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Title

Maternal short stature does not predict their children's adiposity indicators in a nutritional dual-burden sample of urban Mexican Maya

Abstract

Objective: The co-existence of very short stature due to poor chronic environment in early life and obesity is becoming a public health concern in rapidly transitioning populations with high levels of poverty. Individuals who have very short stature seem to be at an increased risk of obesity in times of relative caloric abundance. Increasing evidence shows that an individual is influenced by exposures in previous generations. This study assesses whether maternal poor early life environment predicts her child's adiposity.

Design: A cross sectional study comparing maternal chronic early life environment (stature) with her child's adiposity (body mass index (BMI) z-score, waist circumference z-score and percentage body fat) using multiple linear regression, controlling for the child's own environmental exposures (household sanitation and maternal parity).

Setting: The south of Merida, Yucatan, Mexico, a low socioeconomic urban area in an upper middle income country.

Subjects: Maya schoolchildren aged 7-9 and their mothers (n=57 pairs).

Results: The Maya mothers were very short, with a mean stature of 147 cm. The children had fairly high adiposity levels, with BMI and waist circumference z-scores above the reference median. Maternal stature did not significantly predict any child adiposity indicator.

Conclusions: There does not appear to be an intergenerational component of maternal early life chronic under-nutrition on her child's obesity risk within this free living population living in poverty. These results suggest that the co-existence of very short stature and obesity appears to be primarily due to exposures and experiences within a generation rather than across generations.

1 Introduction

2 The burden of disease in developing countries has been shifting in the last decades from a profile
3 dominated by diseases of under-nutrition and infection to nutrition-related, non-communicable
4 diseases such as cardiovascular disease. Some populations undergo this transition within a
5 generation and many do not eliminate the issues related to poverty⁽¹⁾.

6 Within this context, a great percentage of individuals who experienced chronically poor nutrition
7 in their early lives, and are thus stunted, seem to be more predisposed to obesity later in life⁽²⁻¹⁰⁾.
8 Individuals with chronic under-nutrition (stunting) in early life seem to have reduced energetic
9 demands through metabolic^(6, 9, 11-13) and behavioural shifts⁽¹⁴⁻¹⁶⁾. Such reduced energetic
10 demands may increase an individual's odds of immediate survival under conditions of energetic
11 stress yet have negative longer-term consequences in health and survival. This seems to be the
12 case in situations in which rapid nutrition transition is coupled with high rates of poverty where
13 calories rapidly become more abundant and lifestyles become more mechanized. Stunted
14 individuals predisposed to energy conservation may be at an increased risk of obesity when
15 calories are relatively abundant and energy expenditure requirements are low.

16 The implications of the energy conservation effects of chronic early life under-nutrition may
17 extend into future generations. It is theoretically possible for mothers to transfer energetic
18 strategies to their children⁽¹⁷⁻²¹⁾. In theory, a child born of a stunted mother is more likely to be
19 predisposed to energy conservation than a child whose mother was not stunted. This
20 predisposition may increase the child's risk of excess adiposity. Such intergenerational effects
21 are a consequence of ancestral phenotypes, results of past socioeconomic, cultural, and political
22 conditions, biologically expressed as epigenetic modifications to DNA, but are not mediated by
23 inherited DNA sequences⁽²²⁾. The majority of research into intergenerational influences has
24 focused on exposure during gestation, mainly conducted in rats⁽²²⁻²⁴⁾. A classic example of the
25 intergenerational effects in humans can be seen in the studies reporting on the Dutch Winter
26 Famine⁽²⁶⁾. In these studies, the authors examined the effects of six months of severe wartime
27 rationing (November 1944-May 1945) in the Netherlands. Individuals exposed to the rationing *in*
28 *utero* had children with higher ponderal indices at birth than those unexposed or exposed post-
29 natally. These results suggest that calorie restriction during gestation causes an intergenerational
30 predisposition to the activation of energy conservation mechanisms.

1 However the majority of under-nutrition in the world today is not due to a single acute event,
2 such as severe wartime rationing, but is chronic and linked to persistent poverty and food
3 insecurity⁽²⁷⁾. It is unknown at this point whether chronic under-nutrition, particularly post-
4 natally, in one generation increases the risk for obesity in future generations. Persistent negative
5 conditions have been found to influence the risk of non-communicable, chronic diseases, such as
6 cardiovascular diseases. For example, the history of slavery and systematic marginalization has
7 been linked to the disproportionate cardiovascular disease burden of African Americans in the
8 United States⁽²⁸⁾. African Americans also have the highest rates of adult obesity in the United
9 States⁽²⁹⁾. Since obesity is a precursor to cardiovascular disease and other non-communicable
10 diseases^(30, 31), it is may be possible for obesity to be affected by an intergenerational legacy as
11 well.

