SOCIOECONOMIC FACTORS IN CHILDHOOD AND OBESITY IN ADULTHOOD

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ABSTRACT

Objectives: The purpose of this study was to determine whether differences in stature, sitting height ratio (SHR) as a measure of relative leg length, and socioeconomic status (SES) in childhood are related with adult adiposity in a Portuguese sample.

Methods: A sample of 1298 subjects (814 women and 484 men), community-dwellers from Porto, Portugal, who participated in the EPIPorto study, conducted from 1999 to 2003, ranging in age from 18 to 72 years and who had anthropometric and socioeconomic information, was studied. Body stature, sitting height, body weight, waist circumference and skinfolds thicknesses were measured. SES in childhood was assessed through parental occupation. Adiposity was assessed through body mass index (BMI), %body fat and abdominal obesity. Short stature, high SHR and manual parental occupation were taken as proxies for deprivation in early life and their association with adiposity in adulthood was assessed.

Results: In both women and men high SHR due to relatively short legs was associated with higher BMI and overweight/obesity ($BMI \ge 25 Kg/m^2$) when compared with those with lower SHR. Short stature and low SES (manual parental occupation) were not consistently associated with adiposity in adulthood, when adjusting for the confounding effect of age, smoking and educational level.

Conclusions: The exposure to negative environments in childhood can lead to body segment imbalances and, in consequence, an increased risk of obesity in adulthood. The SHR is more sensitive to detect this effect than stature or parental occupation.

Keywords: Height, leg length, sitting height ratio, socioeconomic status, obesity.

INTRODUCTION

Longitudinal studies have shown that poor growth during the foetal, infant and childhood periods is associated with increased risk of obesity and related chronic diseases in adulthood(Law and others 2002; Schroeder and others 1999). A series of prospective studies of adult cohorts have also shown that leg length, as a marker of childhood growth, is inversely associated with risk of obesity (Smith and others 2003; Smith and others 2002; Velásquez-Meléndez and others 2005), cardiovascular disease(Langenberg and others 2003b; Lawlor and others 2003; Lawlor and others 2004), and the metabolic syndrome (Bogin and Varela-Silva 2003; Davey Smith and others 2001; Han and others 1997; Varela-Silva and others 2007). These studies are based on the notion that poor nutrition during growth and development, particularly during the foetal period and first year of life, produces permanent changes in metabolism and organ function. However, an integrative approach to the effects of the early environment has been lacking. Human growth is a complex influence of genomic, epigenetic, endocrine, environmental and socioeconomic factors. It is therefore, important to bring together the multitude of possible factors which can impact on human growth and development in order to understand the aetiology of these conditions.

The use of leg length as a marker of growth status is related to the cephalocaudal growth gradient, which is the pattern of growth common to all mammals. A special feature of human growth is that between birth and puberty the legs grow relatively faster than other post-cranial body segments. For groups of children and youth, short stature due to relatively short legs is generally a marker of an adverse environment during infancy and childhood (Bogin 1999; Leitch 1951).

Although height and leg length are associated with risk of chronic diseases, the sitting height ratio (SHR), defined as (sitting height / stature) x 100, is likely to show a stronger association, because it controls for differences in overall size between individuals(Bogin and Varela-Silva 2010). SHR gives the percentage of total stature that is due to the length of the head, neck and trunk of the body(Bogin and Varela-Silva 2008). As a marker of earlier growth, the SHR of adults is, in part, indicative of net nutritional status, as it results from the synergetic relationship between nutrition, infection, and physical activity(Bogin and Varela-Silva 2003; Bogin and Varela-Silva

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2010; Varela-Silva and others 2007). Other factors, such as familial inheritance and population genetics, also play a part in the determination of SHR. However, when early life environments are adverse, these genetic and familial factors are often secondary to the power of the environment to determine body proportions (Bogin and Varela-Silva 2010).Low socioeconomic status (SES) during the growing years, as measured by the occupation of the parents, can provide information on additional factors which are at play early in life. These include crowded and unsanitary living conditions, exposure to disease, maternal nutrition, poor diet and feeding habits (breastfeeding), child labour, low physiological stimulus and care from parents (Bogin 1999). In Portugal, these living conditions have been modified with the revolution of 1974. Portugal had been a fascist dictatorship for the previous 41 years, with an overall low standard of living for the majority of the population. Following the revolution, the political system gradually changed to a parliamentary democracy, and over the last 30 years the socioeconomic profile of Portugal has become typical of a developed nation. The study presented here provides with the opportunity to investigate, retrospectively, the association between the quality of the environment during the growing years and the risk of obesity in later life in a modern urban Portuguese population.

