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PEDIATRIC HIGHLIGHT

Association between stunting and overweight among 10–15-y-old children in the North West Province of South Africa: the THUSA BANA Study

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OBJECTIVE: To investigate the relationship between stunting and overweight among 10–15-y-old children of the North West Province in South Africa.
DESIGN: A single cross-sectional study design was used. The study formed part of the THUSA BANA project.
SUBJECTS: The total study population of the THUSA BANA project comprised of 1257 randomly selected subjects, aged 10–15 y.
MEASUREMENTS: Stunting was described as the height below the 5th percentile for age using the CDC standard percentiles. Furthermore, the definitions of overweight and obesity according to the International Obesity Task Force (IOTF) were used, where the cutoff points for body mass index (BMI) corresponds with the adult BMI of 25 and 30, respectively. Anthropometrical variables namely triceps (TSF) and subscapular skinfolds (SSF), waist circumference, weight, height and BMI of the 10–15-y-old subjects were analysed.
RESULTS: Stunting was most prevalent in the rural areas (girls 23.7% and boys 26.7%) compared with urban areas (girls 11.6%, boys 17.1%). The odds ratio and the 95% CI for the association between stunting and overweight in boys and girls were 0.45 (CI 0.16, 1.30) and 0.50 (CI 0.21, 1.19) respectively. Stunted children, 10–14-y-old and living in rural areas and informal settlements, had significantly lower mean BMI and skinfold thicknesses than nonstunted children. The mean BMI and sum of TSF and SSF (TSF + SSF) were similar in stunted and nonstunted children living in urban areas.
CONCLUSION: There is no significant association between stunting and overweight in 10–15-y-old children in the North West Province. However, there is a tendency for girls older than 14 y to start to gain subcutaneous fat, even though at these ages they were still stunted and underweight. Stunted girls in established urban areas had a higher mean TSF + SSF than stunted girls in informal townships. This tendency in urban stunted girls is evident at the onset of menarche and could predict possible problems of overweight as they get older.
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Introduction

In the past, there was a clear association between stunting and access to food; the more food that was available the less the incidence of stunting. In recent times, this association may not be as apparent.[1] In particular, a relationship between stunting and obesity has been postulated in countries undergoing the nutritional transition.[2] An investigation into this relationship is particularly relevant to South Africa, a country in transition. In this regard, the nutrition transition is a sequence of characteristic changes in dietary patterns and nutrient intakes associated with social, cultural and economic changes during the demographic transition. Improved economic conditions and resource availability are associated with higher fat intakes and lower levels of physical activity in transitional communities.[1,3]

However, the occurrence of obesity at present is unprecedented in the history of mankind. This worldwide problem can be considered to be a result of social, economic and cultural problems being encountered by developing and newly industrialised countries, as well as ethnic minorities and the disadvantaged in developed countries. A recent

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national study in South Africa showed that 56.6% of women aged 15 y and older were overweight or obese.[4] An escalating rate of obesity (also an increase in type II diabetes, hypertension, dyslipidaemia and CVD, coupled with cigarette smoking and alcohol abuse) is a frequent outcome of the modernisation or acculturation.[3] The deleterious effects of obesity manifest subsequently in adulthood. However, obesity not only persists from infancy into adulthood, but has been associated with an unfavourable cardiovascular risk profile in the first 20 y of age.[5,6] These studies suggest that childhood is a key period to identify those at risk of becoming obese adults. The development of obesity may start during the adolescent growth spurt period, which is one of the critical periods for the development of obesity identified by Dietz.[7] In girls, menarche indicates that the adolescent growth spurt has been completed and can be used to indicate a new stage of development.[8] Maturation can be assessed through a variety of approaches including skeletal age, appearance of secondary sexual characteristics and menarche. However, many studies of adolescents only report menarche.[5,8] Childhood nutritional stunting, usually an indicator of chronic malnutrition[9,10] and undernutrition,[1,11] has been suggested as a contributory factor to elevated rates of obesity (high weight-for-height) in developing countries. This association between stunting and adolescent and adult obesity has been investigated in several other studies.[1,2,12] Speculative explanations for this association include that the reduced body mass of malnourished children result in decreased basal metabolic rate. At an adequate or excessive energy intake linear growth may be limited when protein and other nutrient intakes are not adequate, but potential for fat deposition is not.[13] In recent times, it has become apparent that the condition of being overweight coexists with undernutrition in many developing countries. In this regard, the prevalence of childhood obesity is a serious problem, for which there is a lack of sufficient data for African countries. In response to this, a World Health Organisation (WHO) Report[3] has identified the relationship between body mass index (BMI) and adiposity in children with retarded linear growth (stunting) as a research priority.

Therefore, a study of the relationship or association between stunting and overweight or obesity among 10–15y-old children of the North West Province in South Africa can clarify the relationship between obesity and stunting and consequently, more appropriate prevention and intervention strategies can be developed.

Methods

Study sample

This research forms part of the THUSA BANA study. THUSA BANA are Setswana (local language) words that mean 'Help the Children'. THUSA is also an acronym for 'Transition and Health during Urbanisation of South Africans'.

This cross-sectional study was carried out on a randomly selected group of children between 10 and 15 y of age in the North West Province of South Africa. The measurements were carried out during school hours by the researchers.

Representativeness

The study population consisted of 44 schools that were selected from the five regions of the province. The study population was further stratified in terms of type of school (high/primary school), age (10–15 y) as well as ethnic group, with at least 100 children from each age group for boys and girls ($6 \times 100 \times 2 = 1200$).

Eventually the study population comprised of two high schools and four primary schools, randomly selected using two-digit random numbers from predominantly black schools out of every region, one high school and one primary school from predominantly white schools and one high school and two primary schools from predominantly coloured (mixed ancestry) and Indian schools. The latter were only chosen from regions 3 and 4, where most predominantly Indian and coloured schools were situated. Subsequently, girls and boys between 10 and 15 y were randomly selected systematically in each school from class lists ($n = 1336$), to be representative of the population of the North West Province. The distribution of subjects by race and gender is presented in Table 1.

