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Health indicators in double burdened urban Maya children and mothers

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A doctoral thesis

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Abstract

Background

Middle-income countries are currently undergoing nutrition transition more rapidly than did high income countries. These populations are therefore at high risk of over-nutrition (obesity) and nutrition-related, non-communicable diseases before the elimination of under-nutrition and infectious diseases. Such nutritional double burden is most common in low SES groups of middle-income countries, such as the Maya of Mexico. Long-term poor environmental conditions during early life results in a population with high levels of chronic under-nutrition (stunting), and a consequent predisposition toward overweight/ obesity, and associated health risks later in life. It is important to be able to identify individuals at an increased risk of diseases related to double burden and to determine whether stunting impacts the ability to identify at risk individuals.

Aims

The overall purpose of this doctoral research was to examine double burden in the urban Maya, a low SES section of the Mexican population which is a current example of a population undergoing significant transition.

The specific aims of this thesis were fourfold: 1) To describe the living conditions and population characteristics of the urban Maya of southern Merida in the spring and summer of 2010; 2) to determine whether body mass index (BMI) predicts adiposity indicators in a sample of women and children with a high prevalence of stunting, 3) to determine whether measures of linear growth in women can be predicted by their recalled childhood environment and 4) To determine whether the relationship between objectively estimated free-living energy expenditure and body composition is altered by stunting.

Methods

Data from interviews, anthropometric measurements and bioelectrical impedance analysis were collected on 58 Maya schoolchildren aged 7-9 years-old and their mothers living in the south of Merida, Mexico. Objective, free-living physical activity monitoring using combined accelerometry and heart rate monitoring of the children was also performed for one week. The interview data was used to describe

the living conditions of the south of Merida. Whether stunting status or body proportions influenced the power of BMI to predict adiposity indicators was assessed for the mothers and the children. The mothers' recalled early life SES was compared to their current measures of linear growth. The children's objectively estimated energy expenditure was compared to their body composition and measurements of body size.

Results

The Maya of southern Merida overall had access to clean drinking water and basic health care and have apparently eliminated acute under-nutrition. Yet they remained double burdened with simultaneous stunting and overweight/ obesity. Individual double burden levels were high, with 70% of the mothers were simultaneously stunted and overweight. Family level double burden was also high, with 28% of the families having an overweight mother and a stunted child. The rates of childhood malnutrition varied widely when using different cut-offs. Child stunting rates were between 15.5% and 37.9% when using -2 z-scores of Frisancho's Comprehensive (created using NHANES data) reference versus the 5th percentile of the WHO reference, respectively. Child overweight/ obesity rates were less than 10% when using weight-for-age on both the Comprehensive and WHO reference charts. Child overweight/ obesity as classified using BMI z-scores was between 27.5 to 34.5% using the Comprehensive and WHO reference, respectively, while child overfat was over 80% when using body fat percentage for age reference curves. BMI predicted adiposity indicators in these Maya children, explaining between 33 and 84% of the variance in arm fat index and waist circumference z-score, respectively. BMI was less strongly related to the mothers' adiposity indicators, explaining between 19 and 70% of the variance in arm fat area and waist circumference. The relationship between BMI and adiposity indicators was unchanged by stunting or body proportions in either mothers or children. Mothers' recalled early life SES was significantly related to but explained little of the variance in her measures of linear growth. Birth decade explained 5% of the variance in stature and the Modernisation index (urban/ rural birth, sugar sweetened beverage consumption, packaged food consumption) explained 5% of the variance in mothers' leg length. Birth order, sibling number and consumer durable ownership were also significantly related to linear

growth of the mothers. These Maya children had high levels of physical activity, as all exceeded the recommended 60 minutes of moderate-to-vigorous physical activity per day. After controlling for fat free mass, short stature did not predict lower resting energy expenditures in the children. However shorter stature did predict lower levels of activity energy expenditure, particularly in girls. Stunted girls had the lowest activity energy expenditures.

Conclusions

These urban Maya tend to have access to basic sanitation and services and are at a very high risk for NR-NCDs with the co-existence of chronic under-nutrition (stunting) and overweight/ obesity. The high rates of stunting do not impact the usefulness of BMI to estimate adiposity nor does stunting appear to impact children's energy expenditure. While BMI is useful to predict adiposity in these urban Maya children, it is not recommended for use in the mothers. Interventions to reduce childhood adiposity need to begin very early in life to most effectively reduce adiposity. Research into the low SES groups of middle-income countries, offers insight to what may occur in low-income countries as they advance in the nutrition transition.

Key words:

Nutrition transition, Mexican Maya, mother-child dyads, double burden, BMI, adiposity, energy expenditure, objective physical activity monitoring

This thesis is dedicated to the women in my family, particularly my mother Sarah Hodson Overmyer Wilson as well as my grandmothers, Margery Louise Hodson Overmyer and Ruth Goates Worlton Wilson. Without them, I am nowhere.

Fate doesn't hang on a wrong or right choice. Fortune depends on the tone of your voice.

-Ben Folds

There is nothing to writing. All you do is sit at the typewriter and bleed.

-Ernest Hemingway

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Publications

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Poster presentations

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Abbreviations

| | |
|---------|---|
| %BF | Percent body fat |
| 2SF | Sum of 2 skinfolds (triceps and sub-scapular) |
| AEE | Activity energy expenditure |
| AEE/kg | Activity energy expenditure divided by body weight in kilograms |
| AEE/FFM | Activity energy expenditure divided by fat free mass in kilograms |
| AFA | Arm fat area |
| AFI | Arm fat index |
| BIA | Bioelectrical impedance analysis |
| BMI | Body mass index (kg/m^2) |
| BMR | Basal metabolic rate |
| CDC | Center for Disease Control |
| CVD | Cardio-vascular disease |
| DHS | Demographic and health survey |
| DOHaD | Developmental origins of health and disease |
| DXA | Dual-energy X-ray absorptiometry |
| ECG | Electro-cardiogram |
| ELL | Estimated leg length |
| FM | Fat mass |
| FMI | Fat mass index (fat mass / stature) |
| FMI2 | Fat mass index (fat mass / stature ²) |
| FMI6 | Fat mass index (fat mass / stature ⁶) |
| FFM | Fat free mass |
| FFMI | Fat free mass index (fat mass / stature) |
| FFMI2 | Fat free mass index (fat mass / stature ²) |
| FFMI3 | Fat free mass index (fat mass / stature ³) |
| FFQ | Food frequency questionnaire |
| GDP | Gross domestic product |
| INCAP | Instituto de Nutrition de Centro America y Panama |
| IOTF | International Obesity Task Force |

| | |
|---------|---|
| KHR | Knee height ratio |
| kJ | kiloJoule |
| J | Joule |
| MAC | Mid-upper arm circumference |
| MET | Metabolic equivalent |
| MJ | MegaJoule |
| NHANES | National health and nutrition examination survey |
| NR-NCDs | Nutrition related- non communicable diseases |
| OF | Overfat |
| OW/OB | Overweight/ obesity |
| PCA | Principle component analysis |
| PAL | Physical activity level |
| PAL/kg | Physical activity level divided by body weight in kilograms |
| PAL/FFM | Physical activity level divided by fat free mass in kilograms |
| RMR | Resting metabolic rate |
| RMR/kg | Resting metabolic rate divided by body weight in kilograms |
| RMR/FFM | Resting metabolic rate divided by fat free mass in kilograms |
| SES | Socioeconomic status |
| SHR | Sitting height ratio |
| TUA | Total upper arm area |
| UMA | Upper arm muscle area |
| WHO | World Health Organization |
| WC | Waist circumference |

Chapter 1. *Introduction*

This chapter introduces the concepts of that are central to this thesis, primarily the relationship between nutrition transition and nutritional status in low socio-economic status groups in middle-income countries.

Throughout the developing world, traditional, rural ways of life based upon agriculture are being replaced with urbanised lifestyles dependent upon purchased foods, a process known as the nutrition transition (Popkin 1993). With this transition comes a decrease in acute under-nutrition and infectious diseases while caloric availability, mechanisation, incidence of non-communicable diseases and life expectancy rise. As lifestyles become more mechanised and calories become easier to obtain, rates of overweight/ obesity (OW/OB) increase in children and adults. Obesity is associated with nutrition-related non-communicable diseases (NR-NCDs) (e.g. cardiovascular diseases (CVD) and type II diabetes) and decreased economic productivity (*ibid*). NR-NCDs most often afflict individuals in later life but increasingly risk factors for and diagnoses of NR-NCDs are being made in children and adolescents as rates of childhood OW/OB rise (James 2006).

The nutrition transition is occurring much faster in currently developing countries than it did in Western Europe and North America, and in some populations, can occur within a generation (Popkin 2002). Such a rapid transition can lead to a population facing diseases related to over-nutrition before eliminating the problems of under-nutrition. In recent years, the co-existence of chronic under-nutrition (linear growth stunting) and acute over-nutrition (a situation known as the nutrition double burden) have been found in populations, households and even individuals, particularly in middle-income countries (Delisle 2008; Doak et al. 2000; Garrett and Ruel 2005; Neufeld et al. 2008; Varela Silva et al. 2011).

Double burden may be difficult to avoid during rapid nutrition transition. Rates of OW/OB in stunted adults are higher than their non-stunted peers in many middle-income countries (Bosy-Westphal et al. 2009; Florencio et al. 2007; Lopez-Alvarenga et al. 2003). This is likely a type of response related to the Developmental Origins of Health and Disease (DOHaD), which describes the broader relationship between early life events and later health (Barker 1995a). Responses to early life insults likely predispose an individual to later life obesity in times of caloric abundance (Leonard et al. 2009). Examining how childhood conditions are related to health may therefore provide useful information about risk for later health outcomes.

With the shift in disease burdens away from those related primarily to acute under-nutrition (e.g. diarrhoea) (UNICEF and WHO 2009) toward those related to over-nutrition (WHO 2006) it is necessary to be able to accurately identify individuals at risk for negative health outcomes. The nutrition transition is largely occurring in

populations that are not directly comparable to populations of European descent either in historical or in genetic terms (Popkin 2002). Thus the tools currently available to identify health risk, which have been developed primarily in European populations, must also be assessed for use in transitioning populations.

This research focuses on the Maya of Mexico. Mexico is an upper-middle-income country which, on the whole, rates relatively favourably in most measures of development, having low and declining rates of infant mortality and under-nutrition alongside high rates of school enrolment and OW/OB. Yet, taking these national average rates at face-value masks difficulties faced by the most marginalised groups. In the middle- and high-socio-economic status (SES) groups of Mexico there is an inverse relationship between wealth and OW/OB, similar to the pattern found in high income countries (Rivera et al. 2002). The low-SES groups tend to be at an earlier stage of the nutrition transition than the high SES groups and have higher rates of under-nutrition. The type of under-nutrition is primarily chronic under-nutrition (stunting) as acute under-nutrition (wasting) is universally low in Mexico (Lutter et al. 2011). In the low SES groups of Mexico, OW/OB is directly associated with increasing wealth in a similar pattern to that found in low-income countries (Fernald 2007). Thus the low-SES groups of Mexico are at the highest risk for nutritional double burden.

Throughout most of the Americas, including Mexico, the Amerindians groups, such as the Maya, have undergone systematic marginalisation since colonial times. While the systematic marginalisation has largely been abolished, Maya still tend to be low SES and are at risk for the associated negative health outcomes. Historically, the Maya are farmers, with agriculture and maize, particularly, playing a central role in their culture and religion. As with most groups in the developing world, many Maya now live in cities and are shifting away from their traditional way of life. Previous studies with the Maya have found that they respond dramatically to changes in environmental conditions (Smith et al 2002), making them a good population to study in the context of the nutrition transition.

The nutrition transition is occurring rapidly in many countries throughout the world, including middle-income countries such as Mexico. Low-income and SES populations often bear the brunt of the consequences of the transition. In middle-income countries, many groups have been systematically marginalised, like the Maya, which places them at a higher risk of being low SES. Understanding the

health risks in these groups is therefore important for health policy planning in these contexts to reduce the inequalities.

Therefore, to better understand these health risks and how they accumulate through childhood, the aims and hypotheses of this thesis are:

Aim 1. To describe the living conditions and population characteristics of the urban Maya of southern Merida in the spring and summer of 2010.

Aim 2. To determine whether body mass index (BMI) predicts adiposity indicators in a sample of women and children with a high prevalence of stunting.

This aim will be specifically tested through the following null hypotheses:

Hypothesis 2a: Stunting will not significantly alter the relationship between child's BMI-for-age z-scores and adiposity indicators (percent body fat; waist circumference z-score; sum of 2 skinfolds; upper arm muscle area z-score; upper arm fat area z-score; and arm fat index z-score).

Hypothesis 2b: Sitting height ratio will not significantly alter the relationship between child's BMI-for-age z-scores and adiposity indicators (percent body fat; waist circumference z-score; sum of 2 skinfolds; upper arm muscle area z-score; upper arm fat area z-score; and arm fat index z-score).

Hypothesis 2c: Stunting will not significantly alter the relationship between adult women's BMI and adiposity indicators (percent body fat, waist circumference, upper arm muscle area, upper arm fat area, arm fat index).

Hypothesis 2d: Sitting height ratio will not significantly alter the relationship between adult women's BMI and adiposity indicators (percent body fat, waist circumference, upper arm muscle area, upper arm fat area, arm fat index).

Aim 3. To determine whether measures of linear growth in women can be predicted by their recalled childhood environment.

This aim will be specifically tested through the following null hypotheses:

Hypothesis 3a: A woman's recalled early life environment will not significantly predict her adult stature.

Hypothesis 3b: A woman's recalled early life environment will not significantly predict her adult estimated leg length.

Hypothesis 3c: A woman's recalled early life environment will not significantly predict her adult sitting height ratio.

Aim 4. To determine whether the relationship between objectively estimated free-living energy expenditure and body composition is altered by short stature.

This aim will be specifically tested through the following null hypotheses:

Hypothesis 4a: Stunting will not significantly alter the relationship between fat free mass and total energy expenditure in children.

Hypothesis 4b: Height-for-age z-score will not significantly alter the relationship between fat free mass and total energy expenditure in children.

Hypothesis 4c: Stunting will not significantly alter the relationship between fat free mass and resting energy expenditure in children.

Hypothesis 4d: Height-for-age z-score will not significantly alter the relationship between fat free mass and resting energy expenditure in children.

Hypothesis 4e: Stunting will not significantly alter the relationship between fat free mass and activity energy expenditure in children.

Hypothesis 4f: Height-for-age z-score will not significantly alter the relationship between fat free mass and activity energy expenditure in children.

A. Thesis structure

This section describes how the chapters of the thesis have been organised.

2. **Literature review:** focuses on the biosocial impacts on health, the double burden of simultaneous over- and under-nutrition as well as the measurement of health. The review is focused on low-income populations undergoing rapid nutrition transition.

3. **Methods:** describes the research methods used during data collection and data cleaning. The chapter also covers methods that are generally applicable to the overall thesis topic. Where relevant, methods that are specific to one results chapter are described in that chapter separately.

4. **The living conditions of the Maya of southern Merida:** aims to provide a contextual framework for the research project by describing the neighbourhood, living conditions and socioeconomic status of the Maya mother-child dyads. The variables that are necessary for later analyses are also shown in the relevant chapter.

5. Descriptive results for anthropometry and energy expenditure:

presents the descriptive results for the anthropometry of the urban Maya mothers and children and the energy expenditure of the children are described in this chapter.

6. The usefulness of BMI as a measure of adiposity in a population with very short stature: examines the relationship between BMI and adiposity indicators in a sample of urban Maya women and children and assesses whether stunting status impacts the relationship.

7. Mothers' measures of recalled early life and linear growth: examines whether early life environment as recalled by an adult Maya woman is able to significantly predict her adult measures of linear growth.

8. Child's energy expenditure, stunting and body composition: examines the relationship between urban Maya children's energy expenditure and stunting on anthropometric z-scores and body composition variables.

9. Discussion and conclusion: brings together the overall findings from each individual results chapter and addresses the main aims of the thesis, identifies gaps for further study and limitations associated with the research as well as discusses policy implications of the findings.

Chapter 2. *Literature review*

This chapter provides a review of the literature on biosocial impacts on health, the double burden of simultaneous over- and under-nutrition as well as the measurements of health. The review is focused on low-income populations undergoing rapid nutrition transition and where possible describes previous research conducted in Mexico or with the Maya to inform the aims and hypotheses of this thesis.

A. Search terms

The primary literature searches for this chapter were done in the Internet databases Web of Science and ScienceDirect. The searches were performed primarily using the following key words: Maya, Latin America, Mexico, developmental origins of health and disease, fetal origins of health, foetal origins of health, adult, child, life course, health care, health policy, diet, nutritional content, nutritional status, stunting, childhood stunting, adult short stature, linear growth retardation, chronic under-nutrition, mortality, morbidity, chronic diseases, diet-related non-communicable diseases, nutrition-related non-communicable diseases, cardiovascular disease, coronary heart disease, type II diabetes, non-insulin dependent diabetes, under-5 mortality, BMI, overweight, obese, adiposity, body composition, fat patterning, fat distribution, over-nutrition, economic penalty, economic cost, developing countries, low socio-economic status, urban poor, urbanisation, modernisation, socio-economic status measurement, long-term memory, secular trend, environment and growth, environment and health, nutrition transition, epidemiological transition, double burden, dual burden, simultaneous over- and under-nutrition, energy expenditure, metabolic rate, resting metabolic rate, basal metabolic rate, activity energy expenditure, total energy expenditure, physical activity, objective physical activity measurement, ActiHeart, combination accelerometry and heart rate monitor, accelerometry and heart rate monitoring.