OBJECTIVES

The goal of this study is to investigate retrospectively the relationship between indicators of poor living conditions during infancy and childhood and the later risk of obesity in adulthood in a Portuguese sample. Adult height and the sitting height ratio are used as markers of growth status during infancy and childhood. Socioeconomic status of the parents is used as an indicator of living conditions during infancy and childhood. Obesity in adulthood is measured at several levels, namely in terms of body mass index (BMI), percentage of body fat estimated from skinfolds thickness and abdominal obesity measured by waist circumference.

METHODS

Participants

The current study is based on the EPIPorto cohort study, a population-based cohort of 2485 Portuguese adults. The target population of this study was permanent residents in the city of Porto, Portugal and the baseline sample was assessed between 1999 and 2003. The participants were recruited by random digit dialing using households as the sampling frame, followed by simple random sampling to select one eligible person among permanent residents in each household(Ramos and others 2004). Initially only individuals older than 39 years were selected and from 2000 on, adults over 18 years old were also eligible, resulting in overrepresentation of those aged 40 years or more. After being selected and informed about study details, each participant was invited to visit the Department of Hygiene and Epidemiology of the University of Porto to complete a demographic, social, behavioural, and medical questionnaire (Ramos and others 2004). In the same session an anthropometric assessment was made of each individual. The proportion of participation was 70% (Ramos and others 2004).

From 2005 to 2008, a follow up evaluation including a second questionnaire and an additional anthropometric assessment were conducted. At this follow up examination, 64.4% of the cohort was reassessed (Figure 1). Since data on sitting height and parents' occupation were collected only at the follow up, the current analysis is based only on participants who attended both evaluations. Since it is safe to assume that sitting height and parental occupation do not change over the course of the two evaluations, it is irrelevant whether they are collected at baseline or follow up. Adiposity indicators, however, were taken from the baseline because at this point in time the sample is more likely representative of the population and uninfluenced by participation in the study.

For the present analysis, all subjects whose age was over 72 years (272 subjects) were excluded because, according to Durnin *et al.* (Durnin and Womersley 1974), the equations to calculate the body fat were not applicable to those aged over 72 years. Thirty individuals with cognitive impairment were excluded because their self-reported data was considered unreliable. Of the remaining 2183 individuals, 68 died over the course of the study, 191 refused to attend the follow up examination and 519 were unreachable. Additionally we excluded 107 participants with missing data on body mass

index (BMI), waist circumference, body fat, smoking and indicators of early-life socioeconomic status (SES). In total, a study sample of 1298 participants aged between 18 and 72 years (814 women and 484 men) with data for all of the variables, was extracted (Figure 1).

Data collection

Information on age, sex, date of birth, education, occupation, parental occupation, smoking history and physical activity were collected by questionnaire. Education level was classified into three groups: \leq 4years, 5-11years or \geq 12years of school enrolment. Both self occupation and parental occupation were dichotomized as non-manual and manual. Parental-occupation was obtained as the highest from both parents' classification or as their father's occupation. Subjects were classified as nonsmokers (never smokers), current smokers (daily or occasional) or former smokers (for at least 6 months). Total and leisure-time physical activity and total energy intake are presented in MET*hour/day and kcal, respectively. Anthropometric measurements included body height, sitting height, body weight, waist circumference and three skinfold thicknesses (triceps, subscapular and supra iliac). Body height and weight were measured with the individuals barefoot and without excess clothing or accessories (Velásquez-Meléndez and others 2005). Body height was measured directly to the nearest 0.1 cm, using a wall stadiometer, with the subject standing and the head in the Frankfurt plane. Sitting height was measured with the subject sitting upright on a base plate, using the same stadiometer and later subtracting the plate's height (Velásquez-Meléndez and others 2005). Body weight was measured to the nearest 0.1 Kg using a digital scale. Waist circumference was measured to the nearest 0.1 cm using a nondistensible tape midway between the lower ribs and iliac crest in the midaxillary line. The three skinfolds were measured with a Harpenden caliper, in the non-dominant side of body. The triceps skinfold was measured at the midpoint of the posterior arm, between the ends of the olecranon and the acromion. The caliper was applied in the vertical axis with the arm extended along the body. The subscapular skinfold was measured in the region immediately below the scapula, with the fold at an angle of 45 degrees to the vertical along the lines of skin cleavage and with the arm resting along the body. The supra iliac skinfold was measured vertically at the midaxillary line and at the midpoint between the last rib and the iliac crest with the arm relaxed. The skinfolds

were clamped between the thumb and forefinger and detached from the body, applying the caliper and reading the fold thickness in millimeters. Three consecutive values were determined for each skinfold. The final value was the mean of the three determinations.