12 Household dual burden involving a stunted mother and an overweight or obese child is a
13 phenomenon linked to rapid nutrition transition and poverty. This household dual burden
14 phenomenon offers an opportunity to investigate intergenerational influences of chronic
15 nutritional stress on obesity risk in humans. The mothers who are stunted experienced early life
16 chronic under-nutrition, primarily before the age of 5⁽³²⁾. Since stunting is so closely linked to
17 poverty and food insecurity, it is likely that the chronic under-nutrition that led to shorter stature
18 in these women but was persistent within their families before and after they were conceived.
19 This is a particularly strong assumption if a sample is drawn from a population with a long
20 history of systematic marginalization and under-nutrition, such as many indigenous Americans
21 groups including the Maya⁽³³⁾. Within this context, we can assume that adult women who are
22 stunted were exposed to under-nutrition *in utero* as well as post-natally, which would program
23 their own metabolism for energy conservation as well as their gametocytes. Therefore, it is
24 possible that stunted mothers pass along a predisposition for energy conservation to their
25 children^(3, 20, 34-36). Such children would be predisposed to have high levels of adiposity and be at
26 increased risk of obesity.

27
28 The aim of this study is to determine whether maternal chronic under-nutrition in early life, as
29 measured by mother's adult stature, has an intergenerational influence on the adiposity of their 7-
30 9 year old Maya children. Within this age range growth is fairly stable⁽³⁷⁾ and adiposity is

predictive of later life fatness, health risks⁽³⁸⁾. We aim to determine whether Maya mothers who experienced chronically poor early life conditions (and are therefore very short) convey an increased risk for greater fatness to their children. The null hypotheses being tested are that maternal stature will not significantly predict: 1) child's body mass index (BMI-for-age z-score); 2) child's percentage of body fat (%BF); and 3) child's waist circumference (WC-for-age z-scores) after controlling for the children's own environmental exposures.

Experimental Methods

Cross-sectional data were collected, between March and July 2010, on 58 Maya mothers and their children (aged 7.00 to 9.99 years), living in the south of Merida, Yucatan, Mexico.

Study population and location

The Maya are the largest indigenous group in the Americas, with a population of 7-8 million⁽³⁹⁾. Similar to other Amerindian populations, the Maya experienced systematic marginalization for centuries. Within Mexico, the official marginalization policies have ended but the Maya people remain one of the poorest ethnic groups in the country⁽⁴⁰⁾ and have high rates of stunting^(41, 42). Between 1500 and the late 20th century A.D., the Maya economy in Yucatan, Mexico was based upon subsistence agriculture, particularly of maize. The Maya also worked as near slaves in the regional agroindustries. Since the late 1970's and 1980's, Yucatecan Maya have migrated to urban and coastal areas in search of employment in construction and tourism related industries, or other low-skilled work. The diets of the Maya in the Yucatan are increasingly dominated by calorie-dense, nutrient-poor foods^(43, 44). The Maya in Merida are a dual burdened population, with high levels of stunting and overweight and obesity (OW/OB)^(41,42).

This study focuses on an urban population of Maya living in the south of Merida, Yucatan, Mexico. Mexico is undergoing nutrition transition^(45, 46). Merida is a rapidly growing city, home to approximately 75,000 Maya speakers in 2010, roughly 9% of the city's population⁽⁴⁷⁾. Merida is highly segregated from north to -south in terms of wealth, infrastructure and services^(48, 49). The south of the city has few services and is physically separated from the rest of the city by an airport runway that makes leaving the south time consuming. While the south is a low income

1 area, Mexico has implemented many public health measures in the past 30 years, including
2 vaccinations and clean drinking water campaigns^(50,51). Thus the south of Merida is a low
3 socioeconomic status area within an upper middle income country⁽⁵²⁾ that is meeting some of the
4 basic requirements for child welfare⁽⁵³⁾.

5 *Recruitment and ethics*

6 This study was conducted according to the guidelines laid down in the Declaration of Helsinki
7 and all procedures involving human subjects were approved by the [name of the ethics
8 committee removed for blinding]. Written informed consent was obtained from the mothers or
9 head of the household and verbal assent from the children. Prior to parental consent given for
10 study participation, the researchers had no contact with the children.

11 Recruitment took place in schools in the south of Merida, where a high proportion of Maya
12 reside. Mexico has official school enrolment rates of almost 100%⁽⁵¹⁾ and therefore schools were
13 targeted for recruitment. Children with two Maya surnames (one from the father and mother)
14 between the ages of 7 and 9 years old were identified using school records. School directors
15 invited the mothers of identified children to group information sessions held at the school using
16 flyers. The study nature and requirements were verbally explained to the parents and information
17 sheets were given out during the information sessions. Spanish was used for all stages of
18 participant interaction because it is the dominant language of the south of Merida and all mothers
19 were comfortable speaking it.

21 Data collection

22 *Anthropometry*

23 Stature, weight, waist circumference (WC) and skinfolds (triceps and subscapular) were
24 measured using standard techniques⁽⁵⁴⁾ on children and mothers.

Bioelectrical Impedance analysis

To assess body composition, bioelectrical impedance analysis (BIA) was measured (BioScan 916, Maltron, UK) on children and mothers and used to calculate percentage of body fat.

Interview

To assess family socioeconomic status, demographics and child's health, the researchers created an interview based upon the most recent Mexican Demographic Health Survey (1987)⁽⁵⁵⁾ and piloted it in the south of Merida. Mothers were interviewed in the family home or school by native Yucatec-Spanish speaking women. Mothers were asked their own and their child's dates of birth. Parity was assessed by asking the mothers how many times they had given birth and how many children they had. These two were perfectly correlated and used as the indicator of maternal parity. Sanitation was assessed by asking whether the family owned a flush toilet, the source of drinking water and method of cleaning drinking water. To assess socioeconomic status, mothers were asked if the family owned the following consumer durable goods: radio, telephone, refrigerator, car, tricycle or bicycle and a television.