This chapter will synthesize the literature found during the aforementioned literature searches. The chapter is arranged into the broad topics of the Maya of southern Merida, biosocial impacts on health, nutrition transition, under-nutrition, chronic under-nutrition, over-nutrition, nutritional double burden and measurement of health risks.

B. The Maya of southern Merida

This research focuses on a sample of Mexican Maya living in an urban environment which has access to most services which are considered necessary by the UNICEF (UNICEF 2010) but is still considered low socio-economic status (SES). Merida is a heterogeneous city, showing social segregation from the affluent north to the impoverished south, where this research took place.

1. *Southern Merida*

This research took place in the southern area of Merida. Merida is the capital of the state of Yucatan in southern Mexico and has been undergoing industrialisation and rapid expansion since the middle of the twentieth century. The city's population grew by 27% in the 1990s, and was home to over 660,000 people in 2000 (Azcorra 2007). Approximately 230,000 Mayas resided in Merida in 2005 (*ibid*).

Merida is a city that is highly segregated in terms of wealth, infrastructure and services. The north and central areas are the most affluent and the south is the least wealthy (*ibid*). The individuals living in the north are involved in the national and international economies and have easy access to services such as schools, markets and refuse collection. The relative lack of such services in the south is notable and individuals must leave the south to gain access to many services. The south is also physically separated from the rest of the city by an airport runway that cuts across the city (see Figure 2.1). This physical boundary makes leaving the impoverished area time consuming, further restricting access to services. The traditions that marginalise of the Maya results in many of the Maya being of low SES and therefore the Maya living in Merida are likely to live in the south of the city.

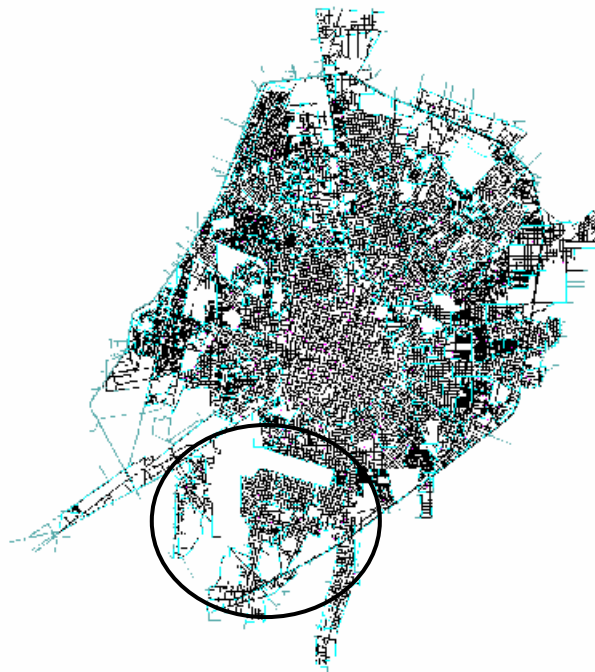


Figure 2.1 Map of Merida. The circle denotes the south area, physically separated from the rest of the city by the airport runway

2. *The Maya*

The Maya have been living in the Yucatan for over 1,000 years. Ever since Europeans began colonising America, all Amerindians have been systematically marginalised (Montejo 1999). The Maya are traditionally farmers, with agriculture and maize being central in their culture and religion (Quijano 2007). The land in the Yucatan is of poor quality, particularly near Merida due to intensive *sisal* cultivation that made the city rich. *Sisal* was one of the main fibres for rope making worldwide. The poor soil quality does not allow subsistence farming alone to support families (Montejo 1999). The income from alternative industries such as tourism or construction is used to offset the gap between income and expenses (Quijano 2007).

This sample of Maya lives in the city of Merida, Yucatan and has largely discarded their traditional farming lifestyle. While the reasons behind this behaviour shift were not addressed as part of this study, it is the impression of one of the local field-team members (primarily Hugo Azcorra (HA)) that two factors are responsible. The first is the poor soil in the Yucatan. Secondly, is a desire of the younger generations to distance themselves from the traditionally marginalised Maya. With distance from traditional culture and modernisation there is an increased potential for monetary wealth (Quijano 2007). In their attempt to gain wealth, they are also discarding much of their traditional culture including farming, language and dress.

3. *Maya genetics*

Though many of the Maya are discarding some of their cultural traditions, they can still be considered a distinct group through the basis of their genetics. Since genes and the genome form the basis of biology, genetic heterogeneity must be controlled in order to be confident that variation in the environment is the main cause of growth variation in a sample. Controlling for genetic variation is particularly important when considering Latin American populations due to the large amount of admixture that has occurred between Amerindian, European and African groups (Bonilla et al. 2005). Latin Americans vary greatly in the percentages of each lineage they carry (Seldin et al. 2007). The most logistically feasible and cost effective method of controlling for such genetic diversity is to sample a relatively genetically homogeneous group. Amerindians can be genetically differentiated from Hispanics of mixed descent (Mao et al. 2007). Little genetic research has been done specifically on the Maya, though several large scale studies have included Maya

(Gonzalez-Martin et al. 2008; Mao et al. 2007; Salzano 2002). These studies have found Maya groups to cluster geographically, distinct from other indigenous and non-indigenous groups with some genetic admixture between neighbouring indigenous groups. The Maya overall can be considered relatively genetically homogeneous (Ibarra-Rivera et al. 2008).

A more complete description of the living conditions of the Maya of southern Merida is found in Chapter 4.

C. *Biosocial impacts on health*

1. *Developmental origins*

In the late 1980s, Barker and colleagues found an individual's stroke risk to be more strongly related to their childhood living conditions than their adult living conditions (Barker et al. 1989a; Barker et al. 1989b). Since then, a large body of evidence has been collected that supports the so-called 'Developmental Origins of Health and Disease' hypothesis (DOHaD) in both animals (Reza-Lopez et al. 2009; Szeto et al. 2009) and humans (Baker et al. 2009; Dietz 1994; Drake and Walker 2004; Gluckman et al. 2008; Leonard et al. 2009; Parsons et al. 2001; Sharrock et al. 2008). DOHaD has been implicated in cardio-vascular disease (CVD) (Heys et al. 2011; Lumey et al. 2009; Siervo et al. 2010; Yu et al. 2011), type II diabetes (Martin-Gronert and Ozanne 2006) and chronic disease risk factors such as high blood lipid levels or hypertension (Gallo et al. 2011; McDade et al. 2005).

The influences of environment on later health may be more important during certain periods of life than others. Throughout the growing years, individuals undergo several critical periods where they are most sensitive to environmental exposures (Cameron and Demerath 2002). These critical periods are characterised by rapid growth and include several life stages: prenatal, infancy/childhood and adolescence. Gestational quality has been found to be significantly related to adult CVD mortality (Barker 1993), fat-free mass (Weyer et al. 2000), metabolic rate (Leonard et al. 2009; Weyer et al. 2000) and lipid profiles (Bunt et al. 2005; Lumey et al. 2009). Dietary quality and growth rate in infancy and early childhood have been related to adult mortality (Barker et al. 2002; Barker et al. 2011; Ness et al. 2005) and the

incidence of stroke (Barker et al. 2009), CVD (Eriksson et al. 2001; Hebert et al. 1993) and diabetes (Barker et al. 2009).

Several mechanistic hypotheses have been published about the link between early life and later disease (Barker 1995b; Gluckman and Hanson 2004; Kuzawa 2005). Most of these hypotheses predict that in response to an insult during one of the critical periods of early life, the metabolism or other regulatory mechanisms in the body are permanently programmed, perhaps in an attempt to cope with future similar insults (Barker 1995a; Gluckman and Hanson 2004; Kuzawa 2005; Kuzawa and Pike 2005). This programming is theoretically beneficial in stable environments as it increases the odds that the child will survive the initial insult and any similar insults in the future. However in populations undergoing rapid changes in diets and lifestyles, early life insults do not necessarily predict insults in adulthood. In currently developing countries, early life under-nutrition is often followed with caloric abundance in later life. The metabolic shifts made toward the early under-nutrition appear to increase the risk of overweight/obesity (OW/OB) and CVD. For example, women who were pregnant during the Dutch Famine of World War II, had children who were more likely to have elevated lipid levels than children who did not experience the Dutch Famine while *in utero*. This effect remained even after controlling for birth weight (Lumey et al. 2009). Also, in an urban population from the Philippines, the mothers had experienced more early life insults and consumed a less obesogenic diet yet were more likely to be OW/OB than their children (Kelles and Adair 2009).

Another factor which operates during early life to impact an individual's later health is SES.

2. Socio-economic influences on health

SES is an indicator that is used as a proxy for the overall quality of the environment, including both social and economic aspects of individuals, households, communities and nations (Bogin 1999). The influence of SES on an individual varies over the life course and with the type of SES being considered. Levels of SES include individual, family, community, regional and national and can be classified based upon their proximity to the individual. Proximal levels, individual and family SES, are very tightly linked with the individual while the more distal levels, community, regional and national, influence the individual in more subtle ways.

Interaction between all levels of SES (e.g. individual and national) occurs as well, complicating the relationship (Fotso and Kuate-Defo 2005). It is worth noting that the proximity of the considered aspect of SES to the individual is influenced by the interaction between more distal levels (Glass and McAtee 2006). For example, a widespread economic recession reduces the job opportunities at the local level, limiting the ability of a family to secure employment. Such financial stress may lead to inter-personal stress within the family and a deteriorating diet and health care for family members leading to health deterioration within the family, to which the youngest household members will likely be the most vulnerable.

Distal SES levels (e.g. community and national levels), have a large impact on individual health and growth. Individuals who migrate to an area with improved infrastructure and access to goods and services tend to have better health outcomes than their peers who have not migrated. For example, Maya children living in the more affluent United States are more than 10 cm taller than those living in Guatemala (Smith et al. 2003). Measures of linear growth, such as stature, are used as an indicator of chronic nutritional status with taller individuals being considered to have experienced better chronic environmental conditions during their growing years (Bogin and Keep 1999; Komlos 1985; Steckel 2009).

It is also commonly found that rural to urban migrant children have better growth outcomes than those who have not migrated. The higher density of facilities and goods in urban areas, such as employment, governmental policy and health care (Frenk et al. 2003) and food availability (Bunt et al. 2005; Silventoinen et al. 2004), tend to be implicated in improved growth and health outcomes rather than the urban environment itself. The access to such facilities and goods are broad indicators for the SES of a community and as such the individuals and families residing in the community/ region/ country.

It must be noted that SES at the individual and family level is also important for health and growth of the child and can modify the more distal measures of SES, such as community SES (Glass and McAtee 2006). Due to high levels of heterogeneity in SES within countries and communities the types of SES most proximal to the individual must also be measured. For example, the educational attainment of individuals is related to their economic earnings (Burnett 2008; Engle and Black 2008; Ostrove and Feldman 1999) and educated women tend to have healthier children than non-educated women, (McCrary and Royer 2011). Also,

family material wealth has been shown to relate to measures of health and growth (Gunnell et al 1998). While an individual's health must be placed in context with the wider community, their own individual and family SES also plays an important role (Glass and McAtee 2006). The remainder of this thesis will primarily focus upon SES at the family and individual levels, particularly in the context of nutrition transition.

D. *Nutrition transition*

1. *Definition*

Currently, developing countries are undergoing the nutrition transition. The phrase nutrition transition encompasses many different types of transition: demographic, economic, dietary and behavioural (Popkin 1993). As countries are becoming wealthier with a reduction in infectious disease burden and, in tandem, are changing their diet and behaviour. The changes associated with nutrition transition have been divided into stages by Popkin (1993): hunter-gatherer, famine, receding famine, degenerative diseases and behavioural change stages. While most countries regardless of economic wealth are undergoing some stage of nutrition transition, this thesis will focus on the shift from receding famine toward degenerative diseases. This is the stage of nutrition transition that is occurring in Mexico (Rivera et al. 2002) and Latin America (Rivera et al. 2004).

Nutrition transition is related to the economy of the population and typically occurs when the economy shifts from being agriculturally based to more industrial or service orientated alongside urbanisation. During transition, the diet shifts toward purchased, processed foods. These behavioural shifts tend to coincide with a demographic transition of decreasing fertility and infectious disease mortality toward chronic disease mortality (Popkin 1993).

The Maya are traditionally farmers, with maize playing a central role in their culture and religion. With modernisation and urbanisation occurring within the Yucatan and Mexico, many Maya now work and live in urban areas or rely on tourism for an income and tend to be of low SES. The current Mexican (Barquera et al. 2008) and Maya (Leatherman and Goodman 2005) diet is highly dependent on purchased foods rather than subsistence agriculture. These purchased foods are often processed, calorie-dense and nutrient-poor, being deficient in micro-nutrients such as vitamins A, B2, B12, E and zinc (Leatherman and Goodman 2005). Within

the Mexican Maya, there appears to be a SES gradient in dietary quality with higher income families having a better diet than families with low or unsteady incomes (*ibid*). Mexico was the second largest consumer of soft drinks in the world in 2005, drinking more than 16 billion litres of Coca-Cola alone in 2005 (ANAPRAC 2005). Mexican adolescents consume more than 20% of their caloric intake in the form of beverages including soft drinks (Barquera et al. 2008). Mexicans and the Maya of the Yucatan are currently consuming a diet that places them at risk for NR-NCDs.

The physical activity levels of Mexican children also appear to be influenced by the nutrition transition. Indigenous Mexican children living a subsistence life style have been shown to be highly active (Malina et al. 2008a). However urban Mexican children have been shown to decrease their levels of physical activity upon entry to primary school (Jauregui et al. 2011) and also in adolescence (Siegel et al. 2011). In urban Mexican children and adolescents, the risk of OW/OB has been shown to increase with television viewing (Hernandez et al. 1999). The behaviour changes associated with the nutrition transition may play a role in the risk for NR-NCDs in urban Mexican children.

2. Disease profile in transition

In the epidemiologic transition, the nutritional shift from receding famine to degenerative diseases is characterised by a transition from an agriculture based economy, toward an economy relying on service industries, with a diet high in purchased foods (Popkin 1993). This economic transition is characterised by the replacement of food high in carbohydrates and fibre toward foods high in fat, calorically dense and low in fibre (Popkin 1996). Such calorie-dense, nutrient-poor foods are often highly processed and purchased. This dietary shift is closely associated with a change in the disease profile of a population. The disease profile tends to transition from a disease load of predominately under-nutrition and infection toward nutrition-related non-communicable diseases (NR-NCDs) (Rivera et al. 2004). For example, the under-5 mortality in Mexico dropped from almost 300 per 100,000 to roughly 75 per 100,000 between 1978 and 1993 (Frenk et al. 2003). During the 1990s, the prevalence of diarrhoea decreased by more than 30% in children (*ibid*). Mexico saw an increase of more than 50% in age-standardised mortality due to NR-NCDs between 1980 and 1999 (Rivera et al. 2004). The rise in

incidence of chronic disease within Mexico has also been rapid (Menezes et al. 2005; Rivera et al. 2002)

The change in disease profile of a population undergoing nutrition transition results in a substantial shift in the focus of health care. Infectious diseases can be addressed by campaigns lead by the health sector, such as vaccination campaigns. NR-NCDs requires an approach that involves many more sectors (e.g.: education, transport, health, trade and agriculture) (Rasanathan and Krech 2011). While this is well understood on a theoretical level, many interventions have had a modest rather than a large impact on behaviour (Arnold et al 2009) or health outcomes (Gourland et al 2011) beyond the intervention period.

Disease profiles of countries are measured in several ways, including direct and indirect measures. Mortality and morbidity are direct measures and are commonly used to assess the type of diseases currently in a population and also the burden of disease (Barros et al. 2005; Helmke 2011). Indirect measures, such as measures of nutritional status, can also be useful to assess the risk of diseases. Under-nourished individuals are more susceptible to infectious diseases (UNICEF and WHO 2009) while over-nourished individuals are more at risk for CVD (Lee et al. 2008; WHO 2006). Nutritional status can be easily determined using a variety of cost effective methods, such as anthropometry (Chumlea 2002). Correct assessment of nutritional status, and therefore disease risk, is important for population monitoring to aid in health policy decisions.

It is useful to examine the overall trends in the association between nutrition transition and health occurring within a country and a population, yet it is also important to examine trends in finer detail in order to understand the heterogeneity within a population. Not all groups transition at the same rate and the low SES groups tend to transition at slower rates than high SES. For example, most of Mexico has mortality rates that are similar to, or better than, upper-middle income countries. However southern Mexico, including Merida, has the highest rate of maternal and infant mortality in all of Mexico, and is similar to a low-middle income country (Stevens et al. 2008).