There was a 94.1% response rate. Owing to parents not giving consent and children being involved in sports activities, practical classes and being too shy to be measured, we could not realize the planned study population of 1336. Eventually, the total study population of the THUSA BANA project comprised 1250 subjects, of which 604 were boys and 646 girls.

The names of the children that formed part of the study were given to the school beforehand so that the children would be ready when the researchers arrived. Informed consent was given by the children, their parents and school

Table 1 Distribution of subjects by age, gender and race ($n = 1250$)

Age (y)	N	*Black*	*White*	*Indian*	*Coloured*
Girls (n = 646)					
10–10.99	116	88	17	4	7
11–11.99	109	84	12	7	6
12–12.99	122	89	18	6	9
13–13.99	100	73	15	6	6
14–14.99	96	69	14	7	6
15–15.99	103	77	15	6	5
Boys (n = 604)					
10–10.99	85	56	16	7	6
11–11.99	115	89	14	7	5
12–12.99	127	98	16	8	5
13–13.99	94	63	14	4	13
14–14.99	89	58	19	5	7
15–15.99	94	69	20	2	3
Total	1250				

principals. The study protocol was approved by the Ethics Committee of the Potchefstroom University.

Experimental procedures

Questionnaires were designed in pilot studies and field-workers were trained to administer questionnaires in the form of demographic and anthropometric data forms. The demographic questionnaire included questions about the subject's stratum of urbanisation, gender, age, language, health status, smoking habits, education level and socioeconomic status. The following classification for strata of urbanisation was used: people living on farms or in small tribal villages were classified as rural, those living on the periurban fringe in informal shacks, without formal water supply, were classified as informal and those living in established urban environments as urban. For girls the onset of menarche and age at menarche was noted. Furthermore, trained biokineticists and one dietitian were responsible for the actual recording of the measurements mentioned below according to the standard methods recommended by Norton and Olds.[14] In this regard, care was taken to calibrate the apparatus used correctly. Quality control methods included the training of fieldworkers, correction of data before and after calculation and duplication of measurements. All anthropometric measurements were carried out during the school hours.

Variables, measuring techniques and apparatus

Techniques of measurement and the apparatus that were used to measure these variables were as follows. Weight was measured to the nearest 0.1 kg with a portable electronic scale (Precision Health Scale, A & D Company, Tokyo, Japan). The subjects were weighed in their underwear. The subjects had to stand in an upright position in the middle of the scale with their weight evenly distributed between their feet. Height was measured by using a portable stadiometer. The subject was barefoot in an upright standing position with heels together, weight evenly distributed between the feet and arms relaxed along the sides. The heels, thighs, top of the back and (if possible) the back of the head had to touch the vertical part of the stadiometer with the head in the Frankfort plane. The subject's hair was flattened with the use of the head level so that the direct reading on the top of the head could be taken. The reading was measured to the nearest 0.1 cm.[15] A height below the 5th percentile for age and gender according to the CDC standard percentiles[16] was classified as stunting.

A flexible Lufkin (Cooper Tools, Apex, NC, USA) steel tape was used to measure circumferences. The metal casing was held in the right hand, while the left hand extended the measuring tape until it may be passed to the right hand ('crossover hand' method). With all the circumference measurements, it was imperative that the measuring tape be held in a parallel position. The tape measure was pulled tightly but carefully around the area being measured, in order to ensure that the tape does not cut into the subject's skin. All circumferences were measured to the nearest 0.1 cm. Waist circumference (WC) was measured halfway between the superior ridge of the ilium and the lower border of the lowest floating rib. Mid-upperarm circumference (MUAC) was measured midway between the acromion process of the scapula and the olecranon process of the ulna.[15]

The skinfold thicknesses were measured with a John Bull (British Indicators, London, UK) skinfold caliper held at a constant pressure of 10 g/mm^2. The location where the skinfold thickness had to be measured was clearly demarcated. A double layer of skin together with the subcutaneous fat was firmly picked up with the thumb and forefinger on the demarcated spot. The skinfold was pulled away from the underlying muscle tissue with the mouth of the caliper placed 1–2 cm under the fingers and at a depth of 1 cm over the skinfold. The reading was taken 2–3 s after the caliper has been placed over the skinfold and the needle of the caliper has stabilised. All skinfold measurements were measured to the nearest 0.2 mm. This included the triceps skinfold (TSF), measured vertically midway between the acromion process of the scapula and the olecranon process of the ulna, and subscapular skinfold thickness (SSF), measured directly underneath the lowest point of the scapula.[15]

In the calculations below, height and weight values are used in specific formulas for the determination of body mass index (BMI). Children were classified as being overweight or obese according to the BMI-for-age cutoff points corresponding to adult BMI of 25 and 30 kg/m^2, respectively,[17] for example at the age of 14 y a BMI of 23.3 kg/m^2 for girls would correspond to an adult BMI of 25 kg/m^2 and a BMI of 23.9 kg/m^2 at 15 y would correspond to an adult BMI of 25 kg/m^2.

Statistical analysis of the data

The SAS computer package (StatSoft, Inc'99) for Windows was used to analyse the gathered data. Firstly, the descriptive statistics of the anthropometric variables were reported for different age groups as described in Thomas and Nelson.[18] The association between stunting and overweight in the children was investigated by using a combination of frequency tables, *t*-tests, phi-coefficient tests, two-way tables and manual odds ratio calculations.

Descriptive statistics of stratum, gender, age and age at menarche were determined by using frequency tables. Furthermore, additional frequency tests included tables of age by stratum, menarche by age, height by weight, weight by age and weight by gender. The Mann–Whitney *U*-test was used to assess differences between the mean TSF, MUAC, waist circumference, weight, height, BMI and TSF + SSF for girls who have reached menarche and those who have not in each age group, since these data tended not to have a normal distribution. Comparisons between stunted and nonstunted subjects were carried out by using *t*-tests, s.d., minimums and maximums for all the anthropometric variables for age and gender. The effect size (small effect size: $d<0.2$; medium effect size: $d=0.2$–0.8; large effect size: $d>0.8$) gives an indication of the size of the difference when comparing the means of two variables.[18,19] The effect size was calculated as the difference in means between stunted and nonstunted children, expressed in pooled s.d..[18,19] We accepted significance at a $P<0.05$. In order to determine the association between stunting and overweight, manual calculations of the odds ratio and the 95% CI were caried out based on the formula by Bland and Altman.[20] Furthermore, chi-squared tests from two-way tables of height by weight for all the 10–15 y-old girls and boys were carried out.