Height below the first quartile of the sample distribution (≤ 152 cm in women and \leq 164cm in men) was considered low, meaning these individuals had short stature as a result of detrimental environment in childhood. The sitting height ratio (SHR) was used as a measure of relative leg length. The SHR is the percentage of total stature that is due to the length of the head, neck and trunk (Bogin and Varela-Silva 2008) and is a better descriptor of body shape than stature or leg length (Bogin and Varela-Silva 2008). SHR and BMI were derived by computation: SHR = (sitting height/height)x100 and BMI =weight $(Kg)/height^2(m^2)$. A SHR above the third quartile of the sample distribution $(\geq 54.14\%$ in women and $\geq 53.29\%$ in men) was considered high, meaning these individuals had relatively short legs for their height as a result of deprivation in early life. BMI was categorized according to the World Health Organization classes (1998): <25Kg/m² (normal weight), 25.0-29.9 Kg/m² (overweight) and \geq 30 Kg/m² (obese). Women and men whose waist circumference were >88cm and >102cm, respectively, were considered to have abdominal obesity, according to the National Cholesterol Education Program - Adult Treatment Panel III (Expert Panel on Detection and Treatment of High Blood Cholesterol in Adults 2001). Body fat, expressed as a percentage of total body mass, was estimated from skinfolds according to Durnin et al. (Durnin and Womersley 1974). %Body fat = [(4,95 / D) - 4,50] * 100 was the equation applied. (D) corresponds to corporal density and was calculated through the density equations according to sex and age (Durnin and Womersley 1974).

Statistical analysis

The participants' characteristics are presented as means for continuous variables (age, total physical activity, leisure-time physical activity, total energy intake, stature, SHR, BMI, body fat and waist circumference) and proportions for categorical variables (education, occupation, parental occupation, short stature, high SHR, smoking, overweight-obesity, abdominal obesity), with their respective 95% confidence intervals (95% CI). The agreement between short stature and high SHR was quantified through the kappa coefficient.

Height, SHR and parental occupation were taken as exposures (independent variables), and their association with adiposity measures (BMI, %body fat and waist circumference) was assessed. After examining the effect of height and SHR categorized by quartiles and excluding the existence of a linear association with adiposity, we arbitrarily dichotomized both by the most deprived quartile (below first quartile for stature and above third quartile for SHR). BMI and waist circumference were analyzed both as continuous variables and dichotomized according to previously defined and universally accepted cut-off points (1998; Expert Panel on Detection and Treatment of High Blood Cholesterol in Adults 2001). Body fat was analyzed as a continuous variable.

The association between the exposures and measures of adiposity was quantified using linear regression models for continuous dependent variables (body mass index, body fat and waist circumference) and binary logistic regression models, for dichotomous dependent variables (overweight-obesity and abdominal obesity). The results are presented as crude, age-adjusted and multivariate-adjusted linear regression coefficients (β) and odds ratios (OR), respectively. Potential confounders were listed *a priori* according to previous knowledge on determinants of adiposity and their association with the exposures. Total and leisure-time physical activity, and energy intake were first assessed as categorical variables, according to the sex-specific quartiles of the study sample. If there was evidence of a linear association with the outcomes, the variable was included in the models as a continuous variable. Potential confounders which were not associated with the outcomes in this sample and did not influence the effect of the main exposure were excluded from the models.

All analyses were stratified by sex due to the pathophysiological differences in adiposity and the differential effect of social exposures by sex. All analyses were weighted for age (18-39 years, \geq 40 years), according to the age composition of the population of Porto at the 2001 census, to account for a skewed age distribution in the baseline sample. All statistical analysis was carried out using STATA® version 9.2.

Ethics

The study was approved by the Ethics Committee of Hospital de S. João and participants gave written informed consent.

RESULTS

Sample characteristics

A description of the study sample is shown on Table 1. The mean age of participants was 47.2 [95% CI (46.2-48.3)] years for women and 45.3 [95% CI (43.8-46.8)] years for men. Almost 60% of this population studied less than eleven years [59.8% of women and 56.8% of men]. More than fifty percent of the individuals had non-manual occupations [56.4%, 95% CI (52.8-60.1) of women and 54.4%, 95% CI (49.3-59.5) of men] and, in the generation of their parents, about a half also had a non-manual occupation.

