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.

Introduction

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

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

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

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

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

The aim of this study is to determine whether maternal chronic under-nutrition in early life, as measured by mother's adult stature, has an intergenerational influence on the adiposity of their 7-9 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

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

Recruitment and ethics

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

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

Data collection

Anthropometry

Stature, weight, waist circumference (WC) and skinfolds (triceps and subscapular) were 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 (age) + 0.51 (sex) + 0.44 (weight) + 1.55 (triceps skinfold) + 0.15 (subscapular skinfold) + 0.54 (stature^2 / resistance) + 0.13 (reactance) – 0.04 (triceps skinfold x stature^2 / 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 (stature^2) – 0.04904 (reactance) + 0.1555 (weight) + 0.1417 (impedance) – 0.0833 (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. [(weight – FFM) / weight] x 100.

Socioeconomic status and sanitation indices creation

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

Statistical analysis

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

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

The predictors were entered in two steps to determine whether each type of indicator mediated or attenuated the relationship of maternal stature on child's adiposity indicators. The steps were: 1) maternal stature, 2) child's environmental exposures (sanitation index, maternal parity). Maternal stature was used as an indicator of maternal early life environment. The co-variates in 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%).

*****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

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

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

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

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

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

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

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

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

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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-score[1]	-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-score[1]	-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-score[1]	0.54	0.83	0.64	1.02	0.58	0.92	1.59	1.33
Waist circumference[2] (cm)	58.85	5.64	61.23	9.46	59.98	7.71	88.46	9.05
Waist circumference z-score[1]	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

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

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

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

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

[5]Missing 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 parity[1]	3-8 v. 1-2				0.304	0.358	0.399
	1-2, 4-8 v. 3				0.151	0.322	0.641
R^2 adj			-0.017			-0.058	

[1]The 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 parity[1]	3-8 v. 1-2				0.426	0.291	0.150
	1-2, 4-8 v. 3				0.233	0.262	0.378
R^2 adj			-0.019			-0.034	

[1]The 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	

[1]Boys set as the reference

[2]The 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.