Results

Urbanisation and ethnicity

About one-third of the children (35.8%) lived in rural areas, 17.8% lived in informal settlements and 46.4% lived in urban areas. We found the highest prevalence of stunting in rural areas (boys: 26.7%, girls: 23.7%) followed by the informal settlements (boys: 26.4%, girls: 13.7%) and the lowest prevalence of stunting in the urban areas (boys: 17.1%, girls: 11.6%). About half (51%) of the stunted girls and 43% of the stunted boys came from the rural areas. Children from urban areas tended to have higher mean BMIs and TSF + SSF than children from rural areas and informal settlements (Table 2). Among stunted girls, the differences between urban girls and girls living in informal settlements were statistically significant. The highest prevalence of overweight and obesity was found among white children (14.2%), compared to black (7.1%), Indian children (6.4%) and children of mixed ancestry (2.9%).

Menarche

Menarche indicates that most of the adolescent growth spurt has been completed. In our study (Table 3), 225 (34.8%) girls had started menstruating at the time of the study. There was a rapid increase in the number of girls reaching menarche at ages 14 and 15 y. The mean age at menarche of the girls was 12.78 y (95% CI 12.6, 12.9), 12.53 y (95% CI 12.24, 12.82) in white girls and 12.95 y (95% CI 12.77, 13.15) in black girls.

The Mann–Whitney *U*-test was used to assess differences between means of TSF, MUAC, waist circumference, weight, height, BMI and TSF + SSF for premenarcheal and post menarcheal girls. Statistically significant differences ($P<0.05$) are shown in Table 3. The mean age at menarche of stunted girls (13.37 y, 95% CI 12.80, 13.95) differed significantly from that of nonstunted girls (12.73 y, 95% CI 12.58, 12.89).

Anthropometrical status

The differences in anthropometric indices between stunting and nonstunting in 10–15-y-old girls are presented in Table 4. Similarly, differences in anthropometric indices between stunting and nonstunting in 10–15-y-old boys are presented in Table 5.

The mean TSF for the 15-y-old stunted girls was greater than the mean TSF for the nonstunted girls, although this difference was not significant (Table 4). The most important observation is that of no significant difference between the mean TSF of stunted and nonstunted 14–15-y-old girls. MUAC is considered to be one of the best anthropometric indicators of total body muscle mass.[15] There was a gradual increase in mean MUAC of boys and girls with age (Tables 4 and 5).

It is clear from Table 5 that stunted boys' mean WC increased gradually with age but a slightly smaller mean was evident at 14 y of age. Contrary to this, the stunted girls' (Table 4) mean WC gradually increased up to the age of 13 y and then there was a sudden increase at ages 14 and 15 y. The WC of 14–15-y-old stunted girls did not differ significantly from the WC of 14–15-y-old nonstunted girls at a significantly greater height of the nonstunted girls.

A gradual increase in weight with age in all the nonstunted subjects was evident (Tables 4 and 5). Furthermore, there was

Table 2 Mean body mass index (BMI) and sum of triceps and subscapular skinfolds (TSF+SSF) of children according to stratum of urbanisation (mean ± s.d.)

	Stratum 1 (rural) n = 55 stunted, 159 nonstunted		*Stratum 2 (informal settlements) n = 28 stunted, 78 nonstunted*		*Stratum 3 (urban) n = 47 stunted, 233 nonstunted*	
	BMI (kg/m²)	*TSF+SSF (mm)*	*BMI (kg/m²)*	*TSF+SSF (mm)*	*BMI (kg/m²)*	*TSF+SSF (mm)*
Boys						
Stunted	15.3 ± 1.62	12.0 ± 7.22	15.1 ± 1.85	12.5 ± 5.41	16.1 ± 1.93	14.5 ± 6.53
Nonstunted	16.5 ± 2.73	16.0 ± 2.27	16.0 ± 2.27	14.8 ± 9.56	17.1 ± 3.05	16.9 ± 11.79
P^a	0.001	0.02	NS	NS	NS	NS
P^b	NS	NS	NS	NS	NS	NS
Girls						
Stunted	15.8 ± 2.45	16.5 ± 8.94	15.1 ± 1.29	14.3 ± 5.0[b]	16.8 ± 2.9	19.9 ± 13.6[b]
Nonstunted	17.4 ± 3.06	23.6 ± 11.73	17.1 ± 2.74	23.6 ± 12.38	18.1 ± 3.5	26.9 ± 13.1
P^a	0.006	0.0008	0.003	0.0005	NS	NS
P^b	NS	NS	NS	0.02	NS	0.02

[a]Student's *t* test, level of significance of difference between stunted and non-stunted subjects.
[b]Student's *t* test, level of significance of difference between stunted subjects of different strata.

Table 3 Differences shown as mean (±s.d.) between anthropometrical variables of premenarcheal and postmenarcheal girls, measured by Mann–Whitney *U* test ($n=646$)

	10 y	*11 y*	*12 y*	*13 y*	*14 y*	*15 y*
TSF (mm)						
Premenarcheal	11.5 (6.1)	12.5 (5.2)	12.8 (6.0)	11.7 (5.5)	12.4 (5.4)	9.9 (2.0)
Postmenarcheal	15.2 (9.9)	13.8 (3.1)	15.5 (4.5)[a]	16.0 (4.7)[a]	18.1 (8.4)[a]	18.7 (7.7)[a]
TSF+SSF (mm)						
Premenarcheal	20.7 (11.4)	22.7 (11.9)	22.9 (10.8)	22.0 (10.8)	23.3 (10.8)	21.3 (6.0)
Postmenarcheal	23.9 (13.2)	26.0 (7.3)	27.5 (8.5)[a]	29.7 (10.8)[a]	35.1 (16.1)[a]	36.2 (17.0)[a]
Stature (cm)						
Premenarcheal	135.6 (8.3)[a]	141.9 (6.9)	146.0 (8.4)	149.1 (7.8)	150.9 (6.3)	156.0 (8.6)
Postmenarcheal	144.6 (3.4)[a]	145.3 (5.1)	153.6 (6.3)[a]	157.1 (6.7)[a]	158.2 (6.9)[a]	158.8 (7.2)
BMI (kg/m^2)						
Premenarcheal	16.2 (3.1)	17.1 (3.3)	17.0 (2.9)	17.0 (2.9)	17.6 (2.6)	18.6 (1.6)
Postmenarcheal	16.5 (3.0)	17.7 (2.6)	19.1 (2.4)[a]	19.1 (2.4)[a]	20.7 (4.1)[a]	20.6 (3.1)[a]
Total n						
Premenarcheal (421)	117	101	104	62	27	10
Postmenarcheal (225)	4	5	18	37	69	92