The average stature for women was 156.9cm [95%CI (156.4-157.3)] and 170.0cm for men [95%CI (169.3-170.7)]. The mean of SHR for women was 53.2% [95% CI (53.1-53.4)] and 52.4% [95% CI (52.3-52.5)] for men. Although a higher proportion of men and women with short stature had high SHR than those with normal-high stature, the agreement between stature below the first quartile and SHR above the third quartile was low (kappa coefficient 0.14 in both men and women). The mean of BMI was over 25 Kg/m² for both sexes [women: 26.3 Kg/m², 95% CI (26.0-26.7); men: 25.8 Kg/m², 95% CI (25.4-26.3)]. In fact, 53.6% of women and 56.9% of men were overweight. Women had a higher percentage of body fat than men [women: 34.9%, 95% CI(34.5-35.4); men: 23.9%, 95% CI (23.3-24.5)]. Most individuals had a normal waist circumference with mean values below 88cm, in women [83.7cm, 95% CI (82.8-84.6)], and below 102cm, in men [90.8cm, 95% CI (89.7-91.9)]. However, a higher percentage of women [32.9%, 95% CI (29.7-36.2)] had abdominal obesity compared with men [10.4%, 95% CI (7.6-13.1)] (Table 1).

A higher percentage of men were current or former smokers (69,1%) compared with women (31.9%). The levels of total physical activity in women were lower than in men [37.3 MET-hour/day 95% CI (36.8-37.8) in women against 38.8 MET-hour/day 95% CI (37.8-39.8) in men]. The same is true for leisure-time physical activity and total energy intake, in which these are also lower for women (Table 1).

Effect of stature and SHR on adiposity

The association between short stature and adiposity, by sex, is shown in table 2. Among women, all crude results demonstrated an association between short stature and BMI, overweight/obesity, body fat, waist circumference and abdominal obesity. After adjustments smoking and education the relationship for age, between overweight/obesity and short stature was the only one maintained. Hence, the risk of overweight/obesity tended to increase in those women whose stature is below 152cm. The likelihood of being overweight/obese increased by 57% [OR=1.57; 95% CI (1.04 to (2.36)] for those with short stature (compared with those with stature > 152cm). The BMI was also associated with short stature but the results were not statistically significant. The waist circumference and abdominal obesity were positively associated with stature. Though the association between stature and waist circumference was statistically significant, the association between abdominal obesity and stature failed to reach statistical significance at the 0.05 level. Women with short stature tended to have smaller waist circumferences and a lower risk of having abdominal obesity. The likelihood of having abdominal obesity decreased by 29% [OR=0.71; 95% CI (0.48 to (1.04)] for those with short stature (compared with those with stature > 152cm). In multivariate analysis, no association was found between body fat and stature.

The positive association between body fat, waist circumference, abdominal obesity and stature was also present among men. However, all the associations were statistically significant and stronger in men than in women. Men with stature below 164cm (short stature) tended to have less body fat, smaller waist circumferences and a lower risk of having abdominal obesity. BMI and overweight/obesity are both inversely associated with stature but results are not statistically significant (Table 2).

Table 3 shows, separately for women and men, the results of the linear and logistic regression analyses, for the association between high SHR and each indicator of adiposity. The BMI for women and men was associated with high SHR, after adjustments for age, education and smoking. For both women and men, BMI increased when the SHR was higher than 54.14% and 53.29%, respectively. This shows that individuals exposed to a detrimental childhood environment and therefore with relative shorter legs (a high SHR) tend to have a greater BMI.

Odds ratios adjusted for age, smoking and education showed a statistically significant association between very high SHR and the risk of being overweight (BMI \geq

25.0 Kg/m2) in both sexes. The risk of being overweight increased with a higher SHR in women and men. Among women, the likelihood of being overweight increased by 52% [OR=1.52; 95% CI (1.03 to 2.24)] for those with a high SHR (compared with those with a SHR < 54.14%). In the case of men, the likelihood of being overweight increased by 80% [OR=1.80; 95% CI (1.06 to 3.06)] for those with a higher SHR (compared with those with a SHR<53.29%). Body fat and waist circumference were not associated with SHR in women and men (Table 3).

