Maternal Anthropometry Is Associated with the Body Mass Index and Waist:Height Ratio of Offspring at 23 Years of Age1,2

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Abstract

Obesity is considered a public health problem worldwide. Evidence from epidemiologic studies has shown that early undernutrition may be a determinant of obesity later in life. Longitudinal studies are scarce in the setting of low- and middle-income countries. In Pelotas, southern Brazil, a birth cohort has been followed since 1982. Between 2004 and 2005, 4297 members of this cohort were interviewed. The aim of this study is to describe the association between maternal anthropometry and offspring BMI and waist:height ratio (WHtR) at 23 y of age in individuals from the 1982 Pelotas birth cohort. Independent variables included maternal prepregnancy BMI and maternal height, obtained in 1982. The BMI and WHtR of the offspring at 23 y of age were the outcomes. The analysis was stratified by sex and restricted to those cohort members belonging to the lower income group. Multivariable linear regression was adjusted for potential confounding or mediating factors according to a hierarchical framework. For each unit of maternal prepregnancy BMI, the offspring BMI increased 0.65 and 0.63 kg/m² in men and women, respectively (P < 0.001). Maternal prepregnancy BMI was directly associated with offspring WHtR in both sexes (P < 0.001). On the other hand, maternal height was not associated with offspring BMI or WHtR. In conclusion, our study suggests that maternal anthropometry is associated with the offspring BMI and WHtR at 23 y of age.

Introduction

Obesity is a prevalent nutritional disorder affecting middle- and high-income countries (1). The relationship between body fat distribution and noncommunicable diseases, particularly cardiovascular diseases and diabetes, is well established (1). Several studies have suggested that intrauterine life is a critical period in the development of later obesity (2–5). Other studies suggest that intrauterine growth restriction, especially when followed by excessive weight gain in childhood, is associated with increased risk of several chronic diseases (6,7). Research on the developmental origins of adult disease in low- and middle-income countries is particularly important if we consider the presence of early undernutrition followed by diets and environments including high energetic density (8).

The long-term effect of maternal undernutrition during pregnancy on offspring body size has been studied. Maternal undernutrition during early gestation was associated with higher BMI and waist circumference (WC) in 50-y-old women (3). Some mechanisms have been proposed to explain this association. First, maternal undernutrition can affect the sensitivity of the fetus's hypothalamic-pituitary-adrenal axis (9), which, in turn affects the appetite and the levels of physical activity later on (4,5). In addition, specific components of the maternal diet, such as a high methionine intake, could modify the expression of genes associated with obesity (10).

On the other hand, maternal body size is strongly associated with the size of newborn children (11), and there is evidence that low birth weight (≤2500 g) is associated with the distribution pattern of central adiposity later in life (12). Because maternal low height, as well as restrictions on energy consumption, during pregnancy are more common among women from a low-socioeconomic status (13), our aim was to describe the association between a couple of maternal characteristics (prepregnancy BMI and maternal height) on the offspring indexes related to obesity at 23 y of age in the 1982 Pelotas birth cohort, in Brazil, a low- to middle-income country.

Methods

The original cohort's baseline characteristics have been previously reported (14). Briefly, the study was conducted in Pelotas, a city in southern Brazil that currently has 340,000 inhabitants. The population
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is mostly white (83%) and displays Portuguese or Spanish background (15). The study was launched as a perinatal health survey, including all 6011 infants born in 3 maternity hospitals (over 99% of all births in the urban area). The 5914 liveborn infants were weighed using calibrated pediatric scales and their mothers were interviewed to determine socioeconomic, demographic, and health-related variables. In 1984 and in 1986, all households in the city were visited in search of children born in 1982 (the children's average age was 20 and 43 mo, respectively). The follow-up rates were 87.3% in 1984 and 84.5% in 1986. The parents were interviewed and the children were weighed and measured with portable equipment.

From October 2004 to August 2005, −98,000 households in the city were traced. During this visit, 4297 cohort members were located and interviewed (2214 men and 2083 women; mean age: 23 y). The follow-up rate was 77%, including deaths (n = 282). The questionnaire included topics such as health status, as well as demographic and socioeconomic characteristics. The anthropometric measures obtained were weight, height, and WC. Earlier publications provide further details about each visit and about the follow-up rates (16,17).

