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INVITED REVIEW

Ethnicity and bone: a South African perspective

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Introduction

There is extensive literature to show that bone mineral density (BMD) and fracture rates differ in children and adults between different ethnic groups in both developed and developing countries. The factors responsible for these differences are currently unclear, but the findings do raise intriguing questions about the role that genetics and the environment play in determining the bone phenotype in various ethnic groups. This review describes the differences in bone histomorphometry, mass, size and strength, and fracture rates between black and white South African (SA) children and adults, compares these findings to those described in other countries and provides possible explanations for some of the differences found between these studies.

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South African demographics

South Africa is classified as a low-middle income country, but has a complex burden of disease including a high HIV/AIDS prevalence, poverty-related infectious diseases, persisting infant and child malnutrition and stunting, as well as a high prevalence of obesity and overweight in children, adolescents and adults [1–3]. It continues, however, to undergo major epidemiologic transitions with increasing urbanization, which may impact its various communities in different ways. Approximately 79% of the SA population (total 49 million) is black,[1] while 8.8% is colored; both groups having been historically disadvantaged within the old SA political regime. The white population, which makes up 9.2% of the population in South Africa, is predominantly northern and/or eastern European in origin, and has settled in South Africa over the last 450 years. The remaining 2.6% of the SA population consists of the Indian/Asian population, who arrived mainly as indentured workers in the last 150 years. Due to previous apartheid laws, racial admixture within the SA black population, who originally migrated from West Africa in the 1600s, is likely to be low, particularly when compared to the United States (US) black population, which has a relatively large degree of admixture [4]. This has been confirmed in unpublished pilot data on 1000 black participants in the Birth to Twenty (BT20) cohort on whom 18 ancestral markers were measured. Little admixture was found in this group suggesting a relatively homogenous population. The SA people of mixed ancestral origin, referred to as 'colored', descended

[1] Race categorization in South Africa refers to the apartheid system of population group classification into black, white, colored and Indian groups and has been retained by the post-apartheid government for demographic reasons in order to redress previous inequalities. It is only used here due to its familiarity as population descriptors.

from slaves brought from East and central Africa, the indigenous Khoisan who lived in the Cape at that time, indigenous Africans, white settlers, and an admixture of Malay, Indian and Asian groups. The influence of different socioeconomic, lifestyle and nutritional environments within South Africa on the original genetic phenotype of the different population groups may help to explain differences in bone structure, mass and strength, and fracture risk in SA populations, and why these findings may be different from those of other countries.

Adult studies (Table 1)

Fracture incidence

Work by Solomon et al. in the late 1960s conclusively showed that the incidence of femoral neck (FN) fractures is low in the SA black population, with a reported fracture rate of 4.5 and 4.2 fractures per 100000 per annum, for black men and women, respectively [5], despite the metacarpal bone density subsequently being shown to be higher in the white than the black groups [6]. At that time, Walker et al. [7] reported that despite calcium intakes of SA men and women being low at between 250 and 400 mg/day and women having numerous pregnancies and long lactations, the metacarpal indices of elderly SA black men and women were not significantly different from international data on white adults. Although there is information on hip fracture rates in SA populations, this is not true for fractures at other sites, and it is unclear whether or not the marked differences in fracture rates seen at the hip apply to other skeletal sites as well. Osteoporosis of the lumbar vertebrae, diagnosed using lateral X-rays, has been reported in black SA women [8], with the study suggesting that the prevalence was lower in black that white subjects. There is also some unpublished evidence to indicate that vertebral fractures are not uncommon in elderly black SA women, and occur at a similar rate to postmenopausal white women (11.5 vs. 8.1%) [9]. In a recent longitudinal study conducted over 5 years, Basu [10] found that 38% of black women over the age of 60 years developed new vertebral deformities of >20%, suggesting a high prevalence of vertebral fractures in the black population in South Africa. More recent data on SA hip fracture is not available.