[a]Statistically significant differences between pre- and postmenarcheal girls ($P<0.05$).

a gradual increase in weight with age in the stunted boys, but the stunted girls displayed a rapid increase in weight at ages 14 and 15 y (Table 4). This coincides with the increase in the incidence of menarche, or puberty (Table 3). In addition, all the girls' mean weights were more than the boys' for the corresponding ages, except for the 10-y-old stunted girls and 15-y-old nonstunted girls.

In this study, there were 134 stunted boys and 105 stunted girls. Thus, the overall prevalence of stunting was 19%. We can estimate that 19% of the 10–15-y-old children of the North West Province are stunted due to the fact that the study population was representative. In Tables 4 and 5, the effect size for the difference in height between stunted and nonstunted subjects was large. Table 4 provides an illustration of some of the most important aspects of our study related to the BMI of girls. The 10–13-y-old boys had similar mean BMIs; however, there was a sudden increase in the BMI after the age of 13 y. There appears to be a low prevalence of overweight for both groups of stunted and nonstunted boys. BMI for the girls increased with age and exhibited a sudden increase at 14 and 15 y. In 14- and 15-y-old stunted girls, the mean BMIs were 19.42 and 21.21 kg/m^2, respectively. Similarly in nonstunted 14- and 15-y-old girls, it was 19.95 and 20.32 kg/m^2, respectively. However, the mean BMI of the 15-y-old stunted girls exceeded the mean BMI of the 15-y-old nonstunted girls, but the difference was not significant (Table 4). Table 2 shows significant differences in the means of BMI and TSF + SSF of stunted and nonstunted children in rural areas. However, in urban areas, no significant differences could be found between these measures of weight status of stunted and nonstunted children.

Table 4 and Figure 1 shows the means of sum of TSF + SSF of the girls. For the stunted girls, there was a rapid increase in the mean TSF + SSF to 27.31 and 39.35 mm at ages 14 and 15 y, respectively. Similarly, the nonstunted girls also displayed a rapid increase in the mean TSF + SSF to 32.52 and 34.34 mm at ages 14 and 15 y, respectively. This coincides with the increase in incidence of menarche for 14 and 15-y-old girls (Table 4). The mean TSF + SSF for the 15-y-old stunted girls exceeded the mean TSF + SSF for the nonstunted girls, although the difference was not significant (Table 4, Figure 1).

Stunting and obesity

The following results were calculated manually by substituting percentages from a two-way table (Table 6) into the odds ratio formula by Bland and Altman.[20] A nonsignificant probability existed for stunted boys ($P=0.19$) and girls ($P=0.11$) to be overweight (Fisher's exact test). The odds ratio for stunted boys to be overweight or above was 0.45 (95% CI 0.16, 1.30), and for stunted girls the odds ratio was 0.5 (95% CI 0.21, 1.19) (Table 6).

Discussion

Demographic data

According to the 1995 census, 90% of the population (adults and children) of the North West Province were black, 8% were white, 1% coloured and 0.3% Indian.[21] As mentioned previously, a total of 1257 subjects participated in this study. However, with some of the anthropometric variables, only 1250 subjects were measured. This study sample is still representative of the entire population of 10–15-y-old children in the North West Province, but with white, coloured and Indian children slightly over-represented to

Table 4 Differences between means (±s.d.) of the anthropometric indices of stunted and nonstunted girls, 10–15 y old, measured by *t*-test