E. *Under-nutrition*

Under-nutrition had traditionally been considered the leading health concern of developing countries (James 2008). Since a very large proportion of the world's

population lives in poverty, obtaining sufficient amounts of food has been the primary worry. Under-nutrition is a lack of metabolic reserves in the form of adipose tissue, and is measured by weight-for-age and weight-for-height. Acute under-nutrition is still common in parts of the world, such as India (Raj et al. 2009). However, Latin America appears to be reducing acute under-nutrition at an acceptable rate (Lutter et al. 2011; Rivera et al. 2004). The prevalence of underweight in Mexico was 3.4% in children under 5 years of age in 2006 (Lutter et al. 2011). Recent studies of Mexican Amerindian groups, one of the more likely groups to experience under-nutrition due to low SES, have not found a high prevalence of acute under-nutrition (Malina et al. 2011a; Malina et al. 2008a; Reyes et al. 2010).

F. *Chronic under-nutrition (stunting)*

Chronic nutritional status is assessed using measures of linear growth, most frequently stature (Steckel 1995). Stature is favoured because it is inexpensive and easy to measure and reflects chronic environmental conditions throughout the growing years (Bogin 1999). Linear growth is highly heritable but is also a complex trait that is controlled by many factors including genetics, hormones and energy availability (Bogin 1999). Energy is a finite resource and must be portioned between maintenance, repair, voluntary movement (physical activity) and growth in children. In adulthood, the energy previously available for growth is shunted to reproduction (Kuzawa 2005). When energy availability is low the body compensates by reducing the energetic cost of maintenance, slowing or stopping growth and, when possible, reducing physical activity. If energy availability is low for a brief period of time, mass and fat reserves are the main tissues reduced. If energy availability is insufficient for several months, linear growth will be slowed or stopped. Following increased energy availability, linear growth can be increased dramatically in a process known as catch-up growth (Tanner 1990). However if energy availability remains low for several years, catch-up growth may not be sufficient to compensate for the long term reduction in linear growth and stunting may occur (Bogin 1999).

Overall, when the living conditions change, so does the mean stature of the population. These secular trends in stature can be used to may reflect living conditions and can be used as a proxy measure (Bogin and Keep 1999; Komlos 1998; Steckel 2009). Stature is more reliable than subjective reports due to difficulties obtaining accurate subjective measures of SES due to difficulties in recall

and possible participant reticence. Such an objective measure of long term environment is very useful as economic development may not lead to an improvement in living conditions (Komlos 1998; Komlos 2008; Komlos and Coclanis 1997; Steckel 1995). For example, individuals who were raised in urban environments tend to be taller than those raised in rural environments (Garnier et al. 2003; Van de Poel et al. 2007). However similar or shorter statures have been found in poor urban areas or informal settlements compared to rural areas (Jenkins 1981; Van de Poel et al. 2007). Thus, simply migrating to an urban area does not universally improve living conditions and in some circumstances, may worsen them. This relationship is not surprising because it is the increased access and utilisation of services in urban areas that is related to improvements in stature (Van de Poel et al. 2007). Such services may be lacking in poor urban areas or informal settlements. Therefore, affluent rural families may have better access to services, such as health care, than low-income urban families.

When the chronic linear growth retardation is intense, stunting may occur. Stunting is defined as a very low height-for-age, the cut-offs for which are discussed in Chapter 3, section E.2 and Chapter 5 section A.3. Chronic under-nutrition, as measured by stunting, is an important health concern for Latin America (Lutter et al. 2011; Rivera et al. 2004). Due to differences in governmental stability and investment into the well being of the people, the prevalence of stunting in under-5 year olds in Latin America is between 5.6% (Costa Rica) and 54.5% (Guatemala) (Lutter et al. 2011). As they have progressed in the nutrition transition, every country in Latin America has reduced the rates of child stunting in the last 20 years. For example, the prevalence in Mexico has been reduced from 21.5% in 1999 to 15.5% in 2006 (*ibid*).

There appears to be a critical period for a long term linear growth failure, occurring in infancy and early childhood (Rehman et al. 2009; Stein et al. 2010). An examination of five birth cohort studies in developing countries found that linear growth failures occurred before 2 years of age and that adult stature was most strongly associated with the occurrence of growth failure before 5 years of life (Stein et al. 2010). It is likely that improper weaning (failure to correctly transition to solid foods with sufficient calories and nutrients to the infant at the proper time) plays a large role in stunting. Weaning is very important because breast milk does not provide sufficient nutrition for infants older than 6 months (Prentice and Paul 2000;

Whitehead and Paul 2000). The complementary food used in developing countries is often high in starch and low in protein, which is inadequate for proper growth (Prentice and Paul 2000). Gastro-intestinal diseases which often afflicts young children, such as diarrhoea, has also been implicated strongly in childhood stunting (Checkley et al. 2008). Regardless of the cause of stunting, if a stunted child is given caloric supplementation after the age of 5, then linear growth is not increased. Rather the excess calories are stored as adipose tissue (Walker et al. 1991).

Short stature in adulthood has repeatedly been linked with an increased risk of mortality and morbidity from CVD and all cause mortality (Langenberg et al. 2005; Lundberg et al. 2002; Paajanen et al. 2010; Song and Sung 2008). A recent meta-analysis of 52 prospective studies of participants who were healthy at baseline, found that, compared to tall individuals (stature >173.9 cm), the mean risk ratio of short individuals (stature <160.5 cm) for all cause mortality was 1.35 (95% CI= 1.25-1.44), for CVD mortality was 1.55 (1.37-1.74) and for coronary heart disease was 1.49 (1.33-1.67) (Paajanen et al. 2010). However it does appear as though the relationship between CVD and stature is not universal, likely due to differences in the nutrition transition stage of populations studied. A study on short stature and CVD in Bangladeshi women failed to find a significant relationship (Hosegood and Campbell 2003). These women were very short, with half of the women being below 148 cm. The high level of adult stunting indicates that the women were raised in an environment that had not yet entered the degenerative disease stage. Before the degenerative disease stage is entered, CVD is not a large health concern, with the majority of morbidity and mortality occurring due to infectious diseases (Popkin 1996).

This thesis will refer to adult short stature as stunting because short stature is caused by the same factors as childhood stunting and is, in fact, the end result of childhood stunting. Also using the same terminology for both mothers and children makes the discussion more succinct. This terminology will be used even though it is acknowledged that the mothers are no longer growing and therefore are not currently experiencing linear growth stunting. Thus the use of the term stunting in adults refers to prior stunting.

It must be noted that the health concerns of adult female stunting do not end with her own wellbeing, but may extend to her offspring. For example, children born to stunted women are more likely to die in infancy (Ozaltin et al. 2010). A stunted

woman has a smaller uterus, restricting her foetus's intrauterine growth, increasing the risk of infant mortality (Goldenberg et al. 2008). Also, a woman who experienced chronic under-nutrition is likely to have fewer metabolic reserves than a well-nourished woman (Wells 2010). Such an intergenerational legacy of under-nutrition may be a health concern for several generations (Kuzawa 2005; Wells 2010).

1. *Urbanisation and transition*

Developing countries are undergoing rapid urbanisation. There is a well documented rural to urban gradient in stature in developing countries, with urban areas having lower stunting rates than rural areas (Coly et al. 2006; Mukuddem-Petersen and Kruger 2004; Oyhenart et al. 2008). Overall it appears as though specific SES factors that are concentrated in urban areas are the cause of this rural to urban stature gradient, such as improved access and utilisation of services and sanitation (Oyhenart et al. 2008; Smith et al. 2005; Van de Poel et al. 2007). Also, behaviour changes with migration from a rural to an urban environment could influence this association. For example, urban dwellers have less traditional eating habits than their rural peers (Torun et al. 2002). Yet, after controlling for SES factors, the rural to urban gradient in stature is greatly decreased (Van de Poel et al. 2007). There actually appears to be an increase in urban stunting rates, which narrows the gap between rural and urban health (Fotso 2007). In some countries, the prevalence of stunting is actually highest amongst the urban poor (van de Poel et al 2007). For instance, when measuring 4-6 year old children in the south of Merida, Mexico, no difference was found in the growth status of migrant children from rural areas and children who had been born and raised in Merida (Azcorra et al. 2009).

2. *SES*

SES has been known to influence growth since the 18th and 19th centuries, when it was shown that low SES children were shorter than high SES children (Bogin 1999). Stature is still linked with SES in almost every country (Malina et al 2009, Siniarska and Wolanski 2005, van de Poel et al 2007), with stunting in under-5 year-olds being most common in low SES groups (van de Poel et al 2007, Zottarelli et al 2007). SES has been shown to impact growth in infancy and childhood (Gunnell

et al. 1998b; Lasker and Mascie-Taylor 1989; Webb et al. 2008), the time at which stunting is most likely to occur (Stein et al 2010).

A range of SES factors are associated with linear growth. For example, poverty (Van de Poel et al. 2008), maternal education (Jehn and Brewis 2009; Jones et al. 2008; Zottarelli et al. 2007), household sanitation, ownership of an indoor flush toilet and material possessions, such as televisions (Jones et al. 2008), have been shown to predict stunting. Other demographic factors such as family size (Jehn and Brewis 2009), subsistence agriculture (*ibid*) and living in a rural area (Zottarelli et al. 2007) are also linked.

Under-nutrition resulting from low-SES appears to be a perpetuating cycle that is difficult to break. Chronic under-nutrition is associated with reduced cognitive function in children (Maluccio et al. 2009), reduced educational attainment (Alderman et al. 2006; Victora et al. 2008) and economic earnings in adulthood (Case and Paxson 2008; Victora et al. 2008). All of these diminish an individual's ability to adequately provide for his or her family, placing the next generation at risk.

3. Diet

Diet has been recognised as a factor influencing growth since at least the Renaissance (Tanner 1981). Macro-nutrients, primarily calories and protein, have a large impact on chronic nutritional status and linear growth (Dumortier et al. 2007; Kinra et al. 2011; Rutishau and Whitehead 1972; Walker et al. 1991).

One of the best studies to show that macro-nutrients impact growth in humans is the Institute of Nutrition of Central America and Panama (INCAP) study. The INCAP longitudinal study examined the impact of a protein supplementation programme on pregnant and nursing mothers and children up to 7 years of age (Habicht et al. 1995a). The study lasted from 1966 to 1977 in four Guatemalan villages. Participants were given free, daily access to either a high protein-fortified drink, called *atole*, or a placebo, low calorie, protein-free drink, called *fresco*. Two villages received the *atole* and two received the *fresco*. A follow-up study was conducted in 1988-1989 to ascertain any lasting effects on the children, then aged 11 to 27 years, who were enrolled in the original study (Martorell 1995). The early-life supplementation had life-long effects on growth. The individuals in the supplemented villages were taller and heavier with more fat-free mass as adolescents and young adults than those in the *fresco* villages (Rivera et al. 1995). These growth differences

were already present at age 3. The *fresco* individuals grew more in adolescence, lessening the differences seen at age 3, though did not completely catch-up to their supplemented peers (Habicht et al. 1995b). Supplementation also appeared to positively influence measures such as work capacity (Haas et al. 1995) and intellectual performance (Maluccio et al. 2009; Pollitt et al. 1995). From the INCAP work, it is clear that protein and energy supplementation can positively impact the growth and development of children in developing countries.

While obviously influential for growth, our understanding of the role of dietary intake is limited by the fact that dietary data are notoriously difficult to accurately collect (Willett 1998). Collecting high quality data is laborious and time consuming for both the participant and the researcher. Obtaining accurate recalls for dietary intake is difficult (Dwyer et al. 1989; Wilkens et al. 1992; Willett et al. 1988). Another problem associated with dietary data is portion sizes. Humans are not able to accurately determine the amount of food they eat and the perception of portion size is affected by food presentation and circumstance. The multitude of difficulties associated with dietary data influence many researchers to focus instead on measures of nutritional status such as anthropometry, rather than diet. Nutritional status is less precise, as it is influenced by factors other than diet, but it provides an accurate and fairly easy to collect measure (Hoffman et al. 2006).

4. *Energy expenditure*

Chronic under-nutrition leading to linear growth retardation appears to be related to alterations in energy expenditure. Stunted children have been found to have lower absolute total energy expenditure than their non-stunted peers but higher total energy expenditure when corrected for body size (Hoffman et al. 2000b; Soares-Wynter and Walker 1996; Wren et al. 1997). It is likely that this relationship is due to the maturation of organs during growth. Infants have a very high metabolic rate for their body size, while adults are more efficient and have a much lower metabolic rate for body size (Butte et al. 1995; Weinsier et al. 1992). Stunted children have been shown to have higher resting metabolic rate per kilogram of body mass (RMR/kg) than their non-stunted, age peers and have lower RMR/kg than height-matched, younger children (Soares-Wynter and Walker 1996). This indicates that stunted children are developmentally delayed in terms of metabolic tissue, perhaps due to a decreased amount of metabolic tissue (primarily muscle and brain).

This difference in RMR/kg appears to continue throughout childhood and into adolescence (Grillo et al. 2005).

When types of under-nutrition (acute and chronic) are grouped together, undernourished children have been shown to have decreased total energy expenditure and activity energy expenditure (Spurr and Reina 1988; Spurr et al. 1986). However when stunted children are specifically examined (rather than stunted and acutely under-nourished children grouped together), their activity energy expenditure tends to be similar to non-stunted children (Hoffman et al. 2000b; Wren et al. 1997). This indicates that stunted children may have similar metabolic reserves for activity energy expenditure as non-stunted children. Furthermore, any influence of stunting on activity levels may be over-ridden by other influences, such as the influence of gender. Boys tend to be significantly more active than girls (Raitakari et al. 1994; Troiano et al. 2008), and the influence of gender on activity levels has been shown to be larger than the influence of stunting (Hoffman et al. 2000b). It appears as though this influence of activity may translate to physical fitness as stunted children have been shown to perform just as well as non-stunted children on measures of aerobic fitness (Malina et al. 2011b). Overall, there are few studies on objectively measured energy expenditure in stunted children and the relationship between stunting and energy expenditure is still unclear.

G. *Over-nutrition*

Over-nutrition is currently recognized as one of the largest health concerns of the 21st century (WHO 2006). The prevalence of OW/OB has been increasing since the 1980s and is currently considered a worldwide pandemic (WHO 2006; WHO 2009). For example, the prevalence of obesity (BMI>30.00) (WHO 2006) in adult Mexican women was 10% in 1987 (Martorell et al. 1998) and by 2006, had increased to 35% (Olaiz et al. 2006), a trend that is also seen in Mexican women living in poverty (Neufeld et al. 2008). The OW/OB prevalence of Mexican children and adolescents was over one third in 1998-9 (Salazar-Martinez et al. 2006), similar to the United States (Ogden et al. 2010; Ogden et al. 2006).

OW/OB is positively correlated with the risk of chronic disease and mortality (WHO 2009). Adipose tissue, particularly the visceral adipose tissue surrounding the internal organs, alters the body's metabolism (Turgeon et al. 2006) and increases the risk of developing NR-NCDs (Lau et al. 2005; Paeratakul et al. 2002). NR-NCDs

such as CVD and type II diabetes are more common in later adulthood than in childhood (Lau et al. 2005). Yet, risk factors for NR-NCDs such as CVD and type II diabetes are increasingly being seen in OW/OB children (Lobstein and Jackson-Leach 2006; Singha et al. 2004). Treatment for NR-NCDs can be difficult in developing countries where the burden of infectious disease remains high and where there is a lack of quality public health care (Jamison and Mosley 1991; Yach et al. 2004). NR-NCDs can also have substantial impacts on a country's economy due to work absenteeism and decreased productivity (Colditz 1999; Wolf and Colditz 1998; Yach et al. 2006).

There appears to be a lifelong legacy of poor health outcomes for an overweight child. Individuals who are OW/OB in childhood are more likely to be OW/OB in adulthood than their peers who were not OW/OB as children (Guo et al. 1997; Serdula et al. 1993). OW/OB children also appear to be more likely to have CVD in adulthood, regardless of adult weight status (Freedman et al. 2001; Gunnell et al. 1998a; Wang et al. 2008). Heavier children have been shown to have higher all-cause and CVD related mortality in adulthood, after correcting for adult body mass index (BMI) (Gunnell et al. 1998a; Reilly and Kelly 2011). Preventing childhood OW/OB is a useful tactic in lowering adult OW/OB rates and associated morbidity (Fowler-Brown and Kahwati 2004; Freedman et al. 2001).

The obesity epidemic in developed countries is thought to be caused by a combination of decreased physical activity (reduced activity energy expenditure) and a wide-spread adoption of a diet high in refined carbohydrates and fat (Fowler-Brown and Kahwati 2004; Wang and Zhang 2006), part of the epidemiologic transition (Popkin and Gordon-Larsen 2004; Popkin 1996). This combination of increasing energy availability and decreasing energy expenditure leads to excess adipose deposition, and obesity. Many developing countries are also adopting diets and behaviours that increase the risk of OW/OB. Yet, developing countries are not following the same transitional path as developed countries, in part due to the speed of the transitions in developing countries. Therefore, the causes of OW/OB in developing countries are less well understood.