Effect of parental occupation on adiposity

The linear regression coefficients (β) and the odds ratios for the association between manual parental occupation and adiposity are presented in Table 4. Among women, there was a strong positive association between all the variables (BMI, overweight-obesity, body fat, waist circumference and abdominal obesity) and manual parental occupation when crude linear regression results are analyzed. Age-adjusted models showed the same strong association. However, when models are adjusted for smoking, education and leisure-time physical activity (only for body fat) this association disappeared altogether. The effect of parental occupation, independent of age and smoking, is fully explained by the level of self education. The education level in men did not have the same effect as in women. The effects of parental occupation were explained by age alone. Interestingly, even after adjustments for age, smoking and education there was still a positive association between overweight and manual parental occupation but it failed to reach statistical significance at the 0.05 level. This suggests that men whose parents had manual occupations are at an increased risk of being overweight [OR=1.55; 95% CI (0.93 to 2.59)] compared with those whose parents had more differentiated (non-manual) occupations.

DISCUSSION

These findings provide evidence for our hypothesis that exposure to poor living conditions during the growth and development period is associated with adiposity in adulthood. The women and men from this population of Porto with relatively short leg length are exposed to a higher risk of being overweight-obese than their counterparts with relatively long legs. Similarly, shorter women (but not shorter men) are also exposed to an increased risk of overweight-obesity compared with their taller counterparts.

The environment is a more powerful force influencing leg length and body proportions than genes (Bogin and Varela-Silva 2010). Also there is evidence that short stature due to relatively short legs (i.e., high SHR) is generally a marker of an adverse environment during childhood (Bogin 1999; Leitch 1951). Conversely, relatively long legs are an indicator of a positive environment during infancy. In this regard, it is important to point out that the present study indentified a relationship between body segment imbalances, a characteristic of inadequate childhood environment, and obesity in adulthood.

The relationship between measures of relative leg length and adiposity was previously reported by other authors. Velásquez-Meléndez *et al.*(Velásquez-Meléndez and others 2005) and Frisancho (Frisancho 2007) studied a similar relationship using the SHR and leg length index, respectively, as measures of relative leg length. The first one studied a group of 669 Brazilian women aged 20-56 years. It was shown that Brazilian women with high SHR presented a higher risk of developing obesity in later life (positive relationship between high SHR and BMI, %body fat and the 3th tertile skinfolds). In the second one 21.021 subjects ranging in age 2 to 90 years who had participated in the third National Health and Nutrition Survey (NHANES III) were studied. A low leg length index was associated with significantly greater amount of body fat (measured by skinfold thickness).

As a marker of earlier growth, short stature has also been associated with a negative environment during childhood and a higher risk of adiposity in adulthood (Sichieri and others 2003; Velásquez-Meléndez and others 1999; Vieira and others 2007). The inverse association between BMI or overweight/obesity and stature, reported in this and previous studies (Sichieri and others 2003; Velásquez-Meléndez and others

1999; Vieira and others 2007) could result simply from the fact that stature is in the denominator in the calculation of BMI. Thus, greater stature is necessarily associated with lower BMI. Compared with high SHR, short stature is additionally associated with smaller waist circumferences and a lower risk of having abdominal obesity for both women and men. Since waist circumference is an absolute measure which does not take into account the overall body size, it is possible that this association merely reflects overall small bodies. The same applies to abdominal obesity since no adjustment in cut-off points are allowed to account for overall body size. Previous similar studies used the waist-to-hip ratio (WHR) instead of waist circumference alone and found opposite results (stature was inversely associated with WHR), which supports our interpretation because the WHR reflects relative amounts of visceral adiposity and accounts for the bone structure at the hip level (Velásquez-Meléndez and others 1999; Vieira and others 2007).

Interestingly, the SHR and stature quartiles do not present a high level of agreement. Therefore, classification of individuals as deprived vary according to the criteria used (high SHR or short stature). SHR is a more sensitive factor to the environment effects than stature and differences in relative legs/trunk proportions between individuals result from growth being affected at different periods with different intensities. This reinforces the idea that stature and SHR convey different information. The SHR controls for differences in overall size between individuals and, therefore, is likely to show a stronger association with obesity and chronic diseases than stature(Bogin and Varela-Silva 2010), as was observed in the current study.