The confidentiality of the information was ensured and an informed consent was obtained at all phases of the study (a verbal consent in the 1980s and a written consent between 2004 and 2005). The Medical Ethics Committee at the University of Pelotas, affiliated with the Brazilian Medical Research Council, approved the study protocol (16).

Because maternal anthropometric measurements were related to factors associated with low income, short stature, e.g. was due to maternal energy restriction rather than to genetic factors, it was decided to restrict the analysis to those subjects from the low-income family group (≥3 times the minimum wage). In 1982, most children born in the city of Pelotas belonged to the lower income group (monthly family income ≤ US$100) (14). On the other hand, 23-y-old women who were pregnant (>3 mo of pregnancy) or who had a child in the 3-mo period before the 2004–2005 follow-up interview (n = 90) were excluded from the analysis.

Maternal prepregnancy BMI and maternal height comprise the main exposure variables in this study. The BMI was estimated dividing the prepregnancy weight in kilograms, as referred by the women [the information was confirmed on the antenatal care register card, in women who were at the beginning of their pregnancy (up to 12 wk)], by the square of the maternal height in meters. The maternal prepregnancy BMI was used as a continuous or categorical variable according the cutoff points (undernutrition, ≤ 18.5; normal, 18.6–24.9; overweight, 25.0–29.9; obesity, ≥30 kg/m²) recommended by the WHO (18). Maternal height was measured by the research team soon after the women's admission to the maternity hospital in 1982. For this analysis, maternal height was also treated as a continuous and ordinal variable (tertile distribution of own sample).

In this study, the dependent variables included offspring BMI and waist:height ratio (WHtR) at age 23 y. These variables were collected during the examination carried out between 2004 and 2005. Standing height was measured to the nearest 1 mm with barefooted subjects using a stadiometer (CMS). Subjects were weighed to the nearest 100 g in their underpants using an electronic scale (SECA-UNICEF). The WC was measured using a flexible tape (Mabbis) with an approach accuracy of 1 cm at the narrowest part of the trunk and it was identified as the midpoint between the lowest rib margin and the iliac crest (19). Weight and height were used to calculate the BMI (weight in kg divided by the square of height in m). Finally, the WHtR was estimated multiplying the WC in cm by 100 and then dividing this figure by the height in cm. BMI and WHtR were treated as continuous variables.

Percentages for sociodemographic factors were reported. Arithmetic means and SD were calculated to describe offspring BMI and WHtR, corresponding to each group of maternal prepregnancy BMI and height. The reported values for the association between maternal anthropometry and offspring body size at 23 y of age are crude and adjusted means [95% CI]. Statistical analyses included the Fisher exact test for categorical variables and the ANOVA for continuous variables. Simple and multiple linear regressions were used for the crude and adjusted analyses. An α value < 0.05 was considered significant.

The effect of main exposures on the continuous outcomes was analyzed by multiple linear regressions, adjusting for confounding and mediating factors. Potential confounding factors (not part of the causal pathway) as well as mediating factors (part of the causal pathway) were evaluated according to a hierarchical approach (20), and those variables whose P-value was ≤ 0.20 were included in the model (21). The model also considered, according to sex, biologically plausible confounding and mediating variables reported in other studies on this topic (2,3,8).

Localized in the distal level of determination within the proposed hierarchical model, the following variables were considered as possible confounders: family income at birth (the monthly income, including all working persons living in the household, in number of minimum wages) in minimum wage; maternal schooling achieved (in years); skin color (defined by the interviewer as white or not white); maternal age (in years); parity (number of previous liveborn deliveries); and maternal smoking during pregnancy.