Other factors, such as genetics (Clement et al. 1995; Ristow et al. 1998) have been shown to increase susceptibility to OW/OB in a minority of cases. Such factors are less modifiable and therefore are less of a public health concern for countries

undergoing epidemiological transition than societal shifts and will not be discussed further in this review.

1. *Urbanisation and over-nutrition*

Urbanisation is one of the societal shifts implicated in the rise in OW/OB. Just as a rural-to-urban gradient in stunting exists, there is a gradient in over-nutrition. The lowest prevalence of obesity tends to be in rural areas, and the highest in urban centres (Aekplakorn et al. 2007; Beltaifa et al. 2009; Mendez et al. 2005; Salazar-Martinez et al. 2006). Mexican women (Mendez et al. 2005) and children (Salazar-Martinez et al. 2006) are more likely to be OW/OB in urban areas than rural areas. However the difference between rural and urban OW/OB prevalence is lessening in Mexico (Fernald et al. 2004).

Similar to the rural-to-urban gradient in stunting, there is strong evidence that it is the difference in environment that is the key to the differences in OW/OB (Azcorra et al. 2009; Beltaifa et al. 2009; Garnier et al. 2003; Jorgensen et al. 2006; Unwin et al. 2006). Migrants tend to adopt the behaviours of life-long urban dwellers (Chattopadhyay et al. 2006; Jorgensen et al. 2006). Several studies have shown that rural to urban migrants are heavier than their rural peers and have a similar weight status to lifelong urban dwellers (Beltaifa et al. 2009; Bogin and MacVean 1981; Garnier et al. 2003). A study previously conducted in the south of Merida, the location for the fieldwork for this thesis, found that the weight status of 4-6 year-old children who were born in rural areas and who migrated to the city and life-long urban dwelling children were the same (Azcorra et al. 2009). Therefore it appears as though the birthplace of low SES children in the south of Mexico is less important than their current residence, when the migration occurs at a young age.

2. *SES and over-nutrition*

The relationship between SES and OW/OB is strong. For the most part, wealthier nations have higher rates of OW/OB than low-income nations and within developing countries those of higher SES tend to be more likely to be OW/OB, especially in the earlier stages of transition (Kelishadi 2007; Mendez et al. 2005). This trend reverses as transition progresses and high income countries have the

opposite trend, with the low SES groups having the highest risk of OW/OB (Amuna and Zotor 2008; Mendez et al. 2005; Monteiro et al. 2004; Prentice 2006).

The relationship between OW/OB and SES in Mexico is complex. When examining the entire population, Mexico follows the trends of the high income countries, as there is an inverse relationship between SES and OW/OB prevalence (Monteiro et al. 2004; Neufeld et al. 2008). However the relationship reverses when examining specific SES groups, particularly the low SES groups (Fernald 2007). Among the low SES groups, SES is positively related to OW/OB. The positive relationship between SES and OW/OB is more similar to low-income countries. The high SES groups can be considered at a later stage of the nutrition transition than the low SES groups.

Individual SES has been shown to be related to OW/OB and associated with CVD risk factors in populations undergoing nutrition transition (Bjerregaard and Dahl-Petersen 2011), including Mexico. In a sample of very low-income Mexican adults, relatively higher SES (educational attainment, housing conditions and consumer durable ownership) and sugar sweetened beverage consumption were found to be positively related to BMI (Fernald 2007). Individual wealth quintiles were positively associated with OW/OB rates in urban Cameroon adults (Fezeu et al. 2006). In South Africa SES and fat mass also had a positive relationship in 9/10 year old urban children (Griffiths et al. 2008). SES is related to other factors that influence over-nutrition, such as diet.

3. *Diet and over-nutrition*

Dietary changes, such as an increase in food availability (Sheehy and Sharma 2010) and altered dietary composition (Zhai et al. 2009), are inherent in the nutrition transition (Popkin 1996). As previously described, the increase in food availability tends to occur through an increase in calorie-dense, nutrient-poor foods as purchased and processed foods become more widely available in transitioning societies. In high income countries such as the U.S., there is a clear inverse relationship between the energy density and cost of food (Beydoun et al. 2008; Darmon et al. 2002; Drewnowski 2004). Within populations that rely on purchased foods, the lower SES groups tend to make dietary choices and purchases based upon price more than the higher SES groups (Darmon and Drewnowski 2008). Fruit and vegetables in urban areas of Latin America are often more expensive than rural

areas (Uauy et al. 2001). In high income countries, the low SES neighbourhoods have been labelled 'food deserts' as they are deprived of healthy foods, with only processed, calorie-dense, nutrient-poor foods available (Drewnowski and Specter 2004). Living in such areas is associated with an increase in BMI and adiposity while neighbourhoods with a high availability of health foods have been associated with decreased BMI (Casagrande et al. 2011). These 'food deserts' have currently only been documented in high income countries (Cummins and Macintyre 2002; Drewnowski 2004) but during epidemiologic transition, most developing countries adopt many behavioural patterns from higher income countries (Popkin 1993). As Mexico is an upper, middle-income country (WorldBank 2010) that is undergoing rapid transition (Stevens et al. 2008), it is very close to having behavioural patterns that mimic high income countries (Rivera et al. 2002; Rivera et al. 2004). Therefore, the low SES populations in Mexico are at risk for a further increase in their OW/OB prevalence.

Increased reliance on purchased foods leads to a change in dietary composition (Zhai et al. 2009) with a higher consumption of sugar (Popkin 1999) and fat and lower consumption of fibre (Walker et al. 2001) compared to a traditional diet. Purchased foods, which are often processed, calorie-dense and nutrient-poor, are often highly palatable, with added sugar and fats, and are associated with higher energy intakes in laboratory settings (Drewnowski 2004). Foods consumed in urban schools in Mexico City have been shown to be on average calorie-rich and nutrient-poor (Lozada et al. 2008). There appears to be a gradient in the dietary quality within Mexican schools with higher SES students consuming a higher quality diet and the lower SES students consuming more calorie-rich nutrient-poor foods. This was due primarily to the high SES students bringing higher quality lunches from home (*ibid*). Dietary quality is also linked with CVD risk, independently of OW/OB status (Colin-Ramirez et al. 2009).

4. Energy expenditure and over-nutrition

A decrease in energy expenditure is also strongly implicated in the rise of obesity. Behavioural changes that occur as part of economic and social transitions often result in altered physical activity patterns. Economic shifts away from physically demanding agriculture toward more mechanised or service orientated industries leads to a decrease in occupational physical activity (Popkin 1993). This is seen in a

difference in activity levels between urban and rural areas, with residents in urban areas having significantly lower activity levels (Assah et al. 2011; Monda et al. 2007; Ng et al. 2009; Yadav and Krishnan 2008; Yamamoto-Kimura et al. 2006). An analysis of Chinese data suggests that over half of the high levels of urban obesity can be explained by lower physical activity levels compared to rural areas (Van de Poel et al. 2009). In Mexico there may also be an SES effect on physical activity, but the existing studies are scarce and tend to rely on subjective measurements. Higher SES students have been shown to exhibit higher levels of physical inactivity than lower SES students, increasing the risk of OW/OB in high SES children (Yamamoto-Kimura et al. 2006).

H. *Nutritional double burden*

With the nutrition transition, the disease profile shifts from one dominated by diseases related to under-nutrition and infection toward a profile dominated by diseases related to over-nutrition (Popkin et al. 1993). However, as is implied from the discussions on over- and under-nutrition, the decrease in under-nutrition does not occur at the same rate as the increase in over-nutrition. Both under- and over-nutrition occur simultaneously in many countries and populations, a phenomenon known as double burden (WHO 2006). Double burden can refer to the co-existence of under- and over-nutrition of any type (Delise 2008). However in this thesis, double burden will only be used to discuss chronic under-nutrition (stunting) and acute over-nutrition (OW/OB) because this is the type of double burden most relevant to the Maya population being studied.

1. *Levels of double burden*

Double burden can exist at the population, household and individual level. A population is typically considered to be double burdened if the prevalence of stunting and OW/OB is above the prevalence expected for a healthy population. The expected prevalence of short stature in a healthy population is roughly 5% (Frisancho 2008) while 15% of a healthy population is expected to be OW/OB (WHO 2006).

Households can be considered double burdened if one member is OW/OB and one is stunted. Double burdened households occur most frequently with an

OW/OB adult woman and a stunted child (Garrett and Ruel 2005). These households are typically urban in middle-income countries (Doak et al. 2005; WHO 2009) undergoing nutrition transition (Doak et al. 2005), such as Mexico. A study including Mexican mother-child dyads found that 24% of indigenous families experienced simultaneous maternal central adiposity and childhood stunting (Barbeau et al. 2007). Florencio et al. (2001) found that 30% of the very low-income households in urban Brazil were double burdened.

A person can be double burdened, being both stunted and OW/OB. Individual double burden occurs most commonly in adults who were chronically under-nourished during their growing years and experienced over-nutrition as adults. Maya children are more commonly stunted than other Amerindians (Crooks 1994) and ethnic groups in the Yucatan (Jenkins 1981) placing them at a high risk of OW/OB in later life due to the epidemiological and nutrition transition currently underway in Mexico (Rivera et al. 2002). Stunted adults are at very high risk for OW/OB. There is evidence that stunting in childhood is associated with metabolic shifts that predispose individuals to later life OW/OB. Stunted adults in a very low-income population in urban Brazil were more likely to be OW/OB (30%) compared to their non-stunted peers (23%) (Florencio et al. 2003). Calories consumed were not able to explain this difference in OW/OB prevalence. In fact, the stunted individuals appeared to consume fewer calories than expected based upon predicted calorie intake that had been adjusted for short stature. Short native Siberian adults have also been shown to be fatter and heavier than their peers, and have higher serum lipid levels (Leonard et al. 2009). This may be partially explained by the apparently permanent metabolic shift associated with chronic under-nutrition during growing years. Fasting fat oxidation has also been shown to be lower and serum lipid levels higher in short women (*ibid*).

Similar metabolic shifts and altered body compositions have been found in children as well. South African children who were stunted at 2 years of age were shorter and lighter but had equal BMIs in late childhood, 7 to 9 years of age, than their non-stunted peers (Cameron et al. 2005b). Stunted children in Brazil have been shown to have energy expenditure equal to non-stunted, weight-for-height matched peers at age 7-11 years (Hoffman et al. 2000b). These stunted children also had lower fat oxidation than their non-stunted peers (Hoffman et al. 2000c) and impaired regulation of energy intake (Hoffman et al. 2000a). In a follow-up study, 36 months

after the initial study, the stunted children gained more weight and less lean mass than their peers (Grillo et al. 2005) and had a greater percentage body fat (%BF) (Martins et al. 2004).

2. Causes and implications of double burden

Dietary failings are seen as the cause of double burden (WHO 2006). A theme of double burden is that it can occur in groups that apparently have adequate access to calories but there is a problem with either the distribution or nutritional content of the food (Garrett and Ruel 2005, WHO 2006). Both under-nutrition and over-nutrition tend to be concentrated in low SES groups and households, the groups with the least education and most restricted food choices. Thus the challenges associated with addressing the causes of both under- and over-nutrition are numerous. In groups with limited access to healthy foods and who are food insecure, the logistical difficulties related to performing an effective dietary and lifestyle intervention are magnified.

There are currently insufficient data to determine what the disease burden from NR-NCDs will be in populations currently undergoing rapid transition. Cross-sectional studies find that stunted adults are more likely to be overweight, while longitudinal findings are mixed (Benefice et al. 1999, Cameron et al. 2005, Gigante et al. 2007, Martins et al. 2004, Martorell et al. 2001, Schroeder et al. 1999, Walker et al. 2002). Gigante *et al.* (2007) found a protective effect of stunting at 2 and 4 years of age on fat mass and BMI at 18 years of age in Brazilian men. Longitudinal studies are lacking in developing countries. Much of the current evidence comes from developed countries, primarily the United States and Scandinavia (Reilly and Kelly 2011). Due to the differences in the development and transition between such countries and the currently developing nations (Popkin et al. 2002), it is not possible to determine the magnitude of the increased risk of obesity related mortality and morbidity in currently developing nations. It is reasonable to assume that the disease burden will be higher in populations currently undergoing rapid modernisation due to double burden.

Having described the importance of nutritional status in the context of transition and SES, the next section will discuss the methods of assessing and estimating nutritional status.

I. *Measurement of health risks*

There are many different ways to measure health risk from diseases related to lifestyle including health seeking behaviour, CVD risk factors (e.g. physical activity levels and dietary composition) and various measures of the environment including SES. For brevity, this thesis will primarily discuss those that were included in the data collection for this thesis.

1. *Chronic nutritional status*

The assessment of chronic nutritional status is typically performed using stature as a measure of linear growth. As previously described, stature has been shown to be very useful as an objective measure of chronic nutritional status, see section D. Populations with chronic stress and sub-optimal nutrition are shorter than those without as many environmental constraints. Under very poor conditions, high rates of stunting occur.

a. *Body proportions*

Despite the wide use of stature, it may be more appropriate to use body proportions, such as the proportion of leg length to stature, rather than overall stature to obtain a more detailed objective assessment of chronic nutritional status during the growing years. During the foetal period, growth follows a cephalocaudal gradient, with the head growing the most rapidly and the lower limbs the least rapidly (Leitch 1951). At birth, the head accounts for a fourth of the body's total length, while in adulthood it accounts for only an eighth (Figure 2.1). The legs account for three-eighths of the body's total length at birth and half of an adult's total height. In infancy and childhood, prior to adolescence, the lower limbs grow the most rapidly. Any chronic insult during childhood will disrupt the supply of energy to the growing section and may result in a permanently small body section. Since the legs are the fastest growing body segment prior to adolescence, leg length may theoretically act as a sensitive indicator to chronic nutritional status during these years (Bogin and Varela-Silva 2010; Leitch 1951).

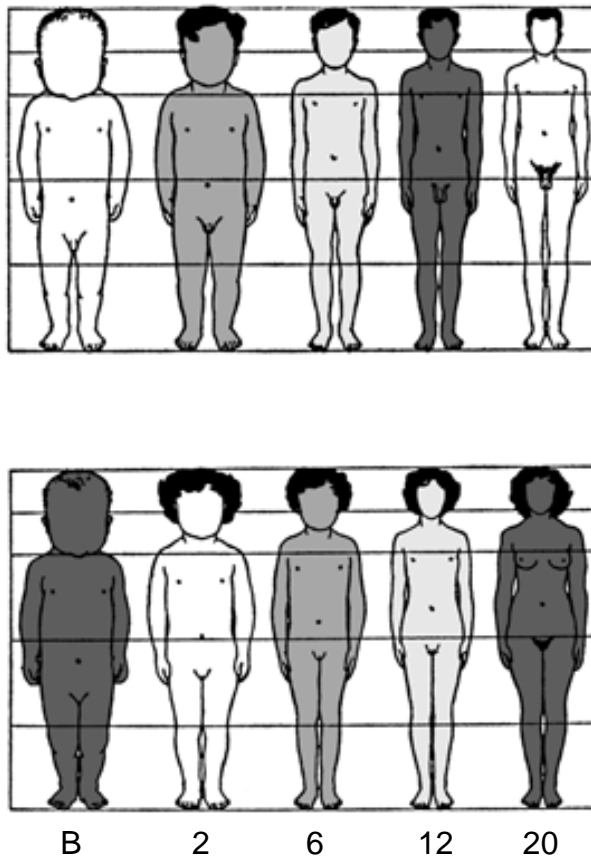


Figure 2.2 Changes in body proportions throughout the growing years.
From Bogin 1999.

There is conflicting longitudinal evidence on the impact of childhood environmental conditions on adult leg length. In the Carnegie (Boyd Orr) cohort born in Britain in 1937-39, childhood SES and diet were found to predict adult stature. When divided into leg and trunk lengths, leg length was more strongly related to childhood conditions than trunk length (Gunnell et al. 1998b). Another longitudinal study in India failed to find an impact of a three-year protein supplementation during pregnancy and in the first six years of life on leg length in adolescence (Kinra et al. 2011). However this intervention study is difficult to interpret as the supplemented individuals are still growing. Also, in this analysis they combine all individuals who were supplemented and failed to separate individuals supplemented *in utero* and infancy from those only supplemented in childhood. Leg length has been shown to not be influenced by *in utero* environment (Gunnell et al. 1999). Therefore, the analysis by Kinra and colleagues is inconclusive with respect to the impact of supplementation on linear growth.

The evidence from longitudinal data and the strong theoretical basis make a strong argument for the use of relative leg length as a proxy for chronic nutritional status and health. Secular increases in stature have been shown to be due to an increase in leg length. Japan experienced a secular increase in stature between 1957 and 1977, almost solely due to an increase in leg length (Tanner et al. 1982). The same has been found in Mayans. Bogin *et al.* (2002) measured Maya children, 5-12 years of age, in both Guatemala and the U.S. The Maya-American children were taller by 11.5cm than their Guatemalan peers, with a majority of the difference, 6.8cm, due to an increase in relative leg length. A cross-sectional study using the NHANES III data found that relative leg length was linked to the poverty income ratio in Mexican-Americans (Frisancho et al. 2001).

