).
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
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Table 1. Published data on the normative bone mineral density in healthy children from different geographical regions
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 endochondral 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
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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
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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 areal 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/cm²) 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
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 (Otte 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). Phosphorous 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-
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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-
Bone density in children

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/cm²) (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)

<table>
<thead>
<tr>
<th>Skeleton Number</th>
<th>Age</th>
<th>Bone type</th>
<th>BMD (g/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HA75</td>
<td>0–3 months</td>
<td>Humerus</td>
<td>0.318</td>
</tr>
<tr>
<td>HA78</td>
<td>12–18 months</td>
<td>Proximal Femur</td>
<td>0.401</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Humerus</td>
<td>0.292</td>
</tr>
<tr>
<td>CS01</td>
<td>2–3 years</td>
<td>Distal femur</td>
<td>0.642</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal humerus</td>
<td>0.666</td>
</tr>
<tr>
<td>CS03</td>
<td>3–5 years</td>
<td>Proximal tibia</td>
<td>0.559</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibula</td>
<td>0.342</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proximal femur</td>
<td>0.829</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal femur</td>
<td>0.732</td>
</tr>
<tr>
<td>HA18</td>
<td>10–12 years</td>
<td>Proximal femur</td>
<td>0.879</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Midshaft femur</td>
<td>1.147</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proximal humerus</td>
<td>0.718</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal humerus</td>
<td>0.687</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Radius</td>
<td>0.610</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proximal ulna</td>
<td>0.658</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal ulna</td>
<td>0.658</td>
</tr>
<tr>
<td>HA35</td>
<td>17–18 years</td>
<td>Proximal femur</td>
<td>1.279</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Midshaft femur (a)</td>
<td>1.790</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Midshaft femur (b)</td>
<td>1.606</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Midshaft femur (c)</td>
<td>1.409</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal femur</td>
<td>0.893</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Midshaft tibia</td>
<td>1.490</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Midshaft fibula</td>
<td>0.943</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lumbar spine</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>L1</td>
<td>0.888</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L2</td>
<td>1.123</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L3</td>
<td>1.570</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L4</td>
<td>1.070</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total</td>
<td>1.055</td>
</tr>
</tbody>
</table>

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 straightforward 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
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 organisation regarding the material discussed in the paper.

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