A phenotype of short stature and overweight/obesity are, together, a risk factor for coronary heart disease, diabetes and other metabolic diseases later in life (Azevedo and others 1999; Florêncio and others 2007; Varela-Silva and others 2007). Many epidemiological studies indicate that differences in the proportions of body are associated with significant differences in the components of the metabolic syndrome (Asao and others 2006; Bray and others 2006; Han and others 1997; Langenberg and others 2003b; Lawlor and others 2002) such as glucose intolerance, insulin resistance and hypertension. Also it has been largely reported that differences in the stature or leg length are associated with coronary heart disease (Lawlor and others 2003; Lawlor and others 2004; Lynch and Smith 2005). Hence, one can conclude that the growth deficits produced by negative environmental conditions during the period of growth and development were extensively associated with health status. Metabolic changes, such as diminished fat oxidation and/or increased carbohydrate oxidation, may occur in children with growth impairment as a physiologic compensation in those exposed to an adverse environment or nutritional deprivation. Hoffman *et al.*(Hoffman and others 2000), after studying a group of Brazilian children demonstrated that those with delayed growth had a high respiratory quotient (RQ), indicating a relatively low fat oxidation that predispose them to weight gain and fat storage than those with advanced growth. RQ is the ratio between carbon dioxide (CO₂) produced and oxygen (O₂) consumed. Generally, a high RQ indicating a relatively low fat oxidation will tend to cause increased fat deposition.

Fetal environmental conditions and genetic factors also play a role in the developing of overweight/obesity, however, they are beyond the scope of this study.

The association of obesity with low SES is less pronounced than with that of high SHR. Low SES (manual parental-occupation) in childhood was associated with an increased risk of obesity in adulthood among Portuguese women, but this is not independent of other factors and is explained by women's education level. Parents with manual occupations may have fewer material resources to offer a better education and offer less stimuli compared with those with non-manual occupations. Inversely, the non-manual parental occupation may lead to more years of schooling of children. In contrast, age explains the association between early life SES and adiposity among men. Since this is a cross-sectional study we are not able to disentangle age from birth cohort. Older birth cohorts are more likely to have had low SES in early life due to historical reasons, and are more likely to be obese at present due to the trend for adiposity to increase with age.

We have only analyzed parental occupation (chiefly the occupation of the father because most mothers worked at home), which is a better indicator of the economic resources in the family. Parental education is a stronger indicator of the quality of growth environment assessing the quality of health care and nutrition, but it was not assessed in this study.

Other studies have found an association between poor socioeconomic conditions in childhood and obesity (Ball and Mishra 2006; Garn and others 1981; Langenberg and others 2003a; Power and Matthews 1997). In 1997, Power *et al.* (Power and Matthews 1997) studied the relationship between social position (based on the father's occupation) at birth and adult health inequalities using a cohort of people who have been followed up for 33 years since birth. By adulthood, participants born into lower social classes had experienced poorer growth in childhood and greater likelihood of obesity in adulthood than those born into higher ones. According to Garn *et al.*(Garn and others 1981) low-income girls and boys (low SES), although initially leaner than their high-income peers, gain more fat during adolescence and beyond and, in consequence, are fatter in later life. Being born into a particular social class cannot itself "cause" obesity. Instead, social class is related to material circumstances of life and access to information which promotes healthy life styles, which ultimately influence energy balance (Parsons and others 1999).

It is plausible that obesity patterns could vary according to the component of SES used as the basis for classifying a sample (Wardle and others 2002). The variable parental occupation used as a measured of SES, may not to be the most suitable indicator of SES of this population and this may explain the absent of a relationship between a low SES during early life and a higher risk of obesity in adulthood.

This population seems to be biased towards the higher socioeconomic status: more than fifty percent of the individuals had non-manual occupations and, in the generation of their parents, about a half also had a non-manual occupation. Individuals with higher SES are more likely to participate in the study. Additionally, we only used individuals that also participated in the follow up evaluation. People with higher SES are more likely to survive and to accept to participate again. A high SHR was much more prevalent among women born after 1960 (data not shown). Since Portugal has experienced dramatic social and economic changes since the 1960s, it was expected there to be more deprived women before 1960 than after. Our hypothesis is that women born before 1960, being deprived, had less probability to participate in this study because they can have died or refused to participate. This would be a selection bias.

The SHR can be overestimated in individuals with high levels of gluteo-femoral fat, which contributes to a higher sitting height, therefore underestimating the relative contribution of the lower limb to total stature (Bogin and Varela-Silva 2008). Since obese people have more gluteo-femoral fat, this would result in an overestimation of the association between SHR and adiposity due to a differential information bias. This should not, however, fully explain this association because it has been confirmed by studies in which leg length is measured radiologically and thus free of this measurement error.