However cross-sectional studies in developing countries have failed to consistently replicate the findings using retrospective measures of early life environment (the approach that is used in this PhD research). In Taiwanese individuals, relative leg length has been associated with linear enamel hypoplasia, a marker of growth disruption, (Floyd 2007; Floyd 2008; Floyd 2009). Studies of recall of early life conditions have weak relationships with leg length. In a study of Central and Eastern European adults, recall of their childhood conditions was significantly associated with measures of linear growth, primarily stature and weakly with leg length (Webb et al 2007). A study in China with adult women found that leg length varied with parental literacy but not measures of childhood material wealth (Schooling et al. 2008b).

This lack of consistent findings may be associated with the difficulties of retrospectively measuring early life factors in developing countries. Therefore, current studies attempting to use relative leg length must ensure that retrospective early life measures are appropriate to the population and sensitive to local SES differences.

2. Acute over-nutrition

BMI is typically used to assess OW/OB at the population level (Popkin and Doak 1998). BMI was developed in populations of European descent and has been applied to many other populations without complete testing of appropriateness. BMI has been widely adopted because it is very easy to measure, requires minimal

training, and uses only height and weight measurements (Ellis 2001). Within developed populations of European ancestry and developing populations of non-European ancestry, BMI correlates with total body adiposity (Ellis 2001) and body composition (Cameron et al. 2009). BMI is particularly useful for measurements of adults as changes in BMI in this age group may be considered to be mostly due to body fat (Ellis 2001) since stature in adulthood remains fairly stable (Bogin 1999). The BMI cut-offs for adult OW/OB are constant: 25 for overweight and 30 for obesity for most populations (WHO 2006). The use of BMI in children is complicated by the changes in body composition that occur during growth and relies on appropriate growth reference curves to be available to define over-nutrition (Frisancho 2008).

BMI is limited by its lack of ability to differentiate between tissue types (Ellis 2001). Therefore an individual with high lean mass and low adiposity may be considered OW/OB while an individual with high adiposity but low lean mass may be considered 'healthy' in terms of BMI. Since CVD risk factors are highly dependent upon body composition and even the location of adipose tissue, it is beneficial to have more specific measures of OW/OB than BMI alone.

Another factor that must be considered when using BMI is body proportions. The legs account for a lower proportion of total body mass than the torso (Bogin and Beydoun 2007). Individuals of the same stature and body composition with differing torso lengths may have differing BMIs, with relatively long legged individuals having lower BMIs. A simple linear regression of SHR onto BMI found a significant and positive relationship between the two in a sample of adults from the United States (Bogin and Beydoun 2007). The long legged Australian Aboriginals have been found to have low BMIs, while skinfold measures did not find low levels of adiposity in this group (Norgan and Jones 1995). By standardising BMI for SHR, the prevalence of low BMIs was reduced from 30% to 7% in this study, bringing it closer to that estimated by the skinfolds. Correcting for SHR was also found to be useful in a sample of Chinese adults (Deurenberg et al. 1999). Applying BMI requires careful examination of the appropriateness of such a measure for the context and the age group for which it is going to be used to define OW/OB.

3. *Energy expenditure*

The measurement or accurate estimation of energy expenditure is vital to understanding the energy balance of populations, particularly those at risk for OW/OB. However accurately measuring energy expenditure, particularly in free-living populations, is very difficult and expensive, particularly in children (Westerterp 2009a). In order to circumvent the logistical difficulties associated with measuring energy expenditure, often physical activity as estimated by movement counts is measured as a proxy (Ward et al. 2005; Westerterp 2009b). However measuring movement results in under-estimations due to device limitations. The devices cannot detect certain types of activity or distinguish intensity levels of all activities (Rowlands and Eston 2007). For example, pedometers can only be used to assess walking and running activities and accelerometers are unable to distinguish between walking while carrying water or heavy loads from walking without a heavy load.

These are well known limitations within the energy expenditure and physical activity fields. In an attempt to circumvent these logistical constraints, new devices are being developed that combine accelerometry with physiological measures such as heart rate or skin temperature (Arvidsson et al. 2009; Corder et al. 2007; Strath et al. 2001). Including physiological measures allows for the intensity of the activity to be assessed, allowing for energy expenditure to be more accurately estimated. While these devices and their analysis software are still undergoing development, they represent a feasible estimate of energy expenditure in free-living populations. Details on the device used to estimate energy expenditure in the main study, the Actiheart, can be found in Chapter 3 section D.3 and a published journal article on the logistical challenges of the device is included in Appendix E.

Collecting data from children requires researchers to overcome all of the logistical issues associated with gathering free-living data (McClain and Tudor-Locke 2009). Children are less likely to understand why they must wear a device, particularly when the monitoring period is over several days, and become non-compliant. They are also less likely to notice if the monitoring device becomes damaged or falls off. In order to gain high quality data from children, their caretakers or parents must be willing to help the researchers and ensure the monitoring device is being worn and remains undamaged.

4. **SES environment**

SES is complicated by the intricate relationship between a family or individual's income and expenditure (Filmer and Pritchett 2001). In many developing country contexts as permanent employment is rare and accurate reporting of income is difficult, proxies for income are typically used, such as consumer durable ownership and occupation (Filmer and Pritchett 2001; Pollitt et al. 2005). Measures of SES that act directly on health, such as sanitation facilities and maternal education are often measured as well (*ibid*). There is also increasing evidence that community level SES impacts health (Drewnowski et al. 2004; Smith et al. 2003). It is best to apply community level SES to the smallest possible geographic area due to the heterogeneity of individual SES (Soobader et al. 2001).

The increasing evidence for DOHaD (Baker et al. 2009; Dietz 1994; Drake and Walker 2004; Gluckman et al. 2008; Leonard et al. 2009; Parsons et al. 2001; Sharrock et al. 2008), highlights the necessity for precise and accurate assessments of early life conditions. The gold standard would be to obtain this information through longitudinal cohorts, with the relevant data being collected in childhood or earlier and applied to conditions in adulthood (Martin et al. 2005; Martorell et al. 1995; Terry et al. 2009). However longitudinal cohorts are logistically challenging, expensive and take decades (Tanner 1981; Terry et al. 2009). For these reasons, the majority have occurred in developed nations. Due to the differences between developed and developing populations (Popkin 2002), the findings of longitudinal studies in developed countries may not be applicable to currently developing populations. Using large scale, economic data are useful (Sheehy and Sharma 2010) however such types of data ignore heterogeneity in living conditions and may not accurately reflect the experiences of the populations and families (Komlos 1985; Soobader et al. 2001). Also using simple economic measures ignores the social aspect of SES, which have been shown to impact health and growth (Aquino et al. 2009; Heckman 2008; Russell 1976).

It is therefore often desirable to obtain an accurate measure of an individual's childhood after childhood has been experienced. One such measure that is used frequently is linear growth or stature (Bogin and Keep 1999; Komlos 1985; Steckel 2009) and less frequently, relative leg length (Leitch 1951). It would be advantageous if recall of childhood conditions could be used as a measure of actual

childhood conditions. While there are many known flaws in human's long term memory (Dwyer et al. 1989; Fuhrer et al. 1999; Schmolck et al. 2000b), it is logical to assume that important factors such as, whether parents were married and what type of toilet facilities the family had, might be well remembered.

J. Summary

The review of the literature has highlighted the need for further investigation into the relationship between nutrition, epidemiological and behavioural transitions and health outcomes, specifically in developing countries. The impacts of these transitions are determined by many factors such as family SES, community SES, diet, behaviours, employment, schooling, *etc.* It is clear that as a population transitions from the receding famine toward the degenerative diseases stage, rates of OW/OB rise dramatically. Poor early life environments are related to an increased risk of obesity and NR-NCDs, increasing the risk to transitioning populations. The nutrition transition is currently occurring very rapidly and as such is exposing many populations to poor environments during early life and caloric abundance in adulthood. This leads to widespread OW/OB and simultaneously high levels of chronic under-nutrition (stunting). A double burden of over- and under-nutrition is particularly common in middle-income countries, such as Mexico, particularly in low SES, urban groups.

In light of this information, the following aims will be addressed in this thesis:

1. To describe the living conditions and population characteristics of the urban Maya of southern Merida in the spring and summer of 2010.
2. To determine whether body mass index (BMI) predicts adiposity indicators in a sample of women and children with a high prevalence of stunting.
3. To determine whether measures of linear growth in women can be predicted by their recalled childhood environment.
4. To determine whether the relationship between estimated free-living energy expenditure and body composition is altered by stunting.

Chapter 3. *Methods*

This chapter describes the research methods used during data collection and data cleaning. The chapter also covers methods that are generally applicable to the overall thesis topic. Where relevant, methods that are specific to one results chapter are described in that chapter separately.

A. *Fieldwork*

The fieldwork took place from February to August 2010. I was in Merida from February to 20 June. Interview piloting and alterations were performed in February and March. Anthropometric training and standardisation occurred in March. The interviews and anthropometric measurements of the mothers took place from 30 March until 7 July. The anthropometry of the children and the objective measurement of their physical activity levels took place between the 30 March and 22 July. M. Ines Varela-Silva (MIV) and Paula Griffiths (PG) provided advice throughout the planning of the research, the data collection and the data analysis and interpretation.

The local field team included the Federico Dickinson (FD), Adriana Vasquez (AV), Jenice Tut (JT), Hugo Azcorra (HA), Sally Lopez (SL), Maria Louisa Avila (MLA) and IVS. FD led the investigation. AV, JT and I were the primary members of the field team. MIV was in the field for the first week of data collection and assisted with recruitment and information sessions. FD, AV, HA and I recruited schools and FD, AV and JT led the information sessions for the mothers. AV, JT and I also attempted to recruit door-to-door. My role in the recruitment was limited due to my limited Spanish skills. Piloting of the interview was performed by HA, AV and JT. In the main data collection, AV and JT performed the interviews and recorded the anthropometry while I took ethnographic field-notes and performed the anthropometry. I was the primary field worker for the physical activity monitoring. SL and MLA conducted interviews and recorded anthropometry when neither AV nor JT were available. HA assisted with the objective physical activity monitoring and children's anthropometry. Upon my departure on 20 June, AV and JT led the field work, completing the remaining interviews, anthropometry and physical activity monitoring.

B. *Participants*

1. *Sample population*

Briefly, the Maya are an indigenous group, who have been systematically marginalised and many currently experience very poor living conditions. Currently, a population of roughly 150,000 Maya are living in urban conditions in the south of Merida, the capital of Yucatan in Mexico. For this research, Maya ethnicity was defined as having two Maya surnames. Surnames have a strong link with genetics

and can be used to differentiate ethnic groups (Colantonio et al 2004). In Mexico, individuals have two surnames, one from their mother and one from their father. Maya surnames are easily identifiable as they are distinct from the surnames of individuals descended from European migrants, primarily from Spain. The population of urban Maya is described in Chapter 2, section B and the living conditions are described in detail in Chapter 4.

a. *Inclusion criteria*

This study included any Maya child aged 7-9 years old attending public school, living with their biological mother in the south of Merida. The children's mothers were also included in the study. The child and mother both had to have two Maya surnames.

2. *Ethics*

Ethical clearance was obtained from the Loughborough University Ethics Committee in the U.K. and the Bioethics Committee of Human Studies of Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional (Cinvestav) in Mexico in February 2010.

Between the submission of the ethics application and initiation of fieldwork, the project received funding from the Wenner-Gren Foundation, Grant #ICRG-93. This allowed the purchase of Actihearts to estimate the child's physical activity, instead of the originally planned, less expensive accelerometers. Also, the opportunity to test Maltron 916 bioelectric impedance equipment, manufactured and commercialised by Maltron International Ltd, became available to estimate body fat percentage. These alterations to the original ethics application were approved, by both the Bioethics Committee of Human Sciences of Cinvestav and Loughborough Ethics Committee prior to the initiation of data collection in April of 2010. Before any recruitment in the schools was done, approval from the Minister of Education of the Yucatan was also obtained.

Recruitment is explained in Section 1.B.3 of this chapter and was done through the mothers, not the children. It was clearly explained to the mothers and children that they could withdraw from the study at any time. Participant information sheets were given to both the mothers and the children. Due to non-universal literacy of the mothers, the study was fully explained orally in Spanish by the local

researchers. At this time the purpose of the study, demands on the participant's time and measures that would be taken were fully explained. Any questions about the study were answered when asked. The mother signed consent forms after the study was explained, understood and they agreed to participate. In some cases, the male head of the household signed the consent form on behalf of the child, due to a strong patriarchal tradition in the Maya culture. All children gave verbal assent prior to their participation. A recording of verbal consent was made if the participant was unable or unwilling to sign their name.

The majority of anthropometric measures took place in the home or in the school. Almost all of the anthropometric measurements of the mother were done in her home with as much privacy as possible, by a female researcher with a female recorder. One mother was measured at the school. It was stressed to the women that they could refuse to undergo a particular measurement and still participate in the study as a whole. Also, if the child was male, it was made clear to the mother and child that the anthropometric measurements could be done by a male researcher, if desired. When the measurements were done at the school, the consent of the directors was obtained and an empty classroom was used with the window shutters closed for privacy. The children measured at the school were always measured after the mothers had been interviewed and measured and given consent for their child. Therefore the mothers were able to withdraw their children from the study if they were not comfortable with any portion of the study. For the anthropometric measurements, the child and a parent (generally the mother) or school director were asked if the boys could remove their shirts and if the girls could wear a tank top and have their shirt pulled up for skinfolds and waist circumference measures. None of children and adults had an issue with this.

Each researcher always had a working mobile phone on their person when in the field. Fieldwork was done by at least two researchers, and three or more when possible. Also FD, the co-principle investigator, was aware of the day's plans before the researchers entered the field.

3. *Participant recruitment*

Participants were recruited through primary schools. In Mexico, children are legally required to attend school until 18 years of age. In practice, almost all urban children attend primary school, though attendance to secondary school is not as

universal. Researchers went to the directors of schools and explained the purpose of the study and obtained a list of the students between 7 and 9 years old. The lists were used to identify children who had two Mayan last names. Both of the surnames were included in the lists. Also included in the list was the child's date of birth and address. If the list did not include either the date of birth or the address of a child identified as Mayan, the researchers returned to the schools to obtain the missing information for the eligible children.

Once approval from the Minister of Education was obtained, the researchers used the school lists to go from house to house to recruit each mother directly. This method was unsuccessful and time consuming as many of the addresses were incomplete or incorrect and occasionally the age of the child was incorrect. Another method attempted was approaching mothers as they picked up their children from school. This was unsuccessful as well because very few of the mothers were Maya or had children in the age range. After several unproductive days in the field attempting these methods of recruitment, it was decided that information meetings at the schools would be the most efficient method of recruitment.

Maya children between the ages of 7-9 years old were identified from school lists. The directors were asked to invite the mothers of these children to an information session held at the beginning of school, after the mothers dropped the children off. Invitations were also sent home with the children. At the meetings, participant information sheets (Appendix A) were given out and the study was explained in detail by the local fieldworkers, AV, JT, FD or IVS. Any questions were answered and the information sheet was explained orally to the mothers who had difficulty reading. The interested mothers filled out contact information sheets that contained the complete name of the mother, child and father of the child, the current address, date of birth of the child and best time to contact or interview the mothers. When possible, the interview was scheduled at the meeting. If no time was scheduled, the researchers went to the houses or phoned at a later date.

Overall, the parents that came to the meetings were very responsive to participating in the study. Over 95% of the Maya parents who came to the meetings and had a child in the age range expressed an interest in participating. Of these, the majority were located and took part in the study. Over the course of three months, eight meetings were held at six schools.

For the majority of cases (54), the interview of the mother and mother's anthropometry was done in her house. The rest took place in the school (2) or the grandmother's house (2). For many of the cases, the children were also measured at this time (42/58). The rest of the children were measured at school several days after the mother had been interviewed and measured. If a measure could not be collected during the time of the interview (for example, the mother having to leave) the researchers returned to the house at a later date to collect the data.

C. Interview

1. Interview content

The mothers were interviewed about their child's health and their family's current socio-economic status. The mothers were also interviewed about their own health and living conditions during their childhood. The questions were focused on six categories: Consumer durable ownership, Economic stability, Family stability, Housing/ sanitation, Modernisation, and Demography. The questions about the family's current SES and SES during the mother's childhood were the same whenever possible. Alterations were made to the mother's childhood questions to accommodate recall error and living conditions at the time. For copies of the interview in English see Appendix B.1 and in Spanish see Appendix B.2.