Age (y)	*Indices*	*Stunted* (n = *105*)	*Nonstunted* (n = *541*)	P	*Effect size* (d)[a]
10	Height (cm)	123.7 (5.9)	138.5 (6.5)	0.0001	2.3
	Weight (kg)	23.8 (2.8)	31.5 (7.7)	0.0001	1.1
	TSF (mm)	8.9 (3.6)	12.3 (6.5)	0.002	0.6
	WC (mm)	53.4 (4.2)	56.8 (6.8)	0.006	0.5
	MUAC (mm)	16.9 (1.9)	19.1 (2.9)	0.0003	0.8
	BMI (kg/m^2)	15.6 (2.1)	16.3 (3.3)	0.26	0.2
	TSF+SSF (mm)	16.9 (8.9)	21.9 (11.8)	0.04	0.4
11	Height (cm)	131.4 (4.1)	143.4 (5.9)	0.0001	2.1
	Weight (kg)	26.8 (2.4)	35.9 (8.3)	0.0001	1.2
	TSF (mm)	9.6 (2.0)	13.0 (5.3)	0.0001	0.7
	WC (mm)	53.5 (2.8)	59.9 (7.8)	0.0001	0.9
	MUAC (mm)	17.7 (1.4)	20.1 (2.4)	0.0001	1.1
	BMI (kg/m^2)	15.5 (1.1)	17.4 (3.4)	0.08	0.6
	TSF+SSF (mm)	16.0 (3.0)	23.9 (12.1)	0.0001	0.7
12	Height (cm)	134.9 (5.6)	150.1 (6.2)	0.0001	2.5
	Weight (kg)	29.9 (5.1)	39.8 (8.6)	0.0001	1.2
	TSF (mm)	10.8 (4.6)	13.8 (6.0)	0.01	0.5
	WC (mm)	55.8 (5.0)	61.3 (6.2)	0.0001	0.9
	MUAC (mm)	18.7 (2.1)	21.0 (2.9)	0.0001	0.8
	BMI (kg/m^2)	16.4 (2.3)	17.6 (3.0)	0.04	0.4
	TSF+SSF (mm)	18.5 (7.6)	24.9 (10.9)	0.002	0.6
13	Height (cm)	140.6 (4.3)	154.8 (6.6)	0.0001	2.8
	Weight (kg)	31.2 (5.2)	44.0 (8.6)	0.0001	1.6
	TSF (mm)	9.1 (2.3)	14.4 (5.7)	0.0001	1.0
	WC (mm)	56.7 (4.2)	63.0 (5.9)	0.0001	1.1
	MUAC (mm)	18.7 (2.6)	21.7 (2.5)	0.0005	1.2
	BMI (kg/m^2)	15.7 (1.9)	18.3 (2.8)	0.0001	1.0
	TSF+SSF (mm)	17.1 (4.7)	26.9 (11.6)	0.0001	0.9
14	Height (cm)	146.0 (2.3)	157.8 (6.6)	0.0001	1.9
	Weight (kg)	41.5 (9.6)	49.9 (10.7)	0.008	0.8
	TSF (mm)	14.2 (7.5)	17.0 (8.1)	0.22	0.3
	WC (mm)	63.4 (8.5)	65.4 (8.7)	0.41	0.2
	MUAC (mm)	21.8 (3.4)	23.0 (3.3)	0.24	0.4
	BMI (kg/m^2)	19.4 (4.2)	20.0 (3.9)	0.66	0.1
	TSF+SSF (mm)	27.3 (14.8)	32.5 (15.8)	0.24	0.3
15	Height (cm)	145.9 (2.6)	160.3 (5.9)	0.0001	2.6
	Weight (kg)	45.2 (6.2)	52.4 (9.4)	0.003	0.8
	TSF (mm)	19.8 (9.0)	17.8 (7.6)	0.48	−0.3
	WC (mm)	65.4 (4.9)	67.1 (7.1)	0.28	0.3
	MUAC (mm)	23.3 (3.0)	23.7 (2.7)	0.72	0.1
	BMI (kg/m^2)	21.2 (2.5)	20.3 (3.2)	0.28	−0.3
	TSF+SSF (mm)	39.4 (21.0)	34.3 (16.2)	0.44	−0.3

[a]Effect size (*d*) is the difference in means between stunted and nonstunted children, expressed in pooled s.d.; guideline values according to Cohen[19]: small effect size: $d<0.2$; medium effect size: $d=0.2$–0.8; large effect size: $d>0.8$.

include a minimum number of subjects, in order to make comparisons between ethnic groups possible.

Urbanisation and ethnicity

Harris *et al*[22] found the proportion of children with stunted growth was greater in nonurban areas than in urban areas in Tibet. Similarly, in our study, we found that stunted growth was most prevalent in the rural areas. In the rural areas, differences in measures of weight status between stunted and nonstunted children were also more pronounced (Table 2). Maturational differences between black and white girls have been reported in the past.[23] It was found in past cross-sectional studies that black girls are further in the maturation process than white girls at both ages 9 and 10 y. This earlier onset of maturation may have a primary role to play in the development of obesity differences between the races.[24] Contrary to other studies,[23,24] our study showed that overweight and obesity, as well as earlier onset of maturation were mostly found in white subjects followed by

Table 5 Differences between means (±s.d.) of the anthropometric indices of stunted and nonstunted boys, 10–15-y old, measured by *t*-test

Age (y)	*Indices*	*Stunted (n = 134)*	*Non-stunted (n = 470)*	P	Effect size (d)[a]
10	Height (cm)	125.2 (2.3)	136.6 (6.4)	0.0001	1.9
	Weight (kg)	24.2 (1.8)	30.5 (7.8)	0.0001	0.9
	TSF (mm)	7.8 (1.8)	10.6 (6.2)	0.003	0.5
	WC (mm)	52.8 (2.4)	57.5 (6.3)	0.0001	0.8
	MUAC (mm)	16.6 (1.2)	18.9 (3.5)	0.0001	0.7
	BMI (kg/m^2)	15.5 (1.5)	16.2 (3.1)	0.19	0.3
	TSF+SSF (mm)	13.8 (2.6)	18.1 (11.6)	0.008	0.4
11	Height (cm)	130.0 (4.9)	141.9 (6.7)	0.0001	1.9
	Weight (kg)	25.8 (3.3)	32.3 (5.6)	0.0001	1.2
	TSF (mm)	7.1 (2.0)	9.3 (3.5)	0.0002	0.7
	WC (mm)	53.9 (2.7)	57.9 (4.5)	0.0001	1
	MUAC (mm)	17.2 (2.1)	18.8 (2.0)	0.004	0.8
	BMI (kg/m^2)	15.3 (1.7)	16.0 (2.1)	0.08	0.4
	TSF+SSF (mm)	12.5 (3.1)	15.7 (5.7)	0.0004	0.6
12	Height (cm)	134.3 (5.0)	146.5 (5.8)	0.0001	2.2
	Weight (kg)	28.3 (4.8)	36.3 (7.5)	0.0001	1.2
	TSF (mm)	9.2 (4.4)	10.8 (5.9)	0.11	0.3
	WC (mm)	56.4 (4.3)	60.4 (6.2)	0.0002	0.7
	MUAC (mm)	18.0 (2.0)	20.0 (2.9)	0.0001	0.7
	BMI (kg/m^2)	15.6 (1.9)	16.8 (2.8)	0.01	0.5
	TSF+SSF (mm)	15.6 (7.2)	19.0 (11.1)	0.05	0.3
13	Height (cm)	139.6 (3.5)	153.5 (7.8)	0.0001	2
	Weight (kg)	32.7 (6.0)	41.7 (11.0)	0.0001	0.9
	TSF (mm)	10.5 (7.3)	10.1 (4.7)	0.80	−0.1
	WC (mm)	59.8 (5.2)	63.2 (7.5)	0.02	0.5
	MUAC (mm)	18.9 (2.9)	20.9 (3.2)	0.01	0.6
	BMI (kg/m^2)	16.7 (2.6)	17.5 (3.2)	0.24	0.3
	TSF+SSF (mm)	18.9 (13.9)	18.1 (8.7)	0.81	−0.1
14	Height (cm)	143.5 (4.8)	160.4 (7.8)	0.0001	2.3
	Weight (kg)	34.9 (4.8)	46.2 (10.7)	0.0001	1.1
	TSF (mm)	9.4 (3.4)	9.7 (7.4)	0.78	0.05
	WC (mm)	59.2 (3.2)	64.1 (7.4)	0.0001	0.7
	MUAC (mm)	19.1 (1.6)	21.5 (2.9)	0.0001	0.9
	BMI (kg/m^2)	17.0 (2.3)	17.9 (3.3)	0.19	0.3
	TSF+SSF (mm)	16.5 (6.6)	17.9 (14.6)	0.53	0.1
15	Height (cm)	151.2 (4.9)	167.0 (7.2)	0.0001	2.3
	Weight (kg)	39.5 (6.6)	53.5 (10.8)	0.0001	1.4
	TSF (mm)	7.3 (2.4)	10.0 (7.7)	0.01	0.4
	WC (mm)	61.8 (5.2)	66.8 (9.6)	0.002	0.6
	MUAC (mm)	20.6 (2.6)	23.7 (3.0)	0.0001	1.1
	BMI (kg/m^2)	17.2 (2.2)	19.1 (3.3)	0.003	0.6
	TSF+SSF (mm)	14.4 (4.7)	19.6 (15.7)	0.02	0.4