Child's birth weight was assessed using maternal recall. When possible, the child's birth certificate was obtained but only four mothers had it available. Within these cases, three of the mothers recalled the birth weights higher than the weight recorded on the birth certificate, indicating that there may be a systematic bias in this variable. For this reason, child's birth weight was not included in the main statistical analysis.

Data analysis

Anthropometry

Body mass index (BMI) was calculated for mothers and children by dividing an individual's weight in kilograms by the square of their stature in meters. Children's age- and sex- specific z-scores were calculated for height, weight, BMI and WC using the Comprehensive reference charts⁽⁵⁶⁾. The Comprehensive reference was created using data from a U.S. national, stratified

sample that over-sampled minorities (NHANES III). The sample contains 27.0% Hispanic and Latinos (mostly Mexicans and Cubans). The Comprehensive reference is the largest sampled, statistically validated growth reference that includes Mexican children.

Maternal stunting was defined as 150 cm^(41, 42, 57). Children were classified as OW/OB using the International Obesity Task Force cut-offs for BMI⁽⁵⁸⁾, the 85th percentile of the Comprehensive reference for WC, as well as the 85th percentile for %BF⁽⁵⁹⁾. Previous analyses have found WC z-scores to be highly related to BMI z-scores within these Maya children⁽⁶⁰⁾.

Bioelectrical impedance analysis

To calculate percentage body fat, indigenous North American specific equations were used for children (Equation 1)⁽⁶¹⁾ and mothers (Equation 2)⁽⁶²⁾. These equations were chosen because no validated body composition equations have been created for Mesoamerican indigenous groups such as the Maya.

Equation 1: Child's percentage body fat = $-0.49 (\text{age}) + 0.51 (\text{sex}) + 0.44 (\text{weight}) + 1.55 (\text{triceps skinfold}) + 0.15 (\text{subscapular skinfold}) + 0.54 (\text{stature}^2 / \text{resistance}) + 0.13 (\text{reactance}) - 0.04 (\text{triceps skinfold} \times \text{stature}^2 / \text{resistance}) - 10.91$

Where sex is coded 0 for boys, 1 for girls; age is in years, weight is in kilograms; skinfold thickness is in millimeters, resistance and reactance is in Ohms and stature is in meters.

Equation 2: Women's fat free mass = $0.001254 (\text{stature}^2) - 0.04904 (\text{reactance}) + 0.1555 (\text{weight}) + 0.1417 (\text{impedance}) - 0.0833 (\text{age}) + 20.05$

Where stature is in centimeters, weight is in kilograms and reactance and impedance are in Ohms.

Fat free mass was transformed into %BF by subtracting fat free mass from weight and dividing the resulting number by weight and multiplying by 100. $[(\text{weight} - \text{FFM}) / \text{weight}] \times 100$.

1

2 *Socioeconomic status and sanitation indices creation*

3 Socioeconomic status and sanitation variables were used to construct two indices. The
4 socioeconomic status index used information on consumer durable goods ownership while the
5 sanitation index used drinking water and toilet facilities. The indices were created by summing
6 affirmative responses to the questions. Each affirmative response (e.g. owning a radio or having
7 a flush toilet) was coded as 1 while negative responses were coded as 0. Families that drank
8 either purchased purified water or cleaned the water were classified as having clean drinking
9 water.

10

11 Statistical analysis

12 All variables were checked for normality using the Kolmogorov-Smirnov test and skewness and
13 kurtosis z-statistics. Independent *t*-tests were used to check for differences in anthropometry and
14 body composition between the sexes. Pearson chi squares were used to determine if significant
15 differences existed in ownership of any consumer durable good and sanitation measure between
16 households with a stunted and non-stunted mother and with a normal weight and OW/OB child.

17 Stepwise multiple linear regressions using the enter method were used to test the hypothesis that
18 child adiposity is predicted by their mother's chronic early life conditions. The dependent
19 variables were child's adiposity indicators, namely, BMI z-score, %BF and WC z-scores. BMI z-
20 score is widely used as a proxy for total body adiposity but is a very crude measurement.
21 Therefore we also used %BF as a measure for total body adiposity. WC z-score is used as an
22 indicator of abdominal adiposity, an independent measure of risk for non-communicable, chronic
23 diseases.

24 The predictors were entered in two steps to determine whether each type of indicator mediated or
25 attenuated the relationship of maternal stature on child's adiposity indicators. The steps were: 1)
26 maternal stature, 2) child's environmental exposures (sanitation index, maternal parity).
27 Maternal stature was used as an indicator of maternal early life environment. The co-variates in
28 the multiple linear regression models were selected to control for the children's own

environmental exposures. Ideally, children's stature z-scores would be used as an indicator of their chronic nutritional environment, but there was significant correlation between child and maternal height ($R=0.440$, $p=0.001$) violating the statistical assumption of independence of predictors. Therefore indicators of the child's chronic environment (household sanitation index and maternal parity) were included as predictors. Maternal parity was not linearly related to indicators of child's adiposity and therefore parity was split into tertiles and entered into the model using dummy variables with the tertile associated with the lowest adiposity indicators used as the reference. The commonly used consumer durable goods ownership index was collinear with both sanitation index and maternal parity, and not a significant predictor of child's adiposity, therefore was not included in the multiple regression analyses.