The consumer durable ownership questions were based on the 1988 Mexican Demographic Health Survey (DHS) (MeasureDHS 2011) questions relating to consumer durable ownership (e.g. radio, television). For the mother's childhood, an additional ownership question related to ownership of animals for consumption or transport was included, as these were considered indicators of wealth in more traditional families but less relevant to the urban context in which the families now reside. Economic stability questions were designed by the research team in order to assess financial stability *i.e.* whether the woman perceived her childhood to include periods of financial instability (e.g. job loss of caretaker). The questions related to family stability were designed to capture the social side of SES and focused upon common problems experienced by the Maya (e.g. migration) and in all societies (e.g. family death). The housing/ sanitation questions were based upon DHS questions relating to the family's living conditions (e.g. electric lighting in the house) and

hygiene (e.g. flush toilet). Modernisation aimed at assessing the woman's exposure to non-traditional culture through place of birth (urban versus rural) and purchased food consumption associated with modern societies (e.g. sugar sweetened beverage). Demographic questions were also based upon the DHS, focusing on family size and sibling mortality. These questions included birth year and the total number of siblings, older siblings and siblings who died before they reached 5 years of age.

It must be noted that the questions for the mother's childhood and the family's current SES are the same, but they measure slightly different factors. It is more accurate to consider the recall questions to measure the mother's perception of her family's SES. Long term memory is not reliable (Dwyer et al. 1989; Hyman and Billings 1998; Schmolck et al. 2000a) and children are often unaware of all the difficulties their families' face, either through ignorance or purposeful buffering by the family. Therefore the mother's childhood SES recall questions were considered to be an indicator of the mother's perceived childhood SES rather than her actual childhood SES. The family's current SES is less subject to recall problems by the mother than her childhood SES as she will be far less buffered from and ignorant of family difficulties as an adult and mother than she was as a child. This distinction is primarily a factor in family and economic stability questions. Experience in the field suggests that other questions relating to the mothers childhood that addressed ownership of consumer durables or access to sanitation facilities are thought to be better recalled. For example, most women very easily remembered the age at which they got their first television. Therefore, while the questions for the family's current SES and the mother's childhood SES were the same, recalled SES was not interpreted as an actual measure of SES.

Another limitation of the childhood recall data was that there was no age specified for the childhood period that the mothers were being asked to recall. In order to approximate the age range these women were reporting as their childhood, the ages at which they obtain consumer durables were examined. Using this item, it became apparent that the oldest age that women reported was 15 years and this is the likely cut-off for childhood. Previous research in these Maya women (Azcorra 2007) using similar questions found a slightly earlier age cut-off, apparently corresponding to the beginning of secondary school.

2. Interview development

The interview was developed over the course of six months, from October 2009 to March 2010. The original questions were based upon DHS (MeasureDHS 2011). The DHS is widely used in developing countries and applying their questions allows for comparisons between this study and others using these standard questions. Other questions, not taken from the DHS, were included to provide information specific to individuals living in the south of Merida and, particularly, Mayans, and were based upon the long term experience of the principle investigators within Merida and the Yucatan.

The interview was discussed in several supervisor meetings between myself and my two thesis supervisors: IVS and PG. Additional input throughout the development process was provided by Professor Barry Bogin, who is a member of the Centre for Global Health and Human Development, a part of the School of Sport, Exercise and Health Sciences at Loughborough University and who has many years of research experience studying the Maya. The interview questions were also discussed at a research meeting of the Centre for Global Health and Human Development to draw upon the experience of colleagues working in other transitioning societies. After revisions were made based upon the comments made during supervisor meetings and the research meeting, the interview was sent to colleagues in Mexico for comments, revision and translation into Spanish. After my arrival in Mexico for fieldwork, several meetings were held between FD, and research assistants HA, AV, JT and myself at Cinvestav to determine the questions to be included and their wording before a piloting exercise was undertaken with the interview. At this stage the food frequency questionnaire (FFQ) was chosen (Appendix B.1), which had been developed by nutritionists at Cinvestav in Merida for use in Merida.

The pilot study of the interview was designed to assess the level to which women in the south of Merida were able to understand the questions being asked. The limited time allotted for fieldwork restricted the sample size of the pilot and precluded an assessment of the reliability or validity of the measures using a statistical examination of the pilot data.

Piloting began after the project received approval from the Minister of Education for the state of Yucatan in Mexico. Directors of primary schools in the

south of Merida were approached and asked to inform some of the mothers about the project. These mothers who agreed to take part in the preliminary interview were interviewed during the second week of March 2010. The interview was then conducted with 4 mothers who did not meet the inclusion criteria for the main study (not Maya) and had children who attended the same schools where recruitment for the main study would occur. The non-Maya mothers were considered appropriate for the pilot study as the Maya have mostly assimilated into the culture of the south of Merida. Also including Maya mothers in the pilot would have decreased the number available for the main study. These four interviews were conducted by HA (two interviews), AV (one interview) and JT (one interview) in or near the schoolyard. The participants were asked to inform the interviewer if they did not understand a question or needed further information. The participating mothers received a kitchen container as thanks for participating.

Later that day, HW, HA and AV further refined, reworded and reformatted the interview based upon feedback and impressions from the pilot interviews. The interview was then given to IVS and FD for comments. Later that week another 2 mothers living in the same community were interviewed by AV and JT using the updated interview. After the second round of piloting, the interview was again revised to clarify the wording of questions and adding questions.

Questions were added to the interview throughout the first week of interviews guided by the observations of research team and IVS and BB. These questions related to the full name of the father and the mother (to assess Maya ancestry), composition of the household, and whether a grandmother helped raise the children. These questions were added toward the end of the interview and were to inform future research.

Approximately halfway through the data collection, it was realised that the food frequency questionnaire did not measure sugar sweetened beverage or packaged food consumption; important variables for nutrition transition. The FFQ had been developed for specific use in Merida by nutritionists at Cinvestav and was therefore not examined closely by HW to ensure all the necessary variables were included. After this substantial error was realised, the researchers attempted to return to each house and ask each mother about their own and their child's sugar sweetened beverage and packaged food consumption. All but two of the mothers were able to be reached and these data were collected.

3. *Puberty assessment*

The older girls included in this study may have begun puberty (Ellison 2002). Therefore, an additional questionnaire was given to the mothers of the girls to assess the girls' pubertal status. The Pubertal Development Scale (Brooks-Gunn 1987) consists of questions regarding body hair growth, breast development, the growth spurt, skin changes and body odour. The scale was designed so that either a caretaker or the girl themselves can answer the questions. For this study, the mother's answered the questions. In these Maya families, the mothers are aware of their children's bodies, especially before and at the beginning of puberty. The houses are often very small with little privacy and no strict modesty rules exist between parents and children in the culture.

4. *Interviewer training and reliability*

A total of 4 interviewers collected data during the fieldwork. AV and JT had both conducted interviews as part of previous studies and were therefore experienced at interviewing prior to the data collection. They were familiarized with this interview during the interview piloting, with each conducting three interviews prior to the initiation of the final data collection. Researchers MLA and SL also had experience conducting interviews for previous studies. Their familiarisation with this interview consisted of observing two interviews during data collection and conducting a third with AV or JT assisting.

5. *Interview procedure*

The semi-structured interviews were conducted in the family home or the schools by a trained interviewer who was a native Yucatec-Spanish speaker. The Maya have strong gender roles and the comfort levels of the mothers are much higher around women than men. For this reason, all interviews were conducted by females, the majority by JT (31/58) and AV (23/58). The remaining interviews (4) were conducted by SL and MLA. These interviews occurred mainly on weekends when both AV and JT had other work commitments.

The interview was not completely standardized. In order to establish a rapport with the mothers, the interviewer did not read the questions verbatim, but instead asked the question in the way they felt best flowed with the conversation. The interviewers had slightly different styles of interviewing. AV was very efficient and

business-like and performed the quickest interviews. The mothers interviewed by AV tended not to volunteer extra information. JT was also quite efficient but was more approachable and the mothers tended to be more loquacious during interviews with JT. Over the course of the fieldwork, JT became more business-like. Overall, little extra, ethnographic information was collected during the interviews. However the fieldworker not performing the interview (often HW) was often able to take notes on the house and property during the interview, while waiting to do anthropometry on the mother. These observational notes form the basis of the description of the living conditions in Chapter 4.

During the FFQ portion of the interview, no time frame for the dietary intake was specified, resulting in data that was not reflective of habitual dietary intake. Due to this oversight, the dietary data are not used in any statistical analysis in this thesis. This is a very clear limitation, as nutritional status is heavily influenced by diet. Therefore, the participants' diets will be described in Chapter 4, section G but will not be used in any statistical tests.

The interviews lasted between 20 and 90 minutes, with the majority taking approximately 45 minutes.

A. Participant feedback

Some immediate feedback was giving to the participants upon completing the interview and physical assessment. Also the children's height and weight were graphed onto a clinical growth chart written in Spanish (CDC 2000). All of the children who were in the house and expressed interest in getting measured, regardless of their eligibility for inclusion in the study, were measured for their height and weight to allow individualised nutritional information to be given to the mothers. No data was recorded by the research team for ineligible individuals. All of the research team, except myself, were trained nutritionists. This allowed quality nutritional information to be given to the mothers under the guidance of trained supervisors, both in their houses and during meetings at the schools after the study.

The nutritionists were able to tell the mothers if the children were growing properly (based on height-for-age and weight-for-age). Also, nutritional advice was given. For example, children should drink milk every day, eat fruits and vegetables and eat breakfast. Also advice on general hygiene was given, such as vegetables

should be cleaned prior to consumption. Any questions the mothers had at this time were answered.

The parents were also invited to a meeting at the school after data collection had been completed to give preliminary results on the children's nutritional status and physical activity levels as well as more nutritional and hygiene advice. These meetings were done school by school and scheduled after all of the participants from each school were measured. The initial two meetings were held at different schools with only one mother attending in total. The information dissemination meetings were thus abandoned. It is likely that the time lag between the mothers being interviewed and the meeting (up to 2 months) was too long for the mothers to remain interested in the study. Future research in this population should include more extensive immediate feedback and information dissemination.

D. *Physical Measures*

1. *Anthropometry*

The technical error of measurement for anthropometry was not calculated. The research team (myself, AV and JT) received careful training both before and during the fieldwork from experienced anthropometrists (FD, BB, IVS).

a. *Measurements*

The anthropometric measures taken were body mass, stature, sitting height, knee height, waist circumference (WC), mid-upper arm circumference (MAC) and skinfolds. For the skinfolds, triceps were taken on the mothers and triceps, sub-scapular and supra-iliac were taken on the children. All anthropometric measurements were taken on the left side of the body. The procedures followed Lohman *et al.* (1988) or National Health and Nutrition Examination Survey (NHANES) (Frisancho 2008), as indicated. When possible the procedures outlined by Lohman *et al.* were followed and NHANES was used when the Lohman procedures were not appropriate for the field.

All of the anthropometric measurements were taken with a research assistant present. The measurements were said aloud by the measurer (HW, JT or AV) and repeated by the assistant as she recorded the reading to ensure the accuracy of the

recording. No mothers expressed the desire for the anthropometric measurements of their male child to be performed by a man. Therefore, all of the measurements were performed by HW, AV and JT, all females.

*i. **Body mass***

Body mass was taken on both mothers and children using the technique described by Lohman *et al.* (1988). The electronic scale was placed on a large piece of wood that was approximately 3 by 2 feet. The participant was asked to step into the middle of the scale in bare or stocking feet and look straight ahead and keep still. The reading was taken to the hundredths of kilograms (0.01 kg) when the display stabilised into a final reading.

*ii. **Stature***

Stature was taken on both the mothers and children using the technique described by Lohman *et al.* (1988) using a freestanding stadiometer. The participant stood on the piece of wood, mentioned above, which had drawings of the correct feet position. The participant stood in bare or stocking feet with their heels together and toes slightly pointed outwards. The measurer held the stadiometer behind the participant's left heel so the arm of the stadiometer would be aligned with the sagittal crest. The assistant ensured the stadiometer was vertical and aligned with the sagittal crest while standing in front of the participant. The researcher ensured that the participant's head was positioned in the Frankfort Horizontal Plane and the participant was standing straight. The participant was then asked to inhale and the arm of the stadiometer was lowered onto the participant's head. The measurement was taken at full inhalation and recorded to the nearest millimetre (0.1 cm).

*iii. **Sitting Height***

Sitting Height was taken on both the mothers and children using the technique described by Lohman *et al.* (1988). The participant sat on a table, with their feet hanging freely and their knees approximately three fingers-widths from the edge of the table. They were asked to place their hands on the tops of their thighs. The stadiometer was placed behind the participant, slightly to the left of the body to align the arm of the stadiometer with the sagittal crest. The assistant stood in front of the

participant and ensured the stadiometer was correctly aligned. The participant was asked to sit as erectly as possible, if necessary the researcher gently pushed the lower back to sit up as straight as possible. The researcher used her right hand to hold the stadiometer and steady the participants head in the Frankfort Horizontal Plane. The participant was asked to inhale deeply and at the full inhalation, the arm of the stadiometer was lowered onto the head with enough force to compress the hair. The measurement was taken at full inhalation and recorded to the nearest millimetre (0.1 cm).

iv. Knee Height

Knee height was taken on both the mothers and children using the technique described for NHANES III data collection (Frisancho 2008). The participant sat with their knee at a 90° angle and their foot on level ground. Stools of different heights were used to ensure the 90° angle. The condyles were located and the calliper arm of the stadiometer was aligned with them. The recording researcher ensured the stadiometer was straight. Then the arm was lowered onto the top of the knee and the measurement was taken to the nearest millimetre (0.1 cm).

v. Waist Circumference

Waist circumference (WC) was taken on both the mothers and children using the technique described by Lohman et al. (1988). WC was not taken on the one woman who was currently pregnant. The researcher stood in front of the participant and located the upper ridge of the iliac crest with one finger (either the middle or ring finger) and the last non-floating rib with their index finger. The assistant then marked the participant's skin at the halfway point of the two fingers by eye using a pen. The researcher stood in front of the participant and circled a tape measure around the waist at the level of the mark. The assistant was behind the participant and helped ensure the tape measure was level. The measurement was taken at normal inhalation, to the nearest millimetre (0.1 cm).

For the one mother who was uncomfortable lifting her shirt, the abdominal circumference was taken over the clothes. The navel was located and the abdomen

was encircled at that level with the assistant ensuring the tape was level. The tape measure was pulled snugly in an attempt to reduce the error introduced by clothing.

vi. *Mid-upper arm circumference*

Mid-upper arm circumference (MUAC) was taken on the mothers and the children. The participant held their arm with their elbow flexed at 90 degrees and the distance between the lateral tip of the acromion process and the acromial process with large callipers. The callipers were held at the acromion process and moved to half of the upper arm measurement and a horizontal mark was made at that point by the assistant. The researcher then wrapped a flexible measuring tape around the arm with the mark between the upper and lower sides of the tape. The assistant ensured that the tape was held horizontal and marked the triceps directly below the tape. The measurement was taken to the nearest millimetre (0.1 cm)

vii. *Skinfolds*

Triceps, subscapular and supra-iliac skinfold measurements were taken on the children using the technique described by Lohman *et al.* (1988). Triceps skinfold was taken on the mother, but not subscapular and supra-iliac in order to preserve the mother's modesty. The callipers were held in the researcher's right hand. The thumb and forefinger of the left hand was used to grasp and elevate the skin and subcutaneous fat approximately 1 cm proximal to the site of measurement. During skin elevation the thumb and index finger were approximately 8 cm apart on a perpendicular axis of the intended skinfold. The calliper jaws were then separated and the calliper was placed over the skinfold and the jaws were gently released. The researcher mentally counted to three and the measurement was taken. The jaws were then separated and the callipers were removed from the skin. Skinfolds measurements were recorded to the nearest millimetre (0.1 cm).

Triceps skinfold was taken at the mark used for the MUAC. The callipers were positioned so that the tips were perpendicularly over the mark.

The position for the subscapular skinfold was determined by finding the inferior angle of the scapula. In individuals with generous fat deposits on the back, the participant's arm was folded behind them to accentuate the shape of the scapula. The skin and subcutaneous fat was grasped at a 45 degree angle, approximately 1 cm below the most inferior point of the scapula.

The suprailiac skinfold was taken on the midaxillary line, directly superior to the iliac crest. The participants were asked to stand in the same position as for the stature measurement. The skin and subcutaneous fat were grasped following the natural fibres of the tissue, inferomedially at 45 degrees.

b. *Training and reliability*

The majority (children: 39/58, mothers: 41/58) of anthropometric measurements were done by myself. Anthropometric measurements were also taken by AV (children: 10/58, mothers: 12/58) and JT (children: 7/58, mothers: 4/58), HA (children: 1/58) and MLA (children: 1/58, mothers: 1/58). AV, HA and MLA were trained in anthropometry by FD and had been regularly taking measurements for a minimum of two years prior to the initiation of this fieldwork. I underwent anthropometric training lead by MIVS in Loughborough University in December of 2009 and also in CINVESTAV in March 2010 by FD. JT was trained by FD at the same time as I did. Reliability studies were performed before data collection began to determine whether the inter-observer variation was appropriate. Using 9 adult participants (7 women), who were not study participants, inter-observer variation was considered appropriate if it was within the technical error of measurement $\pm 5\%$. These measurements were not recorded and are thus not available.