Histomorphometry

Histomorphometric examination of trabecular bone of the iliac crest in SA adults has found that black adults have thicker trabeculae, greater osteoid surface, volume and thickness, as well as a greater erosion surface, which might

Table 1 Comparison of South African (SA) and United States (US) body composition and bone data in adults

	SA	US	References
Anthropometry			
Weight			
Height	*B<W*	*B=W*	[15, 25, 47, 52]
Body composition			
Fat mass			
Lean mass	*B<W*	*B>W*	[26, 47]
Visceral fat	B<W	B<W	[25, 52]
Peripheral fat	B>W	B>W	[25, 52]
DXA measures			
Total hip BMD	B>W	B>W	[15, 18, 46]
Femoral neck BMD	B>W	B>W[a]	[18, 46, 47]
Lumbar spine BMD	B=W; B<W	B>W[a]	[15, 18, 47, 48]
Hip structural analysis			
Bone CSA[neck, intertrochanteric ROI]	B>W	B>W	[46]
Bone CSA[shaft ROI]	*B=W*	*B>W*	[46]
Bone outer diameter[intertrochanteric ROI]	B<W	B<W	[46]
Section modulus[neck, shaft ROI]	*B=W*	*B>W*	[46]
Endosteal diameter[neck, intertrochanteric ROI]	B<W	B<W	[46]
Cortical thickness[neck, intertrochanteric ROI]	B>W	B>W	[46]
Cortical thickness[shaft ROI]	*B=W*	*B>W*	[46]
Buckling ratio[neck, intertrochanteric ROI]	B<W	B<W	[46]
Fracture			
Fracture rate	B<W	B<W	[5, 66–69]

[a] Not adjusted for differences in body size. Italicized data is different between SA and US populations

Table 2 Comparison of South African (SA) and United States (US) body composition and bone data in children

	SA	US	References
Growth			
Weight	*B<W (boys only)*	*B>W*	[28, 56]
Height	*B<W*	*B=W; B>W*	[28–30, 56, 59, 60]
Sitting height	*B<W (boys only)*	*B=W; B<W*	[29, 60, 70]
Body composition			
Fat mass			
Lean mass	*B<W*	*B>W*	[28, 30, 56]
DXA measures			
Whole body BMC	B>W; B=W	B>W	[28, 34, 36, 56, 58, 71]
Lumbar spine BMC	B>W (girls only); B=W	B>W[a]; B=W; B>W	[28, 35, 36, 55–58]
Femoral neck BMC	B>W	B>W	[35, 36, 55]
Mid-radius BMC	B>W	B>W[a] (girls only)	[36, 55]
pQCT measures			
Leg muscle CSA	*B<W*	*B>W*	[30, 59]
Arm muscle CSA	*B<W (boys only)*	*B=W*	[30, 59]
Leg fat CSA	B=W	B=W	[30, 59]
Tibial diaphysis[trabecular BMD]	*B=W*[b]	*B>W*[b]	[30, 59]
Tibial diaphysis[total BMD]	*B=W*[b]	*B>W*[b]	[30, 59]
Tibial diaphysis[total area]	B=W[b]	B=W[b]	[30, 59]
Tibial diaphysis[BSI]	*B=W*[b]	*B>W*[b]	[30, 59]
Radial diaphysis[trabecular BMD]	B>W (girls only)[b]	B>W[b]	[30, 59]
Radial diaphysis[total BMD]	B=W[b]	B=W[b]	[30, 59]
Radial diaphysis[total area]	*B>W (boys only)*[b]	*B=W*[b]	[30, 59]
Radial diaphysis[BSI]	*B=W*[b]	*B>W*[b]	[30, 59]
Tibial metaphysis[total area]	B>W[b]	B>W[b]	[30, 59]
Tibial metaphysis[cortical area]	*B=W*[b]	*B>W*[b]	[30, 59]
Tibial metaphysis[cortical density]	B>W (boys only)[b]	B>W[b]	[30, 59]
Tibial metaphysis[cortical thickness]	B<W[b]	Data not available	[30]
Tibial metaphysis[endosteal diameter]	B>W[b]	Data not available	[30]
Tibial metaphysis[tibial diameter]	B>W[b]	Data not available	[30]
Tibial metaphysis[periosteal circumference]	B>W[b]	Data not available	[30]
Tibial metaphysis[polar strength-strain index]	B>W[b]	B>W[b]	[30, 59]