CONCLUSION

High SHR is associated with BMI and overweight/obesity in this group of Portuguese women and men. This suggests that low relative leg length is a risk factor for overweight/obesity in both sexes. Short stature was shown to be associated with overweight-obesity, only for women. Factors that affect the increase of SHR and the decrease of stature (such as environmental, nutritional and genetic) should be studied in others populations as a potential cause of increased obesity in societies. The association between obesity in adulthood and low SES in childhood is less pronounced compared with the association between short stature, high SHR and obesity, and it is fully explained by intermediate factors such as age/birth cohort and the subjects' educational level.The SHR is more sensitive to assess the effect poor living conditions during childhood and obesity in adulthood than stature or parental occupation.

LITERATURE CITED

- 1998. Guidelines, Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. National Institutes of Health. Obes. Res. 6 (Suppl. 2) 51S–209S.
- Asao K, Kao WHL, Baptiste-Roberts K, Bandeen-Roche K, Erlinger TP, Brancati FL. 2006. Short Stature and the Risk of Adiposity, Insulin Resistance, and Type 2 Diabetes in Middle Age. Diabetes Care 29(7):1632-1637.
- Azevedo A, Ramos E, Hafe Pv, Barros H. 1999. Upper-body adiposity and risk of myocardial infarction. J Cardiovasc Risk 6:321-325.
- Ball K, Mishra GD. 2006. Whose socioeconomic status influences a woman's obesity risk: her mother's, her father's, or her own? Int. J. Epidemiol. 35(1):131-138.
- Bogin B. 1999. Patterns of human growth, 2nd Ed. Cambridge University Press, Cambridge.
- Bogin B, Varela-Silva MI. 2003. Anthropometric Variation and Health: A Biocultural Model of Human Growth. Journal of Children's Health 1(2):149 172.
- Bogin B, Varela-Silva MI. 2008. Fatness biases the use of estimated leg length as an epidemiological marker for adults in the NHANES III sample. Int. J. Epidemiol. 37(1):201-209.
- Bogin B, Varela-Silva MI. 2010. Leg Length, Body Proportion, and Health: A Review with a Note on Beauty. International Journal of Environmental Research and Public Health 7(3):1047-1075.
- Bray I, Gunnell D, Holly JMP, Middleton N, Smith GD, Martin RM. 2006. Associations of Childhood and Adulthood Height and the Components of Height with Insulin-Like Growth Factor Levels in Adulthood: A 65-Year Follow-Up of the Boyd Orr Cohort. J Clin Endocrinol Metab 91(4):1382-1389.
- Davey Smith G, Greenwood R, Gunnell D, Sweetnam P, Yarnell J, Elwood P. 2001. Leg length, insulin resistance, and coronary heart disease risk: The Caerphilly Study. Journal of Epidemiology and Community Health 55(12):867-872.
- Durnin JVGA, Womersley J. 1974. Body fat assessed from total body density and its estimation from skinfold thickness : measurements on 481 men and women aged from 16 to 72 years. Br. Jr. Nutr. 32:77-97.
- Expert Panel on Detection E, Treatment of High Blood Cholesterol in Adults. 2001. Executive Summary of the Third Report of the National Cholesterol Education

Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 285(19):2486-2497.