Post hoc ANOVAs were done to examine if there were differences between anthropometric measures taken by different assessors within the main dataset. Significant differences were found in the triceps skinfolds in both the mothers and the children (Appendix C.1). The significant difference was not systematic between the mothers and children.

2. *Bioelectrical impedance analysis*

Bioelectrical impedance analysis (BIA) was used to estimate the participant's fat mass and fat free mass. BIA was taken using the Maltron BioScan 916, Maltron International Ltd. The BIA device was provided free of cost in exchange for providing Maltron with the BIA and relevant anthropometric data to assist in their construction of a predictive equation for Amerindians. Both mothers and children were measured using BIA. The readings were taken after anthropometry. The participants were asked to lie on a yoga mat without shoes, socks or metal jewellery. The participants were asked to lay still with their legs apart and their arms not touching their torsos.

Measurements were taken on the right side of the body. Electrode pads were attached to the hand between the 3rd and 4th metacarpophalangeal joints and between the distal prominences of the ulna and radius. On the foot, the electrodes were placed between the 3rd and 4th metacarpophalangeal joints and on the ankle, between the medial and lateral malleoli. The yoga mat was cleaned using alcohol wipes before being rolled up and stored between measurement locations.

In order for the readings to be precise, the participant's liquid intake and physical activity must be monitored for 12 hours prior to the test. This was not feasible for this study in a free living population.

The technical error of measurement for BIA was not calculated. As for anthropometry, the research team received careful training both before and during the fieldwork.

3. Actihearts- combined heart-rate and accelerometry

Actihearts were used to objectively and non-invasively measure the child's physical activity for one week. The Actiheart was developed in Cambridge, UK. The Actiheart is lightweight (10g) and waterproof consisting of two electrodes connected by a short lead (Figure 3.1). The electrodes are clipped to standard ECG pads.

The Actiheart has been shown to be a reliable and valid collector of accelerometry and heart rate data (Brage et al. 2005). The R^2 between the movement and acceleration data was 0.99 and the 95% limits of agreement between Actiheart heart rate data and electrocardiogram data were -4.2 to 4.3 beats per minute (*ibid*). The Actihearts have been validated for energy expenditure prediction in pediatric groups, primarily in developed countries with participants of European heritage using double labelled water (Butte et al. 2010) indirect calorimetry and other objective activity monitors (Corder et al 2005). The predicted TEE using the Actiheart was compared to double labelled water in 60, 5-18 year old, free living white, black, Hispanic and Asian children living in the United States over 7 days (Butte et al 2010). The Actiheart was found to vary from the double labelled water by less than 19 kcal/day, with no systematic bias (*ibid*). Comparing the Actiheart prediction equation to indirect calorimetry in 39, 13 year old English children found that the two were not significantly different during running and flat walking though the Actiheart did significantly under-predict AEE during graded walking by 38 kJ/kg/min (Corder et al

2005). The AEE predictions based upon accelerometry only, using data collected from either the Actiheart, ActiCal or Actigraph, were consistently significantly different from the indirect calorimetry results (*ibid*).

This study in Maya children is the first study to use Actiheart in pre-pubertal, free living children in a developing country.

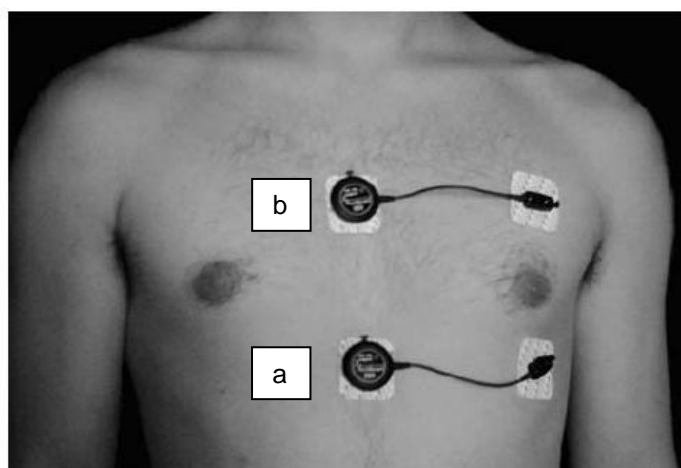


Figure 3.1 Actiheart placement: a) preferable; b) used in the case of breast development.

¹Photo taken from the Actiheart guide to getting started 4.0 (2008).

The Actiheart was worn for 7 days (Tuesday to Monday). The majority of Actiheart were placed on the children at school, after the mother had been interviewed and anthropometry had been performed on the child. School was the best location for the placement of the Actiheart because multiple children were able to be fitted at one time. The children were taken out of class for 10-15 minutes for the placement and calibration of the Actiheart with the consent of the school directors and teachers. The other Actiheart were placed on children at their houses. This method allowed for the researchers to explain Actiheart maintenance and purpose to the mothers or an adult of the house.

Almost all of the Actiheart were collected at school on Monday. Occasionally a child would not be wearing the Actiheart and the Actiheart would either be collected from the child's house or the child brought the device to school the next day. After collection the data was downloaded to a laptop and the Actiheart were recharged, usually overnight.

a. Placement of the ECG pads

The mother or grandmother was present if the Actiheart was placed at home. In the schools, the children were taken by one of the researchers to a private area, generally the director's office, an empty classroom or the bathroom. Only female researchers placed ECG pads on the girls. They lifted their shirts and the areas of their chest where ECG pads were to be attached were cleaned of dead skin and moisture by rubbing with a piece of paper. The ECG pads were then placed on the child's chest and electrodes attached in the bottom position for most of the children (Figure 3.1). For a minority of girls ($n=4$) there was enough tissue on the breast area to obscure the reading if the ECG pads were placed in the lower position. For these girls, the ECG pads were placed in the upper position. Only two of these girls had nipple maturation, indicating that the tissue was breast buds and not adipose deposits (Marshall and Tanner 1969). This distinction in nipple maturation was made by the female researchers when attaching the Actiheart (Marshall and Tanner 1969).

b. *Signal test*

First the Actihearts underwent a signal test, a 2 minute period where the Actiheart is worn by the participant to ensure the device is recording usable data. This short period allows the researchers to determine whether the electrode pads are in the correct position and if the Actiheart is collecting data on heart rhythms.

c. *Walking test*

Once the signal test was complete, individual calibrations were performed. Individual calibrations decrease the error introduced by using reference curves due to inter-personal variability in heart rates. The suggested individual calibration test is an eight minute step test, which increase the pace of stepping throughout the test period. After concern over time constraints, CamNtech and the Medical Research Council Epidemiology Unit were contacted and a 2 minute walking test at a steady pace was recommended. Therefore, the fieldteam selected the two minute walking test in order to conserve time. However an individual heart-rate calibration was not able to be calculated as the walking test was steady state, rather than the varying physical activity intensity levels required. In order to perform an individual calibration, the heart rate at various known physical activity levels must be measured. Therefore, the walking test was not able to be used for individual calibration and the group

reference curve was used instead. The group reference curves were based upon British children (Corder et al 2005).

The child and parents (if present) were shown the movement and heart data for the walking test and explained what each meant. This helped them understand the purpose of the Actiheart.

d. *Instructions and follow-up*

After the signal test and walking test were completed, the child was instructed to act normally and not go swimming. They were told that bathing is fine but to clean around the ECG pads and not put the pads or Actiheart directly in water.

Obtaining objectively measured physical activity in children under free-living conditions is technically and logistically difficult. Many problems with the original procedure were found and therefore the protocol was altered through the beginning of the fieldwork period in order to circumvent the difficulties. These difficulties were primarily related to the participants' young ages and the hot climate. The logistic difficulties were fully discussed in a paper published in the *American Journal of Human Biology* (see Appendix E) and are discussed below.

e. *Logistics of using the Actiheart*

The Actihearts were developed for use in the U.K., where extremely high temperatures are rare. The electro-cardiogram (ECG) pads are designed to stay well adhered to skin for 2 weeks under mild conditions, with regular showering. The high temperatures (33°C in the dry season) and humidity (60%) in the Merida dry season combined with the lack of access to air conditioning leads to constant sweating. The ECG pads did not reliably adhere to the skin for longer than 3 days in these urban Maya children. Difficulties with adhesion reliability was also found in adults in Cameroon (Assah et al 2010). In order to reduce the data lost due to the unreliable adhesion, the ECG pads were changed halfway through the week for all children who began physical activity monitoring on week 3 of Actiheart data collection. The researchers visited the schools or the child's home in order to change the ECG pads. This happened almost exclusively on Friday, four days after the Actihearts were fitted to the children, midway through physical activity monitoring. Also beginning week 3 of

the Actiheart data collection, bags were given containing extra ECG pads and an instruction sheet. The instruction sheet contained information on how to place the ECG pads in case one fell off over the course of the week as well as a number to contact the researchers if a problem with the Actiheart occurred.

Likely due to the young ages of the children, it was found that instructions needed to be given to the mothers directly as the children did not reliably pass on the directions. Therefore, an information sheet was also given, along with the extra ECG pads, to the children to take to their parents from in weeks 3 and 4. However this did not substantially improve data quality. Therefore, from week 5 onwards, the researchers gave instructions and extra ECG pads to the mothers face-to-face on the day the Actiheart was fitted to the children. The mothers were given the instruction sheet and the instructions were also given verbally. The instruction sheet included the telephone numbers of the fieldworkers (AV and JT) if any questions or problems arose throughout the week.

Actiheart was also damaged by the boisterous activity of the children, particularly the boys. The wire of two Actiheart was completely disconnected by boys while they were wrestling (not with each other). As the Actiheart had to be repaired in the U.K. due to the nature of the wire-device connection (clamped and not soldered), the two Actiheart were then out of commission for the rest of the fieldwork period.

Increasing the instructions and information given to the families greatly increased the quality of the data.

E. *Data handling*

1. *Data input and cleaning*

During data entry, the database, code sheet and interview were all in Spanish. For the interview, a code sheet was made to contain each variable and the appropriate code to enter into SPSS so that the interview was fully pre-coded. This eliminated the need for each interview to be individually coded. All interview and anthropometry data were transferred from the paper documents to a spreadsheet in SPSS by AV, JT and myself, and then proofread by AV and JT. Any unclear data point on the paper document was discussed between the fieldworkers who collected

the data (for interviews AV or JT and for anthropometry AV and myself or JT and myself). An agreement was then reached on the data point to be entered. I entered the data collected between 1st April and the 20th of June, while I was in the field. The data collected after I left (21st June to July) was entered by AV and JT. AV and JT checked all of the data before sending the final dataset to Loughborough in early August 2010. After the database was sent, scanned copies of the interview and anthropometry sheets were sent to me in Loughborough. I also cleaned the data and discussed inconsistent answers given by the mothers with the team throughout the data entry and cleaning process.

Then I did a 10 percent re-entry and check of the data, where 6 cases were re-entered and visually checked against the originally entered cases for any discrepancies. *A priori*, the upper limit of acceptable discrepancies was set at one per case with 449 variables. The cases that were re-entered were determined by an online random number generator and were 143, 146, 150, 154, 103 and 114. The number of mistakes found in the cases that had already been cleaned was six, within the limit of acceptable discrepancies. However four of these six discrepancies were found in the FFQ sections of the database and were frame-shift errors. The wrong number of blank cells was included, so the entered data was shifted by one cell. A meeting was held between HW and supervisor PG to decide on any further data cleaning that was required. It was decided that due to the high proportion of mistakes in the FFQ section of the dataset only, that section would undergo further checking by another researcher (HA). HA checked the FFQ sections of the first 15 cases and found no mistakes. Systematic cleaning was halted and considered complete at this stage.

a. Normality

Normality distribution tendencies were checked for the anthropometric variables. The children's anthropometric variables were checked using the sex and age specific z-scores. This allowed a standardised limit to be set. Since no systematic errors were detected in data entry, ratios (BMI, SHR and knee height ratio) were the main variables checked. An individual with a very high or low ratio is likely to be due to an error in data entry as extreme ratios are not biologically likely.

One participant (a mother) was found to have biologically improbable knee height in both the electronic database and the original paper data collection sheet.

She had a knee height ratio of 45.8% (knee height= 79.3 cm and stature of 144.7 cm). As the knee height was biologically improbable but stature was within the range of this population, the knee height was deleted and considered to be missing data.

b. *Derived variables*

Derived variables and ratios were calculated using equations from Frisancho (2008). They include body mass index (BMI), Equation 3.1; sitting height ratio (SHR), Equation 3.2; estimated leg length (ELL), Equation 3.3; total upper arm area (TUA), Equation 3.4; upper arm muscle area (UMA), Equation 3.5; upper arm fat area (UFA), Equation 3.6; and arm fat index (AFI), Equation 3.7.

Equation 3.1: Body mass index

Body mass index = weight (kg) / stature (m)²

Equation 3.2: Sitting height ratio

Sitting height ratio = (sitting height / height) * 100

Equation 3.3: Estimated leg length

Estimated leg length = stature (cm) – sitting height (cm)

Equation 3.4: Total upper arm area

Total upper arm area= mid-arm circumference² / (4 x π)

Equation 3.5: Upper arm muscle area

Upper arm muscle area = (mid-arm circumference – triceps skinfold x π)² / (4 x π)

Equation 3.6: Upper arm fat area

Upper arm fat area = total upper arm area – upper arm muscle area

Equation 3.7: Arm fat index

Arm fat index= (upper arm fat area / total upper arm area) x 100

c. *Bioelectrical Impedance*

The equations chosen to estimate body composition were published by Lohman, Stolarczyk and colleagues (Lohman et al. 1999, Stolarczyk et al. 1994). These estimation equations were derived using data from six indigenous tribes from Arizona, South Dakota and New Mexico in the United States (Lohman et al. 1999, Stolarczyk et al. 1994). The equation for adult women was developed using 151 women ages 18-60 years, following all bioelectrical impedance recommendations for fluid intake and other behaviours (Stolarczyk et al. 1994). Stolarczyk and colleagues developed the formula (Equation 3.8) and compared the results with

hydrodensitometry correcting for individual differences in total body mineral content, as determined using dual x-ray absorptiometry. The developed predictive equation was also compared to three other BIA predictive equations. The equation developed was the most accurate BIA equation tested and was highly correlated with the hydrodensitometry measurement ($R^2=0.803$) with a standard estimate of error of 2.63kg for fat-free mass (FFM). Comparing Equation 3.8 to previously published predictive equations found that the previously published equations significantly overestimated FFM for the sample of women. This included another equation developed using a North American indigenous sample by Rising et al. (1991), which combined men and women into one equation.

The equation for child's body composition was developed and validated using unpublished data by the same research group in the same North American indigenous tribes as for Equation 3.9 (Lohman et al. 1999). The equation was developed using a sample of 98 children with a mean age of 11 years old.

Equation 3.8: American Indian women's fat free mass

$$\text{FFM} = 0.001254(\text{ht}^2) - 0.04904(\text{R}) + 0.1555(\text{wt}) + 0.1417(\text{X}_c) - 0.0833(\text{age}) + 20.05$$

FFM= fat-free mass, ht= height, R=total body resistance, X_c = total body reactance, age= age in years.

Equation 3.9: American Indian child's percentage body fat

$$\text{Percentage body fat} = -0.49\text{age} + 0.51\text{sex} + 0.44\text{wt} + 1.55\text{triceps} + 0.15\text{subscapular} + 0.54(\text{ht}^2/\text{R}) + 0.13\text{X}_c - 0.04 \text{ triceps} \times \text{S}^2/\text{R} - 10.91$$

Sex: 0=boys, 1=girls; triceps= triceps skinfold thickness (mm); subscapular = subscapular skinfold thickness (mm); S= height; R= total body resistance (Ω); X_c = reactance (Ω)

2. Anthropometry classification

Childhood z-scores and percentiles were calculated for stature, weight, WC, sitting height and SHR. These were calculated using the sex specific Comprehensive reference developed by Frisancho (2008). The Comprehensive reference was chosen as it was created using data from the Third National Health and Nutrition Examination Survey (NHANES III) from the United States in 1999-2002. The NHANES III is a stratified, nationally representative sample of the US population, which over-sampled ethnic minorities. The percentage of participants according to

ethnicity is: White (European-American) 34.4%, Black (African-American) 26.8%, Hispanic/Latino (mostly Mexican-American and Cuban) 27.0% and Other 4.3%, with 7.5% missing an ethnic affiliation. NHANES III is, perhaps, the largest sampled, statistically validated growth reference that includes children of Mexican descent. As such, it is considered the most appropriate for use with a Mexican population such as the Maya. Therefore, the z-scores calculated from the Comprehensive references are the one used in statistical analyses.