The following variables (offspring characteristics) were considered as mediators and located at the second level of determination: birth weight, as well as weight-for-age, height-for-age, and weight-for-height Z-scores at 2 and 4 y of age. Except for birth weight, the other anthropometric measures were transformed into Z-scores using the WHO’s international growth reference curves (22). At this level, the female children’s age at menarche and the number of deliveries (parity; both collected between 2004 and 2005) were also included. Finally, the socioeconomic variables [collected between 2004 and 2005: family income in number of minimum wages (categorized as ≤ 3, 3.1–6, 6.1–10, and >10) and the number of schooling years achieved (categorized as 0–4, 5–8, 9–11, and ≥12)] were included in the proximal level.

Additionally, we examined the change in weight between birth and 2 y of age and between 2 and 4 y of age, as well as how these weight gains compared with the weight at the starting age (called “conditional growth”) (23) by using the residual from linear regression.

All analyses were performed using STATA 9.2 (Stata).

Results

This study was restricted to cohort members from lower income families (family income at birth ≤3 times the minimum wages), which represented ≈ 70% of all births in Pelotas in 1982. The comparison between the lower (n = 4077) and higher income (>3 times the minimum wage; n = 1837) groups shows that maternal height and parity differed (P < 0.05) (data not shown). Mothers from low-income families were shorter and had had more deliveries than mothers with a high-socioeconomic status. However, maternal prepregnancy BMI and height were similar in both groups (data not shown).

Of the original 4077 participants from the lower income group formed in 1982 (Fig. 1), 238 individuals died and there were 866 losses to follow-up (n = 1104, not analyzed) (Table 1). At age 23, 2973 participants (1530 men and 1443 women, analyzed group) were interviewed and examined between 2004 and 2005. The group that was not analyzed included more subjects with low birth weight, but there were no differences in relation to maternal prepregnancy BMI or height in comparison with the analyzed group.

The BMI and WHtR at age 23 y in each group of maternal prepregnancy BMI and in each tertile of maternal height are shown (Table 2). Maternal prepregnancy BMI was positively associated with offspring BMI and WHtR for both men and women (P < 0.0001). Offspring BMI was lower among those mothers in the undernutrition category. Female offspring BMI and WHtR differed across the categories of maternal prepregnancy BMI by 5.3 and 6.4 kg/m², respectively (P < 0.0001). On the other hand, maternal height in women was associated only with WHtR but not with BMI. A difference of 0.6 cm in women was detected across the tertiles of maternal height (P = 0.044).
Even after controlling for the confounding and mediating variables, the positive association between maternal prepregnancy BMI and offspring BMI and WHtR at age 23 y remained significant ($P < 0.05$) (Table 3). In male children, for each unit of increase in maternal BMI, BMI and WHtR increased 0.65 and 0.72 kg/m$^2$, respectively. The effect of maternal anthropometry decreased when it was adjusted for mediating factors and it was stronger in men for both indexes related to obesity.

On the other hand, the crude and adjusted analysis of maternal height and offspring obesity indexes showed that maternal height was not significantly associated in either sex (Table 3). For each centimeter of maternal height, male offspring BMI increased 0.20 kg/m$^2$ ($P > 0.05$). In women, a nonsignificant negative effect related to maternal height was observed after adjusting for confounders and mediating factors.

Although birth weight and the attained weight at 2 and 4 y of age are correlated, the findings were similar when adjusting by “conditional growth.” Therefore, only the adjustment by birth weight and Z-scores at 2 and 4 y of age is shown.

Finally, an analysis including the entire cohort members from the different income groups formed in 1982 was conducted and it showed similar findings (data not shown).

**Discussion**

In the low-income population, there was a positive association between the cohort members’ BMI and WHtR and the maternal BMI. At 23 y of age, the indexes related to obesity were not associated with maternal height. The present study was carried out with data from a prospective cohort study to evaluate the association of maternal variables with the occurrence of obesity in adults in a setting developing country. Losses to follow-up were higher in the low-socioeconomic status group. The possibility of survival bias must also be considered for the whole cohort, because the proportion of individuals that was known to have died by the 2004–2005 analysis had to do with low birth weight newborns (16).

Birth weight has been widely reported to be a predictor of chronic diseases (24), and it is usually used as a proxy for fetal growth reflecting the prenatal environment. Studies in low- and middle-income countries are particularly important if we take into account the prevalence and the determinants of early growth failure; moreover, the consequences on body composition in adulthood may differ from those in the developed regions (18).

