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

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1 **Title**

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

4

5 **Abstract**

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

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

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

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

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

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

26

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 nately, 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-nately, 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

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

7

8 **Experimental Methods**

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

11 *Study population and location*

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

23 This study focuses on an urban population of Maya living in the south of Merida, Yucatan,
24 Mexico. Mexico is undergoing nutrition transition^(45, 46). Merida is a rapidly growing city, home
25 to approximately 75,000 Maya speakers in 2010, roughly 9% of the city's population⁽⁴⁷⁾. Merida
26 is highly segregated from north to -south in terms of wealth, infrastructure and services^(48, 49).
27 The south of the city has few services and is physically separated from the rest of the city by an
28 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.

20

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.

25

26

1 *Bioelectrical Impedance analysis*

2 To assess body composition, bioelectrical impedance analysis (BIA) was measured (BioScan
3 916, Maltron, UK) on children and mothers and used to calculate percentage of body fat.

4

5 *Interview*

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

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

21

22 Data analysis

23 *Anthropometry*

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

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

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

8

9 *Bioelectrical impedance analysis*

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

14

15 **Equation 1:** Child's percentage body fat = -0.49 (age) + 0.51 (sex) + 0.44 (weight) + 1.55
16 (triceps skinfold) + 0.15 (subscapular skinfold) + 0.54 (stature² / resistance) + 0.13 (reactance) –
17 0.04 (triceps skinfold x stature² / resistance) – 10.91

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

20

21 **Equation 2:** Women's fat free mass = 0.001254 (stature²) – 0.04904 (reactance) + 0.1555
22 (weight) + 0.1417 (impedance) – 0.0833 (age) + 20.05

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

24

25 Fat free mass was transformed into %BF by subtracting fat free mass from weight and dividing
26 the resulting number by weight and multiplying by 100. [(weight – FFM) / weight] x 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-variables in
28 the multiple linear regression models were selected to control for the children's own

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

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

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

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

19

20 **Results**

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

24 These Maya children were short (more than one standard deviation below the reference median)
25 and light but had adiposity indicators whose means were above the reference median (Table 1).
26 No sex differences in the children's anthropometry were found. Mothers were short (mean
27 stature 147 cm) with 44 (77.2%) being classified as stunted. Mothers were also heavy, with high
28 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.

3

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5

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1 **Tables**

2

3

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

	Children						Mothers	
	Boys		Girls		All		Mean	SD
	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

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

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

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

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

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

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

11

12

13 Table 2: Influence of maternal height on children's body mass index z-

14 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

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

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

18

19

1 Table 3: Influence of maternal height on children's waist circumference
 2 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 parity¹	3-8 v. 1-2			0.426	0.291	0.150
	1-2, 4-8 v. 3			0.233	0.262	0.378
R² adj			-0.019			-0.034

3 ¹The highest tertile (4-8 children) set as the reference
 4 *Model 1 tests for intergenerational influences of maternal chronic early life
 5 environment. Model 2 controls for the child's own environmental exposures.

6
7

8
 9 Table 4: Influence of maternal height on children's percentage body fat in
 10 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 sex¹	2.329	1.792	0.200	1.770	1.786	0.327
Sanitation index				-0.209	1.249	0.868
Maternal parity²	3-8 v. 1-2			6.230	2.423	0.013
	1-2, 4-8 v. 3			3.331	2.187	0.134
R² adj			-0.011			0.056

11 ¹Boys set as the reference
 12 ²The highest tertile (4-8 children) set as the reference
 13 *Model 1 tests for intergenerational influences of maternal chronic early life environment.
 14 Model 2 controls for the child's own environmental exposures.