The sexes are combined in the multiple regression analysis because no significant differences were found between them in preliminary analysis. For the analysis with %BF as the dependent variable, age and sex were included in every model because reference charts were not available for this age group.

The total sample size available for this analysis was 57 mother-child pairs. The power for a multiple linear regression with four predictors with an alpha of 0.8 and a p value of 0.05 has the power to detect an effect size of 0.23, a medium effect⁽⁶³⁾.

All analyses were performed using IBM SPSS 19.0. Significance was set *a priori* at $p<0.05$.

Results

One mother-child dyad was excluded because the child was developmentally delayed, likely due to being born extremely premature (at 28 weeks), weighing one kilogram at birth. The final sample size was 57 dyads (30 boys).

These Maya children were short (more than one standard deviation below the reference median) and light but had adiposity indicators whose means were above the reference median (Table 1). No sex differences in the children's anthropometry were found. Mothers were short (mean stature 147 cm) with 44 (77.2%) being classified as stunted. Mothers were also heavy, with high mean BMI value (30) and high adiposity as indicated by their WC (88 cm) and % BF (42%).

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Table 1 about here

Socio-demographic characteristics are as follows: In terms of sanitation, over three quarters of families drank purified, cleaned water while under two thirds had a flush toilet in the house, bringing the mean additive index score to 1.39 (SD=0.73). Maternal parity was on average 3.25 children (SD=1.38), with a range of 1-8. The majority of the mothers had two (22%) or three (48%) children. The mean child birth weight reported by the mothers was 3.08 kilograms (SD=0.54).

In the multiple linear regression analyses, maternal height was neither a significant predictor of nor explained any variance in any measure of child’s adiposity (Tables 2-4). Also, the household sanitation index was not a significant predictor of any measure of child’s adiposity. Maternal parity was a significant predictor of %BF (Table 4) but not of child’s BMI z-scores (Table 2) or WC z-score (Table 3). Children with fewer siblings had significantly higher %BF than those with more siblings with the significant differences occurring between the children whose mothers had 1-2 children versus 3-8 children, explaining 5.6% of the variance in child’s %BF.

Table 2 about here
Table 3 about here
Table 4 about here

Discussion

This study finds no support, within this sample of urban Maya, for the hypothesis that maternal stature is a significant predictor of her child’s fatness, whether fatness is assessed either

1 by BMI z-scores, %BF, or WC z-scores. There are several possible reasons for this. It is possible
2 that there is a predictive relationship but our sample size is too small for sufficient statistical
3 power. Modest sample size is a limitation, but our power analysis indicates we should be able to
4 detect a moderate effect. It is possible that children aged 7-9 years are too young to show the
5 intergenerational effect of mother's height on child fatness. We consider this unlikely because
6 by age 7 years patterns of growth are usually well established⁽³⁷⁾ and relative fatness at age 7-9
7 years has a reasonably high predictive association with fatness later in life^(64, 65). Another
8 possibility is that there may not be enough variation in maternal height to find a significant
9 relationship with child fatness. Any consideration of Maya growth and nutritional status must
10 begin with an understanding that Maya adults are the shortest people in the contemporary
11 world⁽³⁹⁾. This statement excludes the Central African Pygmies and Philippine Pygmies, whose
12 short average stature is due to some combination of genome mutations, hormonal insufficiencies,
13 missing carrier proteins for hormones, and other neuroendocrine pathology. None of these causes
14 for short stature are known to afflict the Maya. Not only is their average stature very short, but
15 the entire range of height of the adult Maya population is shifted downward and restricted to a
16 relatively narrow range (Bogin, unpublished analysis). The range restriction may reduce height
17 variability to the point that it violates the normality requirements for statistical analysis.

18 In terms of public health measures to decrease childhood obesity rates, it is encouraging
19 that there does not seem to be a significant intergenerational effect of maternal stature on
20 childhood obesity. If true, reducing childhood obesity rates may not lag a generation and can be
21 relatively quickly reversed once an effective intervention is developed and implemented for
22 transitioning populations. Public health interventions are more likely to be funded and supported,
23 particularly by governments, if the benefits are seen quickly. Since the challenges of addressing
24 malnutrition (both under- and over-nutrition) are substantial, a lack of a prolonged
25 intergenerational influence is advantageous for public health programs.

26 However, there is evidence in this study that socioeconomic variables predict high levels
27 of fatness among the children. The Maya of Mexico and Central America have been, historically,
28 very short due to the synergism of nutritional deficiencies, infectious diseases (including
29 parasites) and a legacy of political oppression, war and social instability. The rapidity of the
30 nutrition transition in Mexico has created different environments for growth for the Maya
31 mothers and their children. The Maya mothers in our sample are characterized as nutritional

1 dual-burdened, being both stunted and overweight⁽⁴²⁾. Before the 21st Century, Maya in Mexico
2 and Guatemala were characterized as stunted and thin^(44, 64) and this was the nutritional situation
3 for the mothers when they were children themselves. Recently, the nutrition transition brought
4 about a steep increase in the consumption of high-fat, high caloric foods and snacks^(43, 44, 66, 67).