In the 1982 Pelotas birth cohort, birth weight and weight gain during childhood were associated with either fatness or lean mass later in life (25,26). However, studies on the effect of maternal indicators on offspring body size are scarce.

One of the strengths of this study is that it took into account the confounding and mediating factors during different phases of life. Nevertheless, it lacks data on dietary habits, dietary counseling, and the use of supplements by the mothers during pregnancy, factors that were likely to affect fetal growth.
2 Excluding pregnant women

Because adult height was used to construct offspring anthropometric indicators of obesity at 23 y of age, the family income at birth (in 1982) was used for all the analyses, because a previous study of the same cohort showed that the socioeconomic level at birth is the most important determinant of adult height (27).

Another strength of this study is its prospective approach that reduced the information bias. On the other hand, one possible limitation was the use of the self-reported maternal weight as the independent variable. Although the use of self-referred anthropometric measurements is still being debated, a study of adolescent women in which the self-reported prepregnancy weight was compared with the weight measured before conception resulted in a high correlation coefficient (28). More recently, another study including 150 pregnant women showed an intraclass correlation of 0.95 ($P < 0.05$). However, weight may be underestimated, particularly in overweight women, whereas this tendency does not affect undernourished women (29). Because any error in the maternal report of prepregnancy weight is independent from offspring BMI and WHtR, the associations were not due to an information bias.

This study showed that offspring BMI and WHtR were influenced by the maternal prepregnancy BMI, even after adjusting for confounding and mediating variables. A direct association between the maternal prepregnancy BMI and both outcomes was observed in women and men at age 23 y. These results were similar to those in the Finland birth cohort, where the prepregnancy weight predicted offspring BMI and WC at 31 y of age (30). The effect of maternal prepregnancy BMI, as well as that of birth weight, on obesity at age 33 y was studied in the 1958 British cohort and the weak positive association between birth weight and adult obesity was similar across the tertiles of maternal prepregnancy BMI (31).

Consistent with studies from high-income countries, the direct association between maternal anthropometry and offspring body size has been observed in a low-income population (3,30,31). However, these studies included older individuals, whereas our participants were young adults. On the other hand, if we consider that maternal undernutrition is more frequent in our population and that the effect of the programming hypotheses (24) will be present within a few years, it is necessary to investigate these effects on our cohort at an older age.

The effect of maternal prepregnancy BMI on offspring measurements suggests that the genetic, physiological, and metabolic factors could be influencing offspring BMI, along with environmental and behavioral factors, during the life course (32).

### TABLE 2

<table>
<thead>
<tr>
<th>Maternal BMI, kg/m²</th>
<th>Men</th>
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<th></th>
<th>Women</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>BMI, kg/m²</td>
<td>WHtR</td>
<td>n</td>
<td>BMI, kg/m²</td>
<td>WHtR</td>
<td></td>
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<tr>
<td>≤18.5</td>
<td>110</td>
<td>22.2 ± 3.5 (21.3)</td>
<td>21.7 ± 3.8 (21.2)</td>
<td>99</td>
<td>44.7 ± 4.7 (43.6)</td>
<td>44.7 ± 4.5 (43.8)</td>
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<tr>
<td>18.6–24.9</td>
<td>887</td>
<td>23.2 ± 3.7 (22.6)</td>
<td>23 ± 4.2 (22.0)</td>
<td>744</td>
<td>45.9 ± 5.4 (45.1)</td>
<td>46.9 ± 6.6 (45.2)</td>
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<tr>
<td>25–29.9</td>
<td>210</td>
<td>24.8 ± 4.4 (24.2)</td>
<td>24.6 ± 4.7 (23.2)</td>
<td>210</td>
<td>47.6 ± 6.1 (46.6)</td>
<td>48.2 ± 6.8 (46.9)</td>
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<tr>
<td>≥30</td>
<td>58</td>
<td>25.5 ± 4.3 (24.8)</td>
<td>27 ± 6 (25.6)</td>
<td>59</td>
<td>48.7 ± 5.9 (48.5)</td>
<td>51.1 ± 7.9 (48.7)</td>
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P-value