[a]Effect size (*d*) is the difference in means between stunted and nonstunted children, expressed in pooled s.d.; guideline values according to Cohen[19]: small effect size: $d<0.2$; medium effect size: $d=0.2$–0.8; large effect size: $d>0.8$.

black subjects. It is possible that other confounding factors could have played a role in this outcome, for example low income of most black families.[21] The mean age at menarche of white girls was earlier (12.53 y, 95% CI 12.24, 12.82) than in black girls (12.95 y, 95% CI 12.77, 13.15).

Gender and age (vulnerable periods of life)

The period from childhood to adolescence and then from adolescence to adulthood is considered a particularly nutritionally vulnerable time. Not only does significant physical change take place during these times, but food habits and lifestyles also evolve that form the basis for long-term health. At least two and possibly three critical periods exist in childhood for the development of later obesity and its attendant complications of diabetes, hypertension, hypercholesterolaemia and cardiovascular disease.[7]

In our study, it is clear that the following gender differences exist. In particular, mean TSF is smaller at a later age in boys than in girls. This is related to girls entering

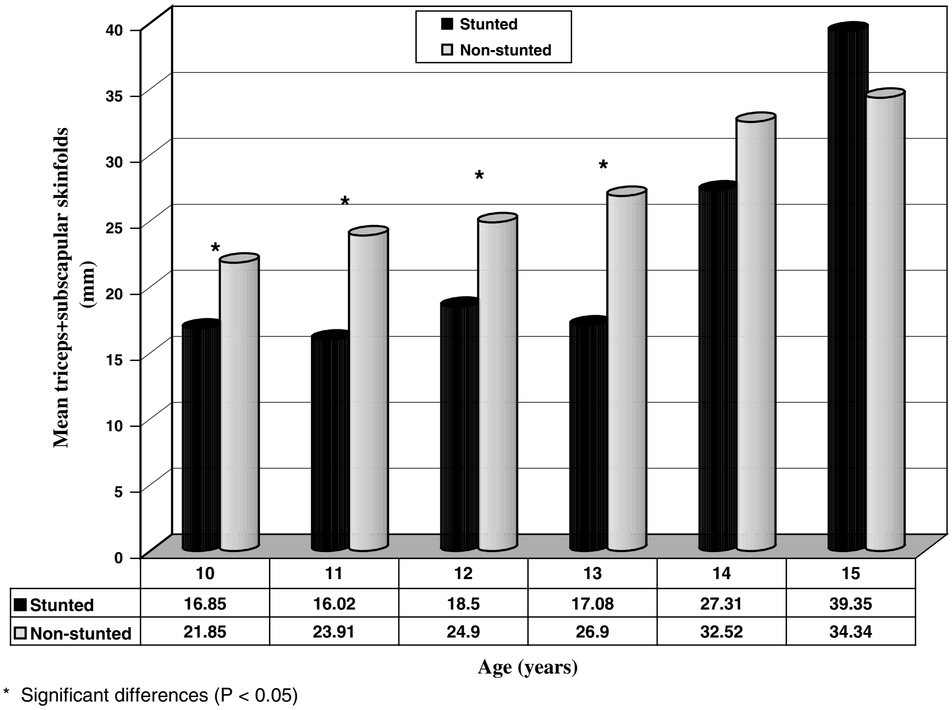

Figure 1 Mean sum of triceps and subscapular skinfolds of stunted compared to nonstunted girls.

Table 6 Influence of gender on the relationship between stunting and prevalence of overweight and obesity

Gender	*Prevalence of stunting (n%)*	*Prevalence of overweight and obesity (n%)*	*Prevalence of overweight and obesity in stunted children (n%)*	*Prevalence of overweight and obesity in nonstunted children (n%)*	*Odds ratio of overweight and obesity being stunted*	*Confidence interval*
Boys	134 (22.2%)	34 (5.6%)	4 (3.0%)	30 (6.4%)	0.45	0.16, 1.30
Girls	105 (16.2%)	65 (10.1%)	6 (5.7%)	59 (10.9%)	0.50	0.21, 1.19
Total	239 (19.1%)	99 (7.9%)	10 (4.2%)	89 (8.8%)	/	/

puberty sooner than boys. Furthermore, WC in boys is slightly smaller at 14 y; however, the girls' WC suddenly increases at 14 and 15 y of age (Tables 4 and 5). This could be explained by the increased muscularity of boys and increased adiposity of girls at the onset of puberty.[7] Linear growth slowed down a bit at 10 y of age in boys, and at 15 y of age in girls. In girls, a possible explanation is the onset of puberty. The increase in the TSF + SSF in the girls coincides with the onset of menarche (Table 4).