[a] Not adjusted for differences in body size

[b] SA data: adjusted for bone age, US data: adjusted for age, sex, tibial length and tibial muscle cross-sectional area (CSA). Italicized data is different between SA and US populations

suggest a stronger microarchitecture, than their white peers [11]. Similar studies of cortical bone showed differences suggestive of microarchitectural advantages and increased bone-forming capacity, including thicker cortices, greater endocortical wall thickness, fewer intracortical osteons, greater osteoid thickness and surface, greater endocortical mineral apposition rate and endocortical bone formation rate, and lower values for eroded surface, in blacks than in whites [12].

Dual energy X-ray absorptiometry (DXA)

Although it has been suggested that BMD may not predict fracture risk in black African women [13], a number of SA studies have shown differences in BMD between ethnic groups. One of the first studies to investigate ethnic differences in BMD, with specific reference to the SA population, was by Kalla et al. [14]. The purpose of their study was to establish reference data for SA women, and included white women and women of mixed ancestral origin. Their results showed no significant differences in lumbar spine (LS) or proximal femur (PF) BMD between the groups. A later study by Daniels et al. [15] assessed age-related differences in BMD in black and white SA female nurses between the ages of 20 and 64 years. They reported a 7, 10, and 13% higher PF BMD in black women compared to white women in the premenopausal, perimenopausal and postmenopausal groups, respectively, after adjusting for differences in height and weight. In the same