- Florêncio TT, Ferreira HS, Cavalcante JC, Stux GR, Sawaya AL. 2007. Short stature, abdominal obesity, insulin resistance and alterations in lipid profile in very lowincome women living in Maceio, north-eastern Brazil. European Journal of Cardiovascular Prevention & Rehabilitation 14(2):346-348.
- Frisancho AR. 2007. Relative leg length as a biological marker to trace the developmental history of individuals and populations: Growth delay and increased body fat. American Journal of Human Biology 19(5):703-710.
- Garn S, Hopkins P, Ryan A. 1981. Differential fatness gain of low income boys and girls. Am J Clin Nutr 34(8):1465-1468.
- Han TS, Hooper JP, Morrison CE, Lean ME. 1997. Skeletal proportions and metabolic disorders in adults. European Journal Of Clinical Nutrition 51(12):804-809.
- Hoffman DJ, Sawaya AL, Verreschi I, Tucker KL, Roberts SB. 2000. Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate and fat oxidation in shantytown children from Sao Paulo, Brazil. Am J Clin Nutr 72(3):702-707.
- Langenberg C, Hardy R, Kuh D, Brunner E, Wadsworth M. 2003a. Central and total obesity in middle aged men and women in relation to lifetime socioeconomic status: evidence from a national birth cohort. Journal of Epidemiology and Community Health 57(10):816-822.
- Langenberg C, Hardy R, Kuh D, Wadsworth ME. 2003b. Influence of height, leg and trunk length on pulse pressure, systolic and diastolic blood pressure. Journal of Hypertension 21(3):537-543.
- Law CM, Shiell AW, Newsome CA, Syddall HE, Shinebourne EA, Fayers PM, Martyn CN, de Swiet M. 2002. Fetal, Infant, and Childhood Growth and Adult Blood Pressure: A Longitudinal Study From Birth to 22 Years of Age. Circulation 105(9):1088-1092.
- Lawlor, Lawlor D, Ebrahim, Ebrahim S, Davey S, Smith GD. 2002. The association between components of adult height and Type II diabetes and insulin resistance: British Women's Heart and Health Study. Diabetologia 45(8):1097-1106.
- Lawlor DA, Smith GD, Ebrahim S. 2003. Association between leg length and offspring birthweight: partial explanation for the trans-generational association between

birthweight and cardiovascular disease: findings from the British Women's Heart and Health Study. Paediatric & Perinatal Epidemiology 17(2):148-155.

- Lawlor DA, Taylor M, Davey Smith G, Gunnell D, Ebrahim S. 2004. Associations of components of adult height with coronary heart disease in postmenopausal women: the British women's heart and health study. Heart 90(7):745-749.
- Leitch I. 1951. Growth and Health. British Journal of Nutrition 5(01):142-151.
- Lynch J, Smith GD. 2005. A life course approach to chronic disease epidemiology. Annual Review of Public Health 26(1):1-35.
- Parsons TJ, Power C, Logan S, Summerbell CD. 1999. Childhood predictors of adult obesity: a systematic review. International Journal Of Obesity And Related Metabolic Disorders: Journal Of The International Association For The Study Of Obesity 23 Suppl 8:S1-S107.
- Power C, Matthews S. 1997. Origins of health inequalities in a national population sample. The Lancet 350(9091):1584-1589.
- Ramos E, Lopes C, Barros H. 2004. Investigating the effect of nonparticipation using a population-based case-control study on myocardial infarction. Annals of Epidemiology 14(6):437-441.
- Schroeder DG, Martorell R, Flores R. 1999. Infant and Child Growth and Fatness and Fat Distribution in Guatemalan Adults. Am. J. Epidemiol. 149(2):177-185.
- Sichieri R, Silva CVC, Moura AS. 2003. Combined effect of short stature and socioeconomic status on body mass index and weight gain during reproductive age in Brazilian women. Brazilian Journal of Medical and Biological Research 36:1319-1325.
- Smith PK, Bogin B, Varela-Silva MI, Loucky J. 2003. Economic and anthropological assessments of the health of children in Maya immigrant families in the US. Economics & Human Biology 1(2):145-160.
- Smith PK, Bogin B, VarelaSilva MI, Orden B, Loucky J. 2002. Does Immigration Help or Harm Children's Health? The Mayan Case. Social Science Quarterly 83(4):994-1002.
- Varela-Silva MI, Frisancho AR, Bogin B, Chatkoff D, Smith PK, Dickinson F, Winham D. 2007. Behavioral, environmental, metabolic and intergenerational components of early life undernutrition leading to later obesity in developing nations and in minority groups in the U.S.A. Collegium Antropologicum 31(1):39-46.

- Velásquez-Meléndez G, Martins I, Cervato A, Fornés N, Marucci MdF, Coelho L. 1999. Relationship between stature, overweight and central obesity in the adult population in São Paulo, Brazil. International Journal of Obesity 23:639-644.
- Velásquez-Meléndez G, Silveira EA, Allencastro-Souza P, Kac G. 2005. Relationship between sitting-height-to-stature ratio and adiposity in Brazilian women. American Journal of Human Biology 17(5):646-653.
- Vieira VCR, Fransceschini SdCC, Fisberg M, Priore SE. 2007. Stunting: its relation to overweight, global or localized adiposity and risk factors for chronic noncommunicable diseases. Revista Brasileira de Saúde Materno Infantil 7:365-372.
- Wardle J, Waller J, Jarvis MJ. 2002. Sex Differences in the Association of Socioeconomic Status With Obesity. Am J Public Health 92(8):1299-1304.