In girls, menarcheal status furnishes reference data that are less detailed but easier to collect, in order to evaluate puberty-related BMI. O'Dea and Abrahams.[25] reported significantly lower BMI of age-matched premenarche *vs* postmenarche girls. However, multivariate tests demonstrate that menarcheal status did not influence BMI when corrected by pubertal stage and age.[26] They demonstrated that degree of pubertal maturation has a greater influence on BMI than age in both genders, but this is more evident in girls.[26] The latter is evident in our study too (Tables 3 and 4). As mentioned earlier, the stunting at 14 and 15 y of age in girls could be related to a late onset of menarche. Stunted girls had a significantly higher age at menarche (13.37 y, 95% CI 12.80, 13.95) than nonstunted girls (12.73 y, 95% CI 12.58, 12.89). This can be explained by the fact that menstruation usually begins a little more than 1 y after peak linear growth is attained,[26] and stunted girls apparently reached peak velocity in linear growth later than nonstunted girls.

Anthropometric status

The stunted girls' mean WC gradually increased up to age 13 y and then there was a sudden increase at age 14 and 15 y. This coincided with a rapid increase in BMI and TSF + SSF at ages 14 and 15 y in the stunted girls (Table 4). Impact of abdominal visceral fat accumulation on metabolic derangement is now under extensive study in adults. Abdominal visceral fat has recently been measured in children.[27–30] These studies have suggested that deleterious effects of visceral adipose tissue on blood lipid risk factors seen in adults are already present in children. Predicting visceral fat from anthropometric measures such as WC, WHR and sagittal diameter has been attempted in both adults and children.[27–30] To date, no single anthropometric index has yet been generally accepted to be superior to others as a surrogate of visceral fat measurement in children, but WC has been suggested as a tool in epidemiological studies to identify children with risk factors for noncommunicable diseases.[30] Previous studies' results suggested that the type of obesity changes with age in obese children. The older obese children tended to gain more fat at the umbilical level than the younger ones, and this was considered as the general worsening of body build during growth in obese children.[30] Similarly, in our study, the older stunted girls exhibit more fat at the umbilical level than the younger ones. Even though these stunted girls were not overweight, they exhibited a tendency to increase in WC at an older age (Table 4).

From Tables 4 and 5 it is clear that the effect size of all subjects for weight and height was large. This is indicative of an important difference in weight and height, respectively, in stunted and nonstunted subjects. It is imperative to note that the mean BMI of the 15-y-old stunted girls was greater, although not significantly, than the mean BMI of the nonstunted girls. The mean BMI values of both the stunted and nonstunted girls at 14 and 15 y were closer to the published cutoff points for overweight according to Cole *et al*,[17] namely 23.3 and 23.9 kg/m^2, respectively, than at earlier ages.

In the present study, the stunted 15-y-old girls had a greater mean TSF + SSF than the nonstunted girls (Table 4, Figure 1). This is indicative of the stunted girls displaying a tendency to gain fat rapidly after 14 y of age despite the stagnation of linear growth. In other studies, it was found that girls had almost 50% greater skinfold thickness than boys at all ages[25,31] and the percentage body fat was lower in obese boys than in obese girls.[28] In a study carried out on shantytown children from Sao Paulo, Brazil, the authors found that low energy expenditure may be a risk factor for weight gain in susceptible populations, this indicates that low energy expenditure may help to explain the increased risk of excess weight gain leading to obesity among shantytown girls compared with shantytown boys.[32]

Stunting and obesity

The association between stunting and overweight or obesity was determined by using the odds ratio calculation.[20] The odds ratio for stunted children to be obese was 0.45 (95% CI 0.16, 1.30) and 0.50 (95% CI 0.21, 1.19) for the boys and girls, respectively (Table 6). This was an indication that both the stunted girls and the boys are significantly less likely to be overweight. The same observation was made by Jinabhai *et al.*[33] in a recent study in the Kwazulu-Natal province of South Africa. The researchers did not measure subcutaneous fat or differences between pre- and postmenarcheal girls. However, in the present study, stunted girls showed a tendency to gain more body fat after 14 y of age. Furthermore, stunted children living in urban areas had similar means of BMI and TSF + SSF as children of normal height (Table 2), indicating that although urban stunted children were significantly shorter, they had similar subcutaneous fat stores as nonstunted children. The urban socioeconomic environment could have favoured fat deposition in these children,[1] since they tended to have higher means of BMI and TSF + SSF than stunted children from rural and informal settlement areas. It was, however, evident that unlike many areas in other developing countries,[1] there appeared to be no strong association between overweight or obesity and stunting. Older stunted girls exhibited more fat at the umbilical level than the younger ones. This could indicate that there is a tendency for stunting and overweight to go together in girls older than 14 y. This association may be due to slowed growth and a changed hormonal response due to poor dietary intakes.[1] Alternatively, the smaller lean body mass, resulting in lower basal metabolic rate could favour fat deposition instead of linear growth at an adequate energy intake.[13,32]

In conclusion, there is no significant association between stunting and overweight or obesity among 10–15-y-old children in the North West Province of South Africa. However, stunted girls showed a tendency to have more subcutaneous fat and to gain fat at the umbilical level than nonstunted girls even though at these ages (10–15 y) they were still underweight. This tendency in stunted girls coincided with the onset of menarche and was apparently more evident in urban areas. Therefore, a better understanding of the critical periods for the development of adiposity, stunting and its sequelae is required. This may also serve to focus preventive and therapeutic efforts on developmental stages, when these efforts are likely to be most cost effective. Further investigations should, therefore, assess disease risks for those remaining fat over long periods and for those overweight and being stunted from childhood and adolescence. This should be carried out in a longitudinal study. Further research needs to be carried out about the mechanisms of the above-mentioned criteria or risk factors, in an attempt to formulate specific interventions for the groups at risk of being overweight and stunted in transitional countries.