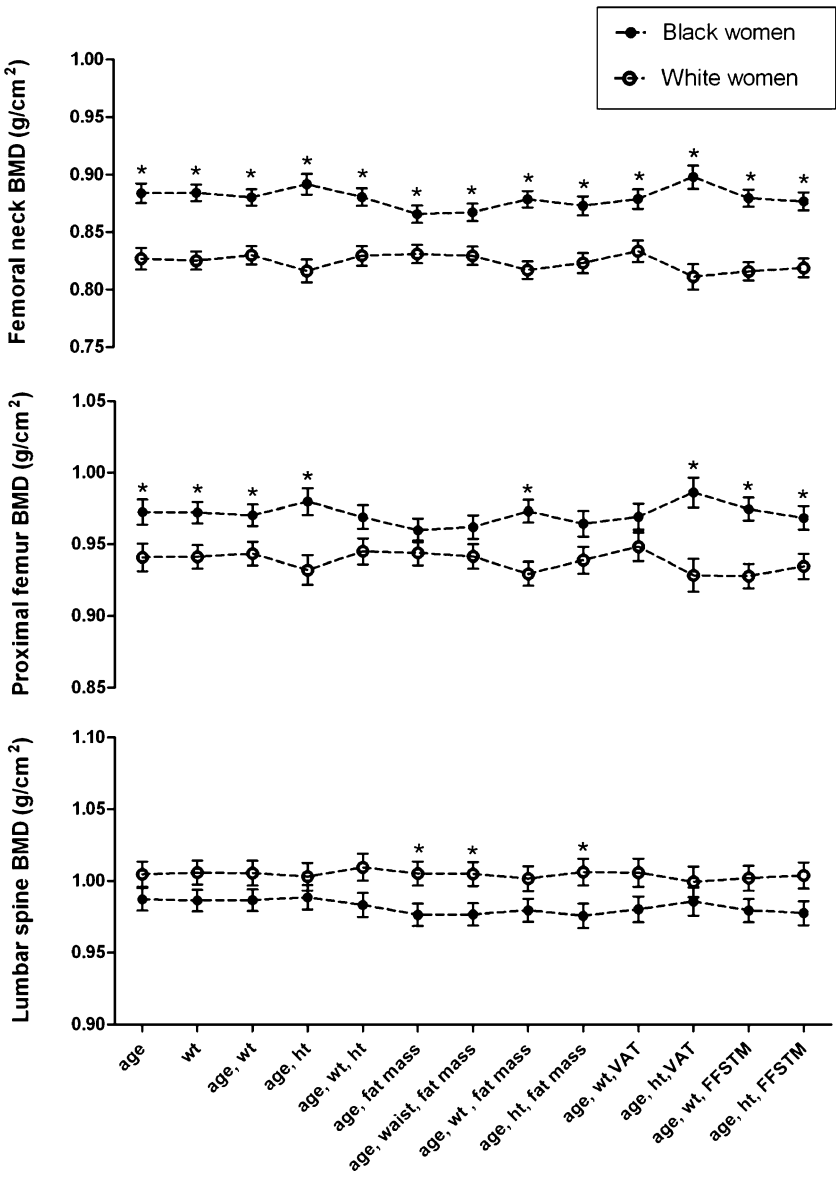

Fig. 1 Ethnic comparison of BMD (g/cm^2) at the femoral neck, total hip and lumbar spine, following adjustment for different combinations of age and body composition variables. *Wt* weight, *ht* height, *VAT* visceral adipose tissue, *FFSTM* fat-free soft tissue mass. *$P < 0.05$, *black* mean different from *white* mean. Reproduced from [18] with permission (licence number 2625850181249)

groups of nurses, no ethnic differences in BMD of the LS or radius were found; however, in the premenopausal group, LS, PF and radial BMD were positively correlated with age in black, but not white, women. In the postmenopausal women ethnic differences were also reported, with BMD at all three sites being negatively correlated with the number of years since menopause in the white, but not in the black, group. These findings are different from those reported from The Gambia where black women were found to have lower BMD than age-matched Caucasians, and lost bone mass after the menopause [13]. Whole body (WB) bone mineral content (BMC) has been reported to be higher in white than black SA women in a study by Rush et al. [16]; however, this difference disappeared when adjustments were made for differences in age, height and weight. Subsequent work by Daniels et al. [17] found bone mineral apparent density, an estimate of volumetric bone density, to be higher at the FN and LS in black than white subjects. The most recent data published in SA women showed FN and PF BMD to be higher, and LS BMD to be lower, in black than white women, after adjusting for differences in body size (Fig. 1) [18].

The continuing increase in BMD with age during the premenopausal period, and lack of bone loss in the postmenopausal period at the LS and PF sites, may explain the lower hip fractures rates in black women; however, the mechanisms for these findings are not known. It is also not known if these patterns are seen in black males on whom there is much less information. These findings suggest that black and white SA women may respond differently to similar stimuli or that there may be different mechanisms or factors influencing bone mass accretion and subsequent

bone loss in black and white SA women. However, as DXA is unable to provide the information necessary to assess possible ethnic differences in bone structure, shape or volume, there is a need for further work using different technologies such as quantitative computed tomography (QCT) to assess bone size, true volumetric density and trabecular bone mass.

The influence of lifestyle and socioeconomic factors on bone

The 2003 SA Demographic and Health Survey reported that at that time there were still ethnic differences in employment, education, income, access to health care and property ownership [19]; all of which may influence BMD directly or indirectly. These differences have probably not changed dramatically in the last decade. White SA women have also been shown to participate in more vigorous leisure-time physical activity, whereas black SA women, many of whom use walking as a means of transport, accumulate more incidental moderate-intensity activity [20]. Further research in black SA women has shown an association between load carrying on the head and LS BMD suggesting that there is a site-specific osteogenic response to loading at the spine [21]; however, load carrying on the head is no longer common amongst increasingly urbanized black women in South Africa. Black SA women also consume a diet which is higher in fat [22] and approximately 25% lower in calcium (mean 436 vs. 577 mg/day) [23] than their white peers. Injectable contraceptive use, which has been shown to impact negatively on site-specific BMD [21] and calcaneal ultrasound BMD [24], is approximately four times more common in black than white SA women [19]. It appears from recently published work, that the contribution of lifestyle and socioeconomic factors to BMD, may differ between black and white SA women [18]. Nevertheless, despite these apparently negative lifestyle and socioeconomic influences on BMD, black women still have a higher BMD at the hip than white women. Future research needs to expand on these findings in order to inform future interventions in SA women.