- 0.001 0.001 0.001 0.001

Maternal height, cm

| Lower tertile | 633 | 23.2 ± 3.7 (22.6) | 23.7 ± 4.9 (22.7) |
| Middle tertile | 461 | 23.5 ± 3.7 (22.8) | 23.4 ± 5.0 (23.3) |
| Higher tertile | 418 | 24.0 ± 4.4 (23.0) | 24.0 ± 5.0 (22.5) |

P-value

- 0.001 0.044

1 Values are means ± SD (median).

2 Adjusted for confounders: maternal age, maternal smoking, maternal parity, maternal education, family income, and skin color.

3 Also adjusted for early or current mediators: birth weight, weight-for-age, height-for-age, and weight-for-height Z-scores at 2 and 4 y; age at menarche and parity women; current family income and education achieved.

### TABLE 3

<table>
<thead>
<tr>
<th>Maternal BMI, kg/m²</th>
<th>Men</th>
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<tr>
<td></td>
<td>n</td>
<td>BMI, β (95% CI)</td>
<td>WHtR, β (95% CI)</td>
<td>n</td>
<td>BMI, β (95% CI)</td>
<td>WHtR, β (95% CI)</td>
<td></td>
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<tr>
<td>Maternal BMI, kg/m²</td>
<td></td>
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<tr>
<td>Crude</td>
<td>0.70 (0.47–0.94)</td>
<td>0.94 (0.66–1.21)</td>
<td>0.76 (0.43–1.09)</td>
<td>0.86 (0.45–1.27)</td>
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<tr>
<td>Adjusted²</td>
<td>0.77 (0.53–1.01)</td>
<td>0.94 (0.66–1.22)</td>
<td>0.81 (0.47–1.14)</td>
<td>0.85 (0.42–1.27)</td>
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<tr>
<td>Adjusted³</td>
<td>0.65 (0.41–0.88)</td>
<td>0.63 (0.36–0.90)</td>
<td>0.72 (0.38–1.07)</td>
<td>0.58 (0.16–1.00)</td>
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<tr>
<td>Adjusted $R^2$</td>
<td>0.24</td>
<td>0.25</td>
<td>0.16</td>
<td>0.18</td>
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</table>

Maternal height, cm

| Crude               | 0.25 (–0.009–0.51) | 0.23 (–0.11–0.58) | 0.14 (–0.22–0.51) | 0.26 (–0.24–0.77) |
| Adjusted¹           | 0.22 (–0.03–0.48) | 0.24 (–0.10–0.59) | 0.11 (–0.25–0.48) | 0.27 (–0.23–0.78) |
| Adjusted²           | 0.20 (–0.05–0.47) | 0.01 (–0.35–0.33) | 0.17 (–0.21–0.56) | 0.07 (–0.43–0.58) |
| Adjusted $R^2$      | 0.21 | 0.23 | 0.13 | 0.19 |
A dose-response relationship between the maternal prepregnancy BMI and the offspring BMI was also observed in both sexes. Thus, maternal obesity may interact with early-life programming in the establishment of diseases on the offspring, suggesting that gene-environment interactions should be considered.

Maternal height was not associated with offspring BMI. These findings were similar to those in the British cohort (31) and to those in a recent study from low- and middle-income countries (7).

The present study did not find an effect of maternal height on offspring WHR. There are few data considering maternal height as a predictor of offspring body fat distribution in either low- or high-income countries. Other studies in older populations have shown that the mother’s height was not associated with cardiovascular or stroke risk in adults, whereas in developing countries, the highest cardiovascular risk was observed in children with shorter parents (31–33).

In conclusion, the present study suggests that maternal anthropometry is associated with offspring BMI and WHR at 23 y of age. Therefore, the findings indicate an intergenerational effect between maternal nutrition and offspring BMI and WHR and, hence, they corroborate the importance of the parental predictors in relation to their offspring’s adult health (7).

**Literature Cited**