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References

1 Popkin BM, Richards MK, Monteiro CA. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *J Nutr* 1996; **126**: 3009–3016.
2 Sawaya AL, Grillo LP, Verreschi I, Carlos Da Silva A, Roberts SB. Mild stunting is associated with higher susceptibility to the effects of high-fat diets; studies in a shantytown population in the city of Sao Paulo, Brazil. *J Nutr* 1998; **128** (Supplement): S415–S420.
3 WHO. *Consultation on Obesity. Preventing and Managing the Global Epidemic*. World Health Organization: Geneva; 1998.
4 Puoane T, Steyn K, Bradshaw D, Laubscher R, Fourie J, Lambert V, Mbananga N. Obesity in South Africa: the South African demographic and health survey. *Obes Res* 2002; **10**: 1038–1048.
5 Aristimuno GG, Foster TA, Voors AW, Srinivasan SR, Berenson GS. Influence of persistent obesity in children on cardiovascular risk factors. *Circ* 1984; **69**: 895–904.
6 Dipietro L, Mossenberg HO, Stunkard AJ. A 40-year history of overweight children in Stockholm: lifetime overweight, morbidity and mortality. *Int J Obes* 1994; **18**: 585–590.
7 Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr* 1994; **59**: 955–959.
8 Martorell R, Khan LK, Schroeder DG. Reversibility of stunting: epidemiological finding in children from developing countries. *Eur J Clin Nutr* 1994; **48** (1): S45–S57.
9 Waterlow JC. Causes and mechanisms of linear growth retardation (stunting). *Eur J Clin Nutr* 1994; **481** (Supplement): S1–S4.
10 Trowbridge FL, Marks JS, Romana GL, Mandrid S, Boutton TW, Klein PD. Body composition of Peruvian children with short stature and high weight-for-height II. Implications for the interpretation for weight-for-height as an indicator of nutritional status. *Am J Clin Nutr* 1987; **45**: 513–525.
11 Waterlow JC. *Protein Energy Malnutrition*. Edward Arnold: London; 1992. p 407.
12 Sawaya AL, Dallal G, Solymos G. Obesity and malnutrition in a shantytown population in the city of Sao Paulo, Brazil. *Obes Res* 1995; **3** (Supplement): S107–S115.
13 Trowbridge FL. Prevalence of growth stunting and obesity. Pediatric nutrition surveillance system. *CDC Surveillance Summ* 1982; **32**: 23SS–27SS.
14 Norton K, Olds T. *Anthropometrica. A Textbook of Body Measurement for Sports and Health Courses*. University of New South Wales Press: Sydney; 1996. p 411.
15 Ross WD, Marfell-Jones MJ. Kinantropometry. In: Macbougal JD, Wenger HA, Green HJ (eds). *Physiological Testing of the High Performance Athlete*. Human kinetics: Champaign; 1991. pp 223–308.
16 Centers of Disease Control. 2000 CDC growth charts: United States. Hyattsville, MD. National Center for Health Statistics. 2000, http://www.cdc.gov/growthcharts.
17 Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *Br Med J* 2000; **320**: 1240–1243.
18 Thomas JR, Nelson JK. *Research Methods in Physical Activity*. Human kinetics publishers: Champaign; 1996. p 485.
19 Cohen J. *Statistical Power Analysis for the Behavioral Sciences*, revised edn. Academic Press Inc: New York; 1977.
20 Bland JM, Altman DG. The odds ratio. *Br Med J* 2000; **320**: 1468.
21 Pretoria Stats SA. *Living in North West. Selected Findings of the 1995 October Household Survey*. Statistics South Africa: Pretoria; 1985. pp 7.
22 Harris NS, Crawford PB, Yangzom Y, Pinzo L, Gylatsen P, Hudes M. Nutritional and health status of Tibetan children living at high altitudes. *N Eng J Med* 2001; **344/5**: 341–347.
23 Freedman DS, Srinvasan SR, Webber LS, Burke GL, Berenson GS. Black–white differences in serum lipoproteins during sexual maturation: the Bogalusa Heart Study. *J Chr Dis* 1987; **40**: 309–318.
24 The National Heart, Lung and Blood Institute growth and health study research group (NHLBI). Obesity and cardiovascular disease risk factors in black and white girls; the NHLBI growth and health study. *Am J Publ Health* 1992; **82/12**: 1613–1620.
25 O'Dea J, Abrahams S. Should body mass index be used in young adolescents? *Lancet* 1995; **345**: 657.
26 Bini V, Celi F, Bertioli MG, Bacosi ML, Stella P, Giglio P, Tosti L, Falorni A. Body mass index in children and adolescents according to age and pubertal stage. *Eur J Clin Nutr* 2000; **54**: 214–218.
27 Freedman DS, Serdula MK, Srinivasan SR, Berenson GS. Relation of circumferences and skinfold thicknesses to lipid and insulin concentrations in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr* 1999; **69**: 308–317.
28 Asayama K, Oguni T, Hayashi K, Dobashi K, Fukunaga Y, Kodera K, Tamai H, Nakazawa S. Critical value for the index of body fat distribution based on waist and hip circumference and stature in obese girls. *Int J Obes Relat Metab Disord* 2000; **24**: 1026–1031.
29 Musaiger AO, Gregory WB. Profile of body composition of school children (6–18 y) in Bahrain. *Int J Obes Relat Metab Disord* 2000; **24**: 1093–1096.
30 Asayama K, Hayashi K, Hayashibe H, Uchida N, Kodera K, Nakazawa S. Relationships between an index of body fat distribution (based on waist and hip circumferences) and stature, and biochemical complications in obese children. *Int J Obes Relat Metab Disord* 1988; **22**: 1209–1216.
31 Steyn NP, Wicht CL, Rossouw JE, Van Wyk Kotze TJ, Van Eck M. Nutritional status of 11-year-old children in the Western Cape. II. Anthropometry. *SA J Food Sci Nutr* 1989; **1**: 21–27.
32 Hoffman DJ, Sawaya AL, Coward WA, Wright A, Martins PA, De Nascimento C, Tucker KL, Roberts SB. Energy expenditure of stunted and non-stunted boys and girls living in the shantytowns of Sao Paulo, Brazil. *Am J Clin Nutr* 2000; **72**: 1025–1031.
33 Jinabhai CC, Taylor M, Sullivan KR. Implications of the prevalence of stunting, overweight and obesity amongst South African primary school children: a possible nutritional transition? *Eur J Clin Nutr* 2003; **57**: 358–365.