The influence of body composition on bone

SA black women are shorter [25], have less lean mass [26], less visceral adipose tissue (VAT) and greater peripheral fat [25] than their white counterparts; characteristics which may account for differences in bone gain and loss. In a study of 240 black and 187 white premenopausal SA women, fat mass, fat-free soft tissue (lean) mass, age, height and ethnicity accounted for 79% of the variance in WB BMC in a regression model. When entered separately, and with no other covariates, fat-free soft tissue mass accounted for 53% of the variance compared to fat mass which only accounted for 40%. In addition, Chantler et al. [18], reported that fat-free soft tissue mass was the most significant contributor to BMD at the LS and hip sites in black women and at hip sites in white women, with fat mass only making a significant contribution to LS BMD in white women.

Studies in children (Table 2)

Growth

Much of the data on ethnic differences in SA children has been obtained from studies utilizing data from a longitudinal cohort of 3273 black, mixed ancestry and white children who were enrolled into the BT20 study at birth in 1990 [27]. When the children were aged 9 years, a subsample of this cohort (The Bone Health cohort; BH) was used to study in depth the bone mass and structural changes that occur during puberty and adolescence. We have shown that white SA children are heavier, taller and have greater lean body mass compared to black SA children [28–30], and a study by Nyati et al. [29] has shown ethnic differences in growth of the axial and appendicular skeletons, with site-specific bone length predicting bone mass better than height. In this study, although there were no differences in skeletal maturity between the ethnic groups at 9 years of age, height and sitting height were greater in the white children compared to their black counterparts; however, segment lengths including subischial length, ulna length, humeral length, and calf length, after adjusting for differences in height, were greater in black than white children. These data suggest that during the prepubertal period there is a proportionally greater growth in the appendicular skeleton in black children and in the axial skeleton in white children. The contribution of regional segment lengths (and thus bone size) to BMC and BMD in these children may help to explain ethnic differences in BMD.

Fracture incidence

Fracture rates in SA children have been reported by Thandrayen et al. [31] using the first 15 years of fracture data from the BT20 cohort. Of the 2031 subjects included in this study, 22% (males 27.5% and females 16.3%) had experienced one or more fractures during their lifetime, giving an overall fracture rate of 18.5/1000 children/annum. There was a significant difference between the black and white ethnic groups with 41.5% of white, and only 19% of black children having experienced a fracture

in their first 15 years of life. Besides the differences in fracture rates between the two ethnic groups, fracture rates in males were approximately double those of females in both groups. Prospective data published on the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC) cohort at 9 years of age have shown an inverse relationship between fracture risk and WB BMD, unadjusted and after adjustment for body and bone size, resulting in the conclusion that bone mass is an important predictor of fracture risk [32]. Although we currently do not have information on the association between bone mass and fracture in the BT20 cohort, we have consistently found higher PF BMD in black than white SA children at various ages, which may help to explain the lower fracture incidence in black children.

Histomorphometry

The ethnic differences in the histomorphometric measures of cortical bone from the iliac crest found in adults appear to become apparent during childhood [33]. This study reported significantly greater osteoid thickness, endocortical wall thickness and cortical thickness in black than white children, and the authors suggested that these differences may help to explain ethnic differences in fracture rate.

DXA

A number of other studies utilizing the BH cohort have reported BMD differences between pre- and early pubertal black and white SA children at 9 and 10 years of age [28, 34–36]. In the study by Vidulich et al. [36] on 10-year-old children, after adjusting for anthropometric and pubertal differences, black children had a greater FN, PF and mid-radius BMD; however, there were no differences at the WB, LS or distal radius. In a study in the same cohort of children at 9 years of age, Micklesfield et al. [35] and McVeigh et al. [28] found similar differences at the two hip sites, but also a small, but significantly greater, LS BMC in black than white girls. Possible reasons for this discrepancy in the results between the 9- and 10-year-old children may be the difference in pubertal status, as all 9-year-old participants were pre-pubertal, while approximately 1% of the 10-year-old cohort was mid-pubertal.