5 Food frequency surveys in Yucatan carried out since the year 2000 found that tortillas
6 and beans remain staples of the diet, but are now purchased as industrially prepared products⁽⁶⁸⁾.
7 Store bought tortillas may have a lower content of vitamins and minerals, such as calcium. A
8 study of food consumption patterns in two rural Yucatec Maya villages found that 65% of
9 households were medium consumers of industrialized foods (up to 33% of all foods consumed)
10 and 11% of families were heavy consumers (up to 53% of all foods eaten were industrialized)⁽⁶⁸⁾.
11 Our ethnographic observations found that the urban Maya of Merida do not practice subsistence
12 farming and rarely have any vegetable garden. Several families had one or two fruit trees on their
13 property (mostly lime trees). The ubiquity and very high intake of sugary drinks led one research
14 team to proclaim that the Yucatan of Mexico has been 'coca-colonized'⁽⁴³⁾.

15 In the multiple regression analysis of this study, maternal parity significantly and
16 positively predicted child's adiposity, so that children from families with four or more children
17 had higher adiposity than those from smaller families. Household size has been linked to
18 household dual burden in other transitioning populations⁽⁶⁹⁾. Within the context of stunting it
19 seems counter-intuitive that larger families have children with more adiposity. Yet this is a
20 transitioning, dual burdened sample and as such, traditional relationships between poverty and
21 malnutrition do not necessarily hold. These Maya families may have transitioned to the stage
22 where their relationship with resource access/ distribution and adiposity is more similar to
23 communities of low SES in the higher income countries. Within higher income countries, larger
24 families of lower SES have been found to have increased obesity⁽⁷⁰⁾. Some have linked the stress
25 associated with food insecurity and poverty to resource distribution and parental behaviours
26 which may lead to child overweight^(71, 72). In such contexts, families with more children may
27 prioritize cheap food that has high caloric density but is also highly processed and thereby
28 offering little in terms of other nutrients^(70, 72, 73).

29 The results from this analysis suggest that high levels of child adiposity may be reversed
30 without a generational lag provided that effective obesity prevention measures can be developed
31 and implemented in a timely manner. To be effective, such measures require political will and

1 must target the underlying issues, such as poverty, in order to improve the nutritional status and
2 health of the population.

4 **References**

- 6 1. Popkin BM (2002) The shift in stages of the nutrition transition in the developing world
7 differs from past experiences! *Public Health Nutr* **5**, 205-214.
- 8 2. Alves JG, Falcao RW, Pinto RA *et al.* (2011) Obesity patterns among women in a slum area
9 in Brazil. *J Health Popul Nutr* **29**, 286-289.
- 10 3. Ferreira HS, Moura FA, Cabral CR, Jr. *et al.* (2009) Short stature of mothers from an area
11 endemic for undernutrition is associated with obesity, hypertension and stunted children: a
12 population-based study in the semi-arid region of Alagoas, Northeast Brazil. *Br J Nutr* **101**,
13 1239-1245.
- 14 4. Florencio TT, Ferreira HS, Cavalcante JC *et al.* (2003) Food consumed does not account for
15 the higher prevalence of obesity among stunted adults in a very-low-income population in the
16 Northeast of Brazil (Maceio, Alagoas). *Eur J Clin Nutr* **57**, 1437-1446.
- 17 5. Florencio TT, Ferreira HS, Cavalcante JC *et al.* (2007) Short stature, abdominal obesity,
18 insulin resistance and alterations in lipid profile in very low-income women living in Maceio,
19 north eastern Brazil. *Eur J Cardiovasc Prev R* **14**, 346-348.
- 20 6. Grillo LP, Siqueira AF, Silva AC *et al.* (2005) Lower resting metabolic rate and higher
21 velocity of weight gain in a prospective study of stunted vs nonstunted girls living in the
22 shantytown of Sao Paulo, Brazil. *Eur J Clin Nutr* **59**, 835-842.
- 23 7. Martins PA, Hoffman DJ, Fernandes MT *et al.* (2004) Stunted children gain less lean body
24 mass and more fat mass than their non-stunted counterparts: a prospective study. *Br J Nutr*
25 **92**, 819-825.
- 26 8. Romaguera D, Samman N, Farfan N *et al.* (2008) Nutritional status of the Andean population
27 of Puna and Quebrada of Humahuaca, Jujuy, Argentina. *Pub Health Nutr* **11**, 606-615.
- 28 9. Said-Mohamed R, Alliot X, Sobgui M *et al.* (2009) Determinant of overweight associated
29 with stunting in preschool children of Yaounde, Cameroon. *Ann Hum Biol* **36**, 146-161.
- 30 10. Sichieri R, Dos Santos Barbosa F, Moura EC (2010) Relationship between short stature and
31 obesity in Brazil: a multilevel analysis. *Br J Nutr* **103**, 1534-1538.
- 32 11. Frisancho AR (2003) Reduced rate of fat oxidation: a metabolic pathway to obesity in the
33 developing nations. *Am J Hum Biol* **15**, 522-532.
- 34 12. Said-Mohamed R, Bernard JY, Ndzana AC *et al.* (2012) Is overweight in stunted preschool
35 children in Cameroon related to reductions in fat oxidation, resting energy expenditure and
36 physical activity? *Plos One* **7**, e39007.
- 37 13. Soares-Wynter SY, Walker SP (1996) Resting metabolic rate and body composition in
38 stunted and nonstunted children. *Am J Clin Nutr* **64**, 137-141.
- 39 14. Hoffman DJ, Roberts SB, Verreschi I *et al.* (2000) Regulation of energy intake may be
40 impaired in nutritionally stunted children from the shantytowns of Sao Paulo, Brazil. *J Nutr*
41 **130**, 2265-2270.
- 42 15. Spurr GB, Reina JC (1988) Patterns of daily energy expenditure in normal and marginally
43 undernourished school-aged Colombian children. *Eur J Clin Nutr* **42**, 819-834.