Peripheral quantitative computed tomography (pQCT)

The difficulties in interpreting DXA-derived BMD in growing children have been well described [37]. QCT provides information that is not available using DXA, including volumetric density and measures of bone geometry and trabecular bone mass, with pQCT providing these data at appendicular bone sites. Using pQCT, we have shown that 13-year-old SA children have ethnic and sex differences in bone size and strength at both the diaphyseal (38%) and metaphyseal (4%) sites of the appendicular skeleton [30]. Ethnic differences in bone size were more apparent at the tibial diaphyseal site, where black children had greater total bone area, tibial diameter and periosteal circumference than white children. Black children also had reduced cortical thickness and greater endosteal diameter than white children, although there was no difference in cortical bone area between the groups. Despite thinner cortices and a larger marrow cavity in black children, the differences resulted in a 10–20% greater polar strength-strain index in these children, and translates into black children having an appendicular skeleton that is more resistant to bending and torsional forces. At the metaphyseal sites of the radius and tibia, however, we were unable to show any significant ethnic differences in bone strength, as measured by the bone strength index. The finding of ethnic differences at the 38% tibia, a predominantly cortical site, but not at the 4% radius and tibia, predominantly trabecular sites, mimics the ethnic differences we see between black and white adults and children using DXA. BMD at the hip sites, which consist of 50–75% cortical bone, has repeatedly been shown to be higher in SA black adults and children than in their white peers, while LS BMD, composed of >66% trabecular bone, has been shown to be either the same in black and white groups [15], or higher in white than black women [18].

The influence of lifestyle and socioeconomic factors on bone

Although increased sports participation in children may predispose to fracture risk, it is widely accepted that the mechanical loading associated with weight-bearing physical activity is positively correlated with BMD [38]. We have previously shown that physical activity during adolescence correlates with a higher BMD later in life [39], and socioeconomic status (SES) and lifestyle factors, including physical activity, are also associated with BMD outcomes in pre- and early pubertal SA children [28, 40, 41]. Norris et al. [40] have shown that two measures of SES status, namely, caregiver's marital/cohabiting status, an indicator of social support, and the presence of a television in the home, an indicator of greater disposable income, were associated with WB bone area and BMC in pre- and early pubertal children.

The influence of body composition on bone

We have confirmed in 9-year-old black and white children the strong relationship between WBBMC and the other components of body composition, namely, fat mass and

fat-free soft tissue mass, as has been reported in the international data [34, 42]. Although lean mass in SA children, adolescents and adults has been found to be lower in black compared to white groups, our data in 13-year-old black and white children still support the important relationship between muscle area and bone size [30]. We have found a significant positive relationship between muscle cross-sectional area and diaphyseal cortical bone area in all groups, with the correlation coefficient ranging from 0.45 to 0.60. The mechanostat theory hypothesizes that the increase in muscle forces that occur during growth influences the size and strength of the bone [43, 44]. Our findings suggest that cortical bone in black and white children may respond differently to mechanical forces or that the response is modulated by other environmental factors, as black children have larger bone diameters despite smaller muscle cross-sectional areas than white children.

The influence of pubertal development on bone

Although sex-specific changes in bone morphology have been shown to occur with pubertal development [45], the influence of ethnicity is not as clearly understood. To determine if bone changes associated with pubertal development are ethnic-specific we compared various metaphyseal and diaphyseal bone parameters, as measured by pQCT, at different pubertal stages in black and white children at 13 years of age [30]. At the metaphyseal sites of the radius and tibia, the differences in the various bone parameters between early, mid- and late pubertal stages were similar in black and white groups. However, at the 38% tibial diaphysis, cortical area, cortical density, cortical thickness and bending strength were greater in black girls who were in late puberty compared to those in early and/or mid-pubertal stages. This difference between the pubertal stages was not seen in boys or white girls. Analyses of our longitudinal cohort study should give us a clearer understanding of how ethnicity influences bone size and geometry during pubertal development.

Comparisons between populations in different countries

In both developed and developing countries, BMD is higher at the FN and PF sites in black populations compared to their white counterparts [15, 18, 46, 47]. Although LS BMD has also been reported to be higher in African Americans than white Americans [47, 48], it has been shown to be similar between ethnic groups in South Africa [15], or to be higher in white than black SA women, after adjusting for differences in body size [18] (Fig. 1). In trying to understand the pathogenesis of these differences it is important to recognize that environmental factors associated with geographic location may affect bone mass or architecture. Nelson et al. [46] compared BMD and cross-sectional geometric properties of the PF, using the hip structural analysis program, in black and white postmenopausal women from Detroit (USA) and Johannesburg (South Africa). They found higher BMD, greater bone cross-sectional area and smaller periosteal and endosteal diameters, but greater cortical thickness, in black women from both countries compared to their white counterparts. These findings suggest greater bone strength in the black groups in both countries. Of interest was the finding that the differences were smaller between the same ethnic groups in the USA and South Africa than between different ethnic groups within the same country. We have obtained similar results in 9-year-old children enrolled in the BH cohort, whom we compared to children of a similar age from Detroit [34]. We found WB BMC, after adjusting for age, weight, height and sex, to be 7–8% lower in children of European ancestry compared to those of African ancestry, irrespective of geographic location. These data suggest that despite black children being exposed to very different environmental factors in each country, they still have an inherent advantage in BMC when compared to children of European ancestry. Possible reasons for the differences in BMC have been proposed and include differences in bone turnover [49] and in the frequency of various genetic polymorphisms [50, 51]. In addition, marked inter-country differences were noted with SA children having significantly higher WB BMC compared to US black and white groups, possibly indicating that there are differences in bone mass within what may be considered the same 'racial' group, but living in different geographic regions. Similar findings have been noted in adults, with SA women having significantly higher LS BMD than their US counterparts [14], which provides further evidence that although there appear to be some ethnic similarities irrespective of geographic location, the differing environmental influences associated with living in different countries may have marked effects on the original genetic phenotype of different groups. These influences may be mediated via anthropometric factors such as height and body composition, which may or may not differ between ethnic groups depending on factors such as socioeconomic and nutritional status associated with geographic location. SA differences in anthropometry, mentioned earlier, are in contrast to US differences. US black women are of similar height [52], have greater lean mass [53], less VAT and greater peripheral fat [52] than US white women, while SA black women are shorter [25], have less lean mass [26], less VAT and greater peripheral fat [25] than SA white women. In West Africa, however, Gambian women have a significantly lower BMI than age-matched Caucasian

women [13], and show more similar patterns of bone gain and loss to SA white, rather than black, women. These inter-country differences in anthropometric and body composition measures may help to explain some of the inter-country differences in bone mass seen in women of different ethnic origins. The geographic variation in hip fracture incidence has been well described in a review by Dhanwal et al. [54], in which they report a higher hip fracture incidence in developed/industrialized countries compared to developing countries. There is a paucity of recent data on fracture rates in South Africa; however. the data from 1968 mentioned earlier, reported the hip fracture rate of black SA women to be approximately one-tenth of that of age-matched white Western European populations from Scotland and Sweden [5]. It is suggested that as South Africa continues to become more developed, with an increasing elderly population who participate in less physical activity, hip fracture rates will be an additional burden on an already over-burdened health system.

Studies from South Africa and the USA have reported similar ethnic differences at the PF and FN [55]; however, in contrast to SA data, US studies have shown LS BMD to be higher in African American than white children [55–57]. It appears that the ethnic differences described in US children might be related in part to differences in body size as in some of the studies after adjusting for differences in body size, the differences at the LS no longer reach significance [58]. Differences in body size and rates of pubertal development between ethnic groups within South Africa and the USA, may help to explain some of the ethnic differences in BMD and bone strength in both children and adults between the two countries. Anthropometric differences between the ethnic groups in childhood [34] and early adolescence [30] indicate that patterns of growth between black and white populations in South Africa are not the same as those in high-income countries. White US children are lighter, have similar or shorter height, and have lower lean mass than their black peers [56, 59, 60]; the reverse being true between black and white SA children. SA black girls have shown a marked secular trend in the age at menarche, which in the past has been markedly delayed compared to white SA girls, with a decline of approximately 0.5 years per decade compared to 0.2 years per decade in white girls (Fig. 2) [61]. Data from the BH cohort show black and white girls now having similar ages of onset of menarche (12.4 vs. 12.5 years). In the USA, black girls reach menarche earlier than white girls [62]. Data from another African country, Ghana, also document a dramatic decline in menarcheal age from 16.06 years of age in 1989 to 14.9 years of age in 2008, a decline of 0.65 years per decade [63]. Less information is available for secular trends in pubertal development in boys; however, evidence from several studies in South Africa suggests that the picture may be different as SA black boys present with delayed onset and rate of pubertal development compared to white boys [30, 64]. This pattern may explain the increased prevalence of stunting seen in black boys in early adolescence [64], something which has not been reported in US studies of boys of the same age. Yet despite what may be considered 'unfavorable' anthropometric measurements and poorer nutrition, such as lower calcium intake [41] and lower (but not deficient) serum 25-hydroxyvitamin D levels [65], not only have we shown consistently higher BMD at the hip in SA black compared to white children, but also a lower fracture incidence [31].