- 1 16. Wilson HJ, Dickinson F, Hoffman DJ *et al.* (2012) Fat free mass explains the relationship
2 between stunting and energy expenditure in urban Mexican Maya children. *Ann Hum Biol*
3 **39**, 432-439.
- 4 17. Emanuel I (1986) Maternal health during childhood and later reproductive performance. *Ann*
5 *N Y Acad Sci* **477**, 27-39.
- 6 18. Drake AJ, Walker BR (2004) The intergenerational effects of fetal programming: non-
7 genomic mechanisms for the inheritance of low birth weight and cardiovascular risk. *J*
8 *Endocrinol* **180**, 1-16.
- 9 19. Gluckman PD, Hanson MA (2004) The developmental origins of the metabolic syndrome.
10 *Trends Endocrinol Metab* **15**, 183-187.
- 11 20. Kuzawa CW (2005) Fetal origins of developmental plasticity: are fetal cues reliable
12 predictors of future nutritional environments? *Am J Hum Biol* **17**, 5-21.
- 13 21. Wells JC (2010) Maternal capital and the metabolic ghetto: An evolutionary perspective on
14 the transgenerational basis of health inequalities. *Am J Hum Biol* **22**, 1-17.
- 15 22. Patti ME (2013) Intergenerational programming of metabolic disease: evidence from human
16 populations and experimental animal models. *Cell Mol Life Sci* **70**, 1597-1608.
- 17 23. Benyshek DC, Johnston CS, Martin JF (2006) Glucose metabolism is altered in the
18 adequately-nourished grand-offspring (F3 generation) of rats malnourished during gestation
19 and perinatal life. *Diabetologia* **49**, 1117-1119.
- 20 24. Bertram C, Khan O, Ohri S *et al.* (2008) Transgenerational effects of prenatal nutrient
21 restriction on cardiovascular and hypothalamic-pituitary-adrenal function. *J Physiol* **586**,
22 2217-2229.
- 23 25. Jimenez-Chillaron JC, Isganaitis E, Charalambous M *et al.* (2009) Intergenerational
24 transmission of glucose intolerance and obesity by *in utero* undernutrition in mice. *Diabetes*
25 **58**, 460-468.
- 26 26. Painter RC, Osmond C, Gluckman P *et al.* (2008) Transgenerational effects of prenatal
27 exposure to the Dutch famine on neonatal adiposity and health in later life. *BJOG* **115**, 1243-
28 1249.
- 29 27. WHO (2005) *Health and the Millennium Development Goals*. Geneva, Switzerland: World
30 Health Organization.
- 31 28. Kuzawa CW, Sweet E (2009) Epigenetics and the embodiment of race: developmental
32 origins of US racial disparities in cardiovascular health. *Am J Hum Biol* **21**, 2-15.
- 33 29. Flegal KM, Carroll MD, Kit BK *et al.* (2012) Prevalence of obesity and trends in the
34 distribution of body mass index among US adults, 1999-2010. *JAMA* **307**, 491-497.
- 35 30. James WP, Rigby N, Leach R (2006) Obesity and the metabolic syndrome: the stress on
36 society. *Ann N Y Acad Sci* **1083**, 1-10.
- 37 31. Ogden CL, Yanovski SZ, Carroll MD *et al.* (2007) The epidemiology of obesity.
38 *Gastroenterology* **132**, 2087-2102.
- 39 32. Stein AD, Wang M, Martorell R *et al.* (2007) Growth patterns in early childhood and final
40 attained stature: data from five birth cohorts from low- and middle-income countries. *Am J*
41 *Hum Biol* **22**, 353-359.
- 42 33. Bogin B, Keep R (1999) Eight thousand years of economic and political history in Latin
43 America revealed by anthropometry. *Ann Hum Biol* **26**, 333-351.
- 44 34. Drake AJ, Liu L (2010) Intergenerational transmission of programmed effects: public health
45 consequences. *Trends Endocrin Met* **21**, 206-213.

35. Pembrey ME, Bygren LO, Kaati G *et al.* (2006) Sex-specific, male-line transgenerational responses in humans. *Eur J Hum Genet* **14**, 159-166.
36. Skinner MK, Manikkam M, Guerrero-Bosagana C (2010) Epigenetic transgenerational actions of environmental factors in disease etiology. *Trends Endocrin Met* **21**, 214-222.
37. Cameron N (2002) Human growth curve, canalization, and catch-up growth. In *Human growth and development*, pp. 1-20 [N Cameron, editor]. Amsterdam: Academic Publishers.
38. Cameron N, Demerath EW (2002) Critical periods in human growth and their relationship to diseases of aging. *Am J Phys Anthropol Supp* **35**, 159-184.
39. Bogin B (2012) The Maya in Disneyland: Child Growth as a Marker of Nutritional, Economic, and Political Ecology. In *Nutritional Anthropology: Biocultural Perspectives on Food and Nutrition*, 2nd ed., pp. 231-244 [DL Dufour, A Goodman and GH Peltó, editors]. Oxford: Oxford University Press.
40. Bracamonte P (2007) *Una deuda historica. Ensayo sobre las causas de pobreza secular d los mayos yucatecos*. Mexico, D. F.: CIESAS-Porrúa-ICY.
41. Azcorra H, Dickinson F, Rothenberg SJ (2009) Family migration and physical growth in Merida, Yucatan, Mexico. *Am J Hum Biol* **21**, 398-400.
42. Varela-Silva MI, Dickinson F, Wilson H *et al.* (2012) The nutritional dual-burden in developing countries- how is it assessed and what are the health implications? *Collegium Antropol* **36**, 39-45.
43. Leatherman TL, Goodman A (2005) Coca-colonization of diets in the Yucatan. *Soc Sci Med* **61**, 833-846.
44. Leatherman, Goodman AH, Stillman T (2010) Changes in stature, weight, and nutritional status with tourism based economic development in the Yucatan. *Econ Hum Biol* **8**, 153-158.
45. Rivera JA, Barquera S, Campirano F *et al.* (2002) Epidemiological and nutritional transition in Mexico: rapid increase of non-communicable chronic diseases and obesity. *Public Health Nutr* **5**, 113-122.
46. Rivera JA, Barquera S, Gonzalez-Cossio T *et al.* (2004) Nutrition transition in Mexico and in other Latin American countries. *Nutr Rev* **62**, S149-157.
47. Azcorra H, Varela-Silva MI, Rodriguez L *et al.* (2013) Nutritional status of Maya children, their mothers, and their grandmothers residing in the city of Merida, Mexico: Revisiting the leg-length hypothesis. *Am J Hum Biol* **23**, 398-400.
48. Dickinson F, Garcia A, Perez S (1999) Social differentiation and urban segregation in a Mexican regional metropolis. In *Problems of megacities: social inequalities, environmental risk and urban governance*, pp. 345-358 [A Guillermo Aguilar and I Escamilla, editors]. Mexico, D. F.” Universidad Nacional Autonoma de Mexico.
49. Fuentes JH (2005) *Espacios, actors, practicas e imaginarios urbanos en Merida, Yucatan, Mexico*. Merida, Mexico: Universidad Autonoma de Yucatan.
50. Frenk J (2006) Bridging the divide: global lessons from evidence-based health policy in Mexico. *Lancet* **368**, 954-961.
51. Frenk J, Gonzalez-Pier E, Gomez-Dantes O *et al.* (2006) Comprehensive reform to improve health system performance in Mexico. *Lancet* **368**, 1524-1534.
52. World Bank (2010) <http://data.worldbank.org/country/mexico>. (accessed 13 October 2011).
53. UNICEF (2011) Millennium Development Goals. <http://www.unicef.org/mdg/>. (accessed 5 December 2011).
54. Lohman TG, Roche AF, Martorell R (1988) *Anthropometric standardization reference manual*. Champaign, IL: Human Kinetics Books.

55. DHS (1989) *Mexico: encuesta nacional sobre fecundidad y salud 1987. Demographic and Health Survey*. Mexico, D. F.: Institute for Resource Development/ Macro Systems, Inc, Columbia, Maryland, USA.
56. Frisancho AR (2008) *Anthropometric standards: an interactive nutritional reference of body size and body composition for children and adults*. Ann Arbor, Michigan: University of Michigan Press.
57. Lara-Esqueda A, Aguilar-Salinas CA, Velazquez-Monroy O *et al.* (2004) The body mass index is a less-sensitive tool for detecting cases of obesity-associated co-morbidities in short stature subjects. *Int J Obes Relat Metab Disord* **28**, 1443-1450.
58. Cole TJ, Bellizzi MC, Flegal KM *et al.* (2000) Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* **320**, 1240-1243.
59. McCarthy HD, Cole TJ, Fry T *et al.* (2006) Body fat reference curves for children. *In J Obes* **30**, 598-602.
60. Wilson HJ, Dickinson F, Griffiths PL *et al.* (2011) How useful is BMI in predicting adiposity indicators in a sample of Maya children and women with high levels of stunting? *Am J Hum Biol* **23**, 780-789.
61. Lohman TG, Caballero B, Himes HJ *et al.* (1999) Body composition assessment in American Indian children. *Am J Clin Nutr* **69**, 764S-766S.
62. Stolarczyk LM, Heyward VH, Hicks VL *et al.* (1994) Predictive accuracy of bioelectrical impedance in estimating body composition of Native American woman. *Am J Clin Nutr* **59**, 964-970.
63. Cohen J (1992) A power primer. *Psychol Bull* **112**, 155-159.
64. Bogin B (1999) *Patterns of Human Growth*. 2nd ed. Cambridge: Cambridge University Press.
65. Roche AF (1992) *Growth, Maturation and Body Composition: The Fels Longitudinal Study 1929-1991*. Cambridge, UK: Cambridge University Press.
66. Gurri FD, Pereira GB, Moran EF (2001) Well-being changes in response to 30 years of regional integration in Maya populations from Yucatan, Mexico. *Am J Hum Biol* **13**, 590-602.
67. Gurri FD, Balam G (1992) Regional integration and changes in nutritional status in the central region of Yucatan, Mexico: A study of dental enamel hypoplasia and anthropometry. *J Hum Ecol* **3**, 417-432.
68. Izquierdo OP, Beutelspacher AN, Izaba BS *et al.* (2012) Frecuencia del consume de alimentos industrializados modernos en la dieta habitual de comunidades mayas de Yucatan, Mexico. *Estudios Sociales* **20**, 155-184.
69. Oddo VM, Rah HJ, Semba RD *et al.* (2012) Predictors of maternal and child double burden of malnutrition in rural Indonesia and Bangladesh. *Am J Clin Nutr* **95**, 951-958.
70. Drewnowski A, Specter SE (2004) Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr* **79**, 6-16.
71. McCurdy K, Gorman KS (2010) Measuring family food environments in diverse families with young children. *Appetite* **54**, 615-618.
72. Beydoun MA, Wang Y (2008) How do socio-economic status, perceived economic barriers and nutritional benefits affect quality of dietary intake among US adults? *Eur J Clin Nutr* **62**, 303-313.
73. Darmon N, Drewnowski A (2008) Does social class predict diet quality? *Am J Clin Nutr* **87**, 1107-1117.