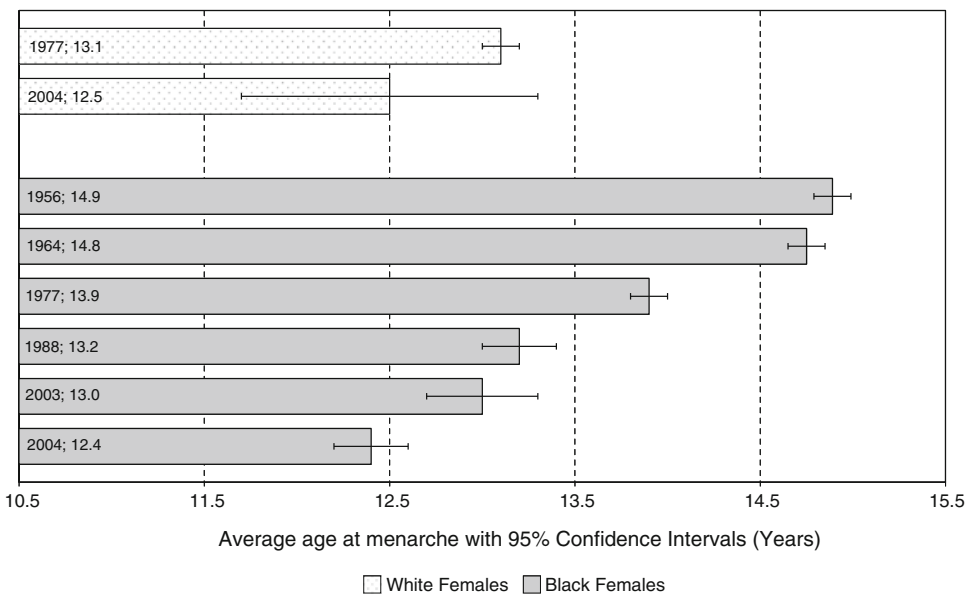

Fig. 2 Average menarcheal age (95% confidence intervals) for white and black urban SA girls between 1956 and 2004. Reproduced from [61] with permission (licence number 2624060189628)

Conclusions

Ethnic comparisons in bone mass, bone architecture and fracture rates have been studied in a number of different populations, with most studies from the USA reporting differences between African Americans and white Americans. Thus it is frequently assumed that black populations in Africa and other parts of the world will have similar patterns of bone gain and loss to African Americans, and that ethnic differences will be similar worldwide. It is, however, clear from our studies in South Africa that although there are similarities in the findings between ethnic groups in the USA and South Africa, there are also differences as SA blacks do not have higher LS or radial BMD, even after adjusting for differences in body size. Of considerable interest from a mechanistic point of view is the finding that despite adverse environmental conditions, including poor nutrition and low physical activity levels, as well as an unfavorable body composition, black SA children and adults have greater PF and FN BMD, greater bone strength and a decreased hip fracture incidence compared to SA whites. Although these bone differences appear to be similar to those reported between US ethnic groups, factors such as body composition, pubertal development and lifestyle vary greatly between SA and US populations. Future directions within this field of research should explore the adaptation of the different ethnic groups in these countries, and the possible over-riding role of genetics. These findings also highlight the need for the development of local bone mass reference data, as the US data for African Americans are not appropriate for the black populations of other countries.

Conflict of interest None.

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