Tables

Table 1: Anthropometric variables for urban Yucatec Maya children and mothers*

	Children						Mothers	
	Boys		Girls		All		Mean	SD
	Mean	SD	Mean	SD	Mean	SD		
N (%)	30	52.6	27	47.4	57	100	57	100
Age (yrs)	8.33	0.82	8.56	0.72	8.44	0.78	34.28	6.33
Height (cm)	122.23	5.62	122.47	7.80	122.34	6.68	146.84	4.58
Height z-score¹	-1.09	0.85	-1.19	0.88	-1.13	0.86	-2.20	0.73
Weight (kg)	26.10	4.51	28.16	7.86	27.07	6.35	63.69	10.06
Weight z-score¹	-0.59	0.78	-0.22	0.91	-0.42	0.86	-0.08	0.63
BMI	17.38	2.20	18.50	3.69	17.91	3.02	29.52	4.21
BMI z-score¹	0.54	0.83	0.64	1.02	0.58	0.92	1.59	1.33
Waist circumference² (cm)	58.85	5.64	61.23	9.46	59.98	7.71	88.46	9.05
Waist circumference z-score¹	0.18	0.68	0.57	0.82	0.37	0.77	0.30	0.58
Percent body fat^{3,4,5}	26.84	6.22	30.01	7.18	28.40	6.81	42.28	4.28

¹Age and sex specific z-scores created using the Comprehensive reference⁽⁵⁵⁾

²One mother excluded from waist circumference measurements due to pregnancy, n=56

³Children's percent body fat calculated using an indigenous North American equation⁽⁶⁰⁾

⁴Women's percent body fat calculated using an indigenous North American women equation⁽⁶¹⁾

⁵Missing data for mothers' %BF, this analysis n=54

*No significant difference found between the sexes using an independent *t*-test, *p*<0.05

Table 2: Influence of maternal height on children's body mass index z-scores in urban Yucatec Mayans using multiple linear regression*

	Model 1			Model 2		
	B	SE	P	B	SE	P
Constant	1.720	3.995	0.669	1.110	4.224	0.794
Maternal height (cm)	-0.008	0.027	0.771	-0.004	0.029	0.894
Sanitation index				-0.096	0.188	0.613
Maternal parity¹	3-8 v. 1-2			0.304	0.358	0.399
	1-2, 4-8 v. 3			0.151	0.322	0.641
R² adj			-0.017			-0.058

¹The highest tertile (4-8 children) set as the reference

*Model 1 tests for intergenerational influences of maternal chronic early life environment. Model 2 controls for the child's own environmental exposures.

Table 3: Influence of maternal height on children's waist circumference
z-scores in urban Yucatec Mayans using multiple linear regression*

			Model 1			Model 2		
			B	SE	p	B	SE	P
Constant			0.538	3.291	0.871	0.635	3.436	0.854
Maternal height (cm)			-0.001	0.022	0.950	-0.004	0.024	0.872
Sanitation index						0.028	0.153	0.853
Maternal	3-8 v. 1-2					0.426	0.291	0.150
parity ¹	1-2, 4-8 v. 3					0.233	0.262	0.378
R ² adj			-0.019			-0.034		

¹The highest tertile (4-8 children) set as the reference

*Model 1 tests for intergenerational influences of maternal chronic early life environment. Model 2 controls for the child's own environmental exposures.

Table 4: Influence of maternal height on children's percentage body fat in urban Yucatec Mayans using multiple linear regression*

			Model 1		Model 2			
			B	SE	P	B	SE	P
Constant			20.869	29.568	0.484	14.138	29.613	0.635
Maternal height (cm)			-0.019	0.193	0.922	-0.016	0.195	0.937
Child's age			0.782	1.153	0.500	1.270	1.133	0.268
Child's sex ¹			2.329	1.792	0.200	1.770	1.786	0.327
Sanitation index						-0.209	1.249	0.868
Maternal parity ²			3-8 v. 1-2			6.230	2.423	0.013
			1-2, 4-8 v. 3			3.331	2.187	0.134
R ² adj					-0.011			0.056

¹Boys set as the reference

²The highest tertile (4-8 children) set as the reference

*Model 1 tests for intergenerational influences of maternal chronic early life environment. Model 2 controls for the child's own environmental exposures.