Z-scores and percentiles were also calculated using the WHO growth reference. The WHO references are reworked curves from the 1977 NCHS reference curves based upon participants in United States, cross-sectionally sampled between 1959 and 1970 in the National Health Examination Survey (NHES), Cycles I-III. In the National Health Examination Survey, ethnic distributions were not targeted in the stratified sampling method.

a. *Classification of Height*

Classification of stunting lacks a clear biologically based cut-off (Song and Song 2008). Therefore, the choice of cut-offs must be decided based upon the relevance to each sample and population. Stunting classifications for children tend to be either the 5th percentile or the -2 z-scores of an internal or external reference of height-for-age. The WHO recommendation of -2 z-scores is the most commonly used definition and is more rigorous than the 5th percentile (Lutter et al 2010). The 5th percentile is recommended by the Center for Disease Control and Prevention (CDC) (CDC 2002). The children in this study were classified as stunted using both the 5th percentile and -2 z-scores of the Comprehensive reference and WHO reference (Frisancho 2008). The majority of statistical analyses use the Comprehensive reference.

The classification of adults as stunted has far less consensus than children, primarily because it is acceptable to use percentiles, z-scores or stature in centimetres. As growth in adults has ceased it is more convenient to deal in whole numbers for cut-offs. Cut-offs previously used for adult female stunting are 145 cm (Hernandez-Diaz et al. 1999), the 10th percentile of internal references (Bosy-Westphal et al. 2009) or the 25th percentile of internal references (Florencio et al. 2007). The most commonly used cut-off of adult female short stature in Mexico is 150 cm (Lara-Esqueda et al. 2004; Lopez-Alvarenga et al. 2003; Varela-Silva et al 2009),

which corresponds to the 4.6th percentile and -1.68 z-score using Frisancho's Comprehensive reference (2008) and to the 2.22 percentile and -2.01 z-score on the WHO references (Frisancho 2008).

The mothers of this sample were classified as stunted using both 145 cm and 150cm as cut-offs.

b. *Classification of overweight, obese and wasting*

BMI was used to classify the mothers and children as overweight. Mothers with a BMI between 25 and 29.99 were classified as overweight and greater than or equal to 30 were classified as obese. International Obesity Task Force (IOTF) age and sex specific guidelines were used to classify the BMI of children as overweight and obese (Cole et al. 2000).

Mothers with a %BF greater than or equal to 34% were classified as overfat (OF) using the Comprehensive reference (Frisancho 2008). Childhood OF classification based upon %BF was performed using the age and sex specific 85th percentile defined by McCarthy et al. (2006) constructed with English children.

Children whose weight-for-age percentiles were below the 5th percentile were classified as wasted (Frisancho 2008).

c. *Abdominal obesity*

Waist circumference was used to classify individuals as having central obesity. For the mothers, the cut-off for adult women of 88cm for central obesity was used (Frisancho 2008). This has previously been shown to have high sensitivity in classifying CVD risk factors in Yucatec women (Guatemala), both in stunted and non-stunted individuals (Gregory et al. 2007).

3. *Actiheart data*

The Actiheart software was used to calculate energy expenditure using branched equation models (CamNtech 2009). Branched equation models have two equations, one for heart rates below the 'flex point' and another for heart rates above. The flex point differentiates between activity levels (resting versus active) as it is the mean of the highest resting heart rates and the lowest heart rate during the lowest

intensity of an incremental exercise test. At heart rates below the 'flex point', the slope of the line is flatter than the slope used above.

As there is inter-personal variation in heart rates and fitness levels, individual calibration is recommended. As discussed above (section 3.D.3.e), a steady state walking test was performed instead of the incremental step test. Therefore it was not possible to determine the lowest heart rate during the lowest intensity of the incremental exercise test, eliminating the possibility for individual calibration. Thus the external group calibration curves were used to predict energy expenditure from heart rate and accelerometry counts in these analyses of urban Maya children. The group calibration curves were created using 39 U.K. children (23 boys) aged 13 years (Corder et al 2005). The error introduced into the sample through using an external group calibration curve is likely similar across the sample, as the sample is fairly genetically homogeneous and has experienced similar chronic environments. Therefore, intra-sample comparisons can be made but the error is likely too high for comparisons with external samples.

The Actiheart data were cleaned using Actiheart software version 4.52. The most common missing data are the heart rate data due to poor electrode adhesion. Heart rate data were most often missing for short periods of time. Therefore, the Actiheart software performs a straight line interpolation when heart rate data are missing for less than 5 minutes. Thus, in the data cleaning any periods with missing heart rate data lasting 5 minutes or longer were removed. If a period of missing heart rate data that lasted at least 5 minutes was flanked on each side by one minute of heart rate data and then missing heart rate data, the period of removing the data was continued. This was by far the most common reason for the removal of data. Data were also removed when there was a mismatch in the heart rate and accelerometry data, the reason for less than 5% of removed data. For example, extended periods of high accelerometry counts and low heart rates were removed.

An issue arises when attempting to determine whether to include days with incomplete data. Exclusion of incomplete days can cause a selection bias while inclusion can result in an inaccurate representation of activity (Ward et al 2005). Therefore it is necessary to define what the minimal amount of data can be considered for a day in order to balance acceptable data loss with data quality. There are no guidelines for the definition of a day's worth of data for Actiheart, but the discussion has been occurring for other types of physical activity monitors for several

years. The guidelines for accelerometers were used for this analysis. While there are several options for the definition of a day, the most straightforward was chosen (Ward et al 2005). Sleep was defined as a period in the night with no or very little movement and a sustained lower heart rate than during day hours. Waking hours were considered all other hours. A minimum of 10 hours of usable data during waking hours was selected as a day of usable data (Ward et al 2005). The hours of waking usable data were determined by calculating waking time and subtracting the minutes of removed data.

In monitoring free living physical activity, it is recommended that the first day of monitoring be eliminated from analyses due to reactivity (Clemes et al. 2008; Ward et al. 2005). Individuals, including children, who know their physical activity levels are being monitored tend to increase their physical activity for a period of time. Such a reaction is problematic when the goal of the research is to gain a picture of habitual physical activity. These children were aware that it was their movements that were being monitored, as they spent up to 15 minutes doing the signal and walking test. Another concern with including the first day of monitoring was that it was a shortened day, as the Actiheart was placed on the children at school and not upon their waking. For both of these reasons, the first day of monitoring, Tuesday, was not included in any analyses.

The number of days as well as the day of the week is also important for habitual physical activity monitoring. Differences in physical activity patterns between weekdays and weekend days are well described. It is therefore necessary to measure physical activity during the week and weekend. It is generally recommended that a minimum of 3 days, preferably 5, are used to estimate an individual's habitual physical activity or energy expenditure. Due to the quality control measures put in place, only 34 had 3 days of usable data, including weekdays and weekend days and excluding the first day of monitoring. This is out of the 53 children who were given an Actiheart for one week. Due to the proportionately large increase in sample size gained from using 3 days instead of 5 as criteria, 3 days of data, 2 weekdays and 1 weekend day, were used.

Physical activity levels (PALs) were also calculated by dividing absolute RMR by absolute AEE. In order to adjust energy expenditure for body size, both RMR and AEE were divided by body weight (kg) and also FFM (kg). The Actiheart software also calculated metabolic equivalents (METs) for the data, providing an estimate of

the number of minutes spent in light (MET<3), moderate (3-6) and vigorous (>6) activity.

4. *SES index construction*

The impact of SES on health and growth is multi-faceted and complex. A single variable measuring SES is not able to capture the multiple dimensions of SES and its relationship with health. Therefore multiple SES variables that addressed the same dimension of SES were grouped together to form an index that allowed for multi-dimensionality of the concept. Such a method has been used in previous research. For example Sheppard et al. (2008) recommended creating and using an index for SES when SES was not the primary outcome of the analysis. Hernandez-Diaz et al. (1999) used principle component analysis (PCA) to compute an aggregate SES score by inputting all SES variables simultaneously and using the first factor extracted. Using the latter method with SES data from this Maya sample, the first PCA factor explained over two thirds of the variance in current SES.

The indices created were attempting to capture the different dimensions of consumer durable ownership, family stability and economic stability. These indices were derived using PCA and additive methods. These indices were created for the mother's recalled early life and the family's current SES. The other SES variables were not grouped into indices because they have been shown to work independently and capture other environmental effects besides SES (e.g. flush toilet) or the variables that theoretically group together may not have a consistent effect on health. For example, the number of siblings the mother had exhibited a U-shaped relationship with her stature while birth order was linear. These two factors may have theoretically been combined into one index as they both capture aspects of the maternal demographic environment, but because of this inconsistent relationship with stature they were not grouped together.

The indices were created by grouping variables together using both statistically driven PCA and theory driven index development. The PCA grouped indices combined variables that statistically loaded well together, which made for very unintuitive combinations such as grouping refrigerator ownership with problematic migration and nothing else. The theory driven groupings were therefore favoured because theory was used to guide the combination of variables put together. This means that indices included similar factors, the meaning of which could be discussed

and interpreted in a relatively straightforward way. Therefore, the indices created by using theory driven methods to group the variables were chosen to include in further analyses.

However exploratory PCA was useful in determining variables that would theoretically be grouped together but did not belong in their theoretical grouping. Variables related to ownership of goods, such as consumer durables, grouped together. However it was consistently found that ownership of a bicycle or tricycle, (both in the mother's early life and currently) was not related to the other ownership variables. Also, whether the family had a *patio grande* (fruit trees or vegetables in the garden) did not statistically belong in any group. This held true whether *patio grande* was coded as a bivariate (yes/no) or continuous (number of plants in the garden). Therefore, bicycle/ tricycle and *patio grande* were omitted from the final ownership indices. Ownership of a television was universal and therefore could not be included due to the lack of variation in this measure.

For some variables the lack of a statistical grouping was not a concern on theoretical grounds. It was found that the variables estimating family stability (e.g. death in the immediate family, divorce of parents; Appendix B, question 32) did not group together. This was unsurprising as one type of family instability does not necessarily follow another type of instability. For example, a serious illness in the family is not necessarily related to a problematic migration in the family. Nevertheless all of these factors do measure family stability and so it is logical to have an additive measure of a family's experience of these types of event based upon strong theoretical grounds. Therefore, a cumulative score of these experiences was created. Table 3.1 shows the variables included in each index grouped using theory as the basis for construction.

The materials of the mother's childhood home were combined into an overall score based upon the roof, walls and floor. The materials were classified based upon 1 to 3 categories (see Table 3.2). Floor materials were only classified based upon permanency as there was a clear delineation between dirt and other floors, which were mainly concrete. Wall and roof materials were classified by permanency, traditional materials and whether the materials polluted the air. Within the wall and roof categories, one point was added for non-traditional, permanent and non-air polluting materials. These category scores were then added together and the mean was calculated for both the walls and roof. The flooring score was simply one point

for a permanent floor and zero for a dirt floor. The mean score for the roof and wall were each added to the flooring score to create an overall measure of housing materials. This overall measure of housing quality was used in subsequent analyses. Current houses were not classified in this way due to the homogeneity of building materials used (see Chapter 4, section A.2).

Table 3.1 Variables included in each theory driven SES index

| Ownership | Family stability | Economic stability |
|-------------------------|--|-----------------------------|
| Radio | Serious illness in family | Job loss of caretaker |
| Telephone | Death in family | Pawning of valuable item(s) |
| Refrigerator | Divorce of caregivers | Selling of valuable item(s) |
| Car | Problematic migration of family member | |
| Television ¹ | Other problems ³ | |
| Animals ² | | |

¹Not included in the current indices when variables are coded as owned or non due to current universal ownership

²Only included in the mother's early life indices

³Most commonly alcohol abuse

a. Theory driven principle component analysis

Using theory driven grouping, the variables were forced together and a PCA was performed using varimax rotation on variables for ownership, family stability and economic stability. The PCA weighted each variable individually allowing for variables to have differing importance in the models. The first component variable extracted was saved and used as the index score.

Table 3.2: Mother's recall of her childhood housing materials with the materials classified as traditional, permanent and air polluting

| | | N | Traditional | Permanent | Air polluting |
|--------------|----------------------------|----------|--------------------|------------------|----------------------|
| Roof | Palm leaves | 12 | Y | N | N |
| | Wood | 5 | Y | N | N |
| | Sheet metal | 6 | N | N | N |
| | Cardboard soaked in petrol | 14 | N | N | Y |
| | Asbestos | 5 | N | N | Y |
| | Mortared rubble | 3 | Y | Y | N |
| | Concrete blocks | 5 | N | Y | N |
| | Cement | 8 | N | Y | N |
| Walls | Earth | 14 | Y | N | N |
| | Grass | 1 | Y | N | N |
| | Wood | 5 | Y | N | N |
| | Mortared stone | 7 | Y | Y | N |
| | Cardboard soaked in petrol | 9 | N | N | Y |
| | Concrete blocks | 12 | N | Y | N |
| | Cement | 10 | N | Y | N |
| Floor | Earth | 20 | n/a | N | n/a |
| | Concrete | 35 | n/a | Y | n/a |
| | Tile/ brick | 3 | n/a | Y | n/a |

b. Theory driven additive indices

Within the theoretical groupings, simple addition was performed in order to construct the additive indices for ownership of goods, family stability and economic stability. The most desirable category for each measure was coded as one with the less desirable coded as zero. For example, owning a refrigerator was coded as one, not owning a refrigerator was coded as zero. Also having divorced parents was coded as zero while not having divorced parents was coded as one. The scores for each grouping were summed and averaged to create the additive indices. This gives every item in the score the same weight in the index, whereas the PCA method weights the different components according to their correlations with the other measures included in the index.

For the current ownership index, another level of complexity was available for analysis, the age at which the family obtained each item. The proportion of the child's life the family had owned the item was calculated. These proportions were then averaged to create the proportional additive index for ownership. This approach allowed television ownership to be included as not every family had owned a television for the child's entire life, allowing for some variability in the measurement. Though this information was collected for the mother's early life, the lack of a clear endpoint for their growing years did not allow this information to be used in an index.

5. Pubertal stage assignment

The pubertal characteristics of the Maya girls as reported by their mothers are shown in Table 3.3. Gonadarche is often viewed as the beginning of puberty (Plant 2002). Peak height velocity and an increase in adiposity are two of the physical characteristics of puberty. As childhood linear growth and body composition are the foci of this thesis, girls will be excluded if they are in gonadarche.

The limitations of the tool and method used for collecting this data force caution when classifying these girls as gonadarchal. Self-report is unreliable due to the subjectivity and the ability of the participant to understand and relate the questions to their own bodies. The problems are magnified when a third party, such as mothers, are interviewed. Due to the confounding factors of the mid-childhood growth spurt and subcutaneous adipose tissue in the breast area, reports that these two factors have ‘just started’ are dubious. Thus, only reports that the factors are ‘definitely underway’ can be considered relatively reliable, though both ‘just started’ and ‘definitely underway’ will be examined.

Table 3.3 Maya mother’s report of their daughter’s pubertal characteristics and their stage of development (n)

| Pubertal stage | Characteristic | Stage of development | | |
|-------------------|----------------|----------------------|----------------|---------------------|
| | | Not yet started | Barely started | Definitely underway |
| Adrenarche | Body hair | 26 | 1 ^a | 0 |
| | Acne | 25 | 2 | 0 |
| | Oily skin | 21-no | 6- yes | |
| | Odour change | 21 | 4 | 2 |
| Gonadarche | Growth spurt | 15 | 10 | 2 |
| | Breast growth | 15 | 10 | 2 |

^aThe body hair was reported in the underarms.

In this sample of Maya girls, the majority of girls with reported growth spurts, were classified as ‘barely started’. It is likely that this question is primarily picking out the children in the mid-childhood growth spurt, which occurs between 6 and 8 years of age, and not peak height velocity, which tends to occur after the age of 10. The original English wording was ‘*During certain times, young people start to grow a lot and quickly. This is called a growth spurt. Would you say that your growth in height: has not yet begun to spurt..., has barely started, is definitely underway or seems complete?*’ (see Appendix B for the full English version of the interview). The lack of

specificity of the question of the growth spurt makes it impossible to determine whether the answer is related to the mid-childhood growth spurt associated with adrenarche or the more intense growth spurt that occurs with gonadarche.

Older girls are more likely to be in gonadarche and thus an independent t -test was performed comparing the decimal ages of those with reported growth spurts. There was no significant difference in the ages of girls who had begun their growth spurts and those that had not ($T=1.29$ (25) $p=0.209$). The girls with no reported growth spurt were slightly younger (mean age=8.40 yrs, SD=0.85) than those with reported growth spurts (mean age=8.76 yrs, SD=0.58). Thus it is likely that these girls were not in the gonadarchal growth spurt but the mid-childhood growth spurt. The wording of the growth spurt question makes it impossible to determine which of these options the mother was considering when she answered the question.

The development of breast tissue can be confused with non-puberty related adipose deposits in overweight or overfat girls. A trained assessor can differentiate the breast tissue from subcutaneous fat based upon the nipple development and breast shape. However without training, distinguishing can be difficult. It is very unlikely that the untrained mothers were able to make this distinction. To assess the relationship between adiposity and reported breast development in this sample of Maya girls, a chi square was performed. Girls who had any breast development were compared to those who had not for overweight based upon BMI as well as over-fat based upon %BF. The girls who had breast growth were significantly more likely to be overweight based upon BMI (X^2 (1)= 10.800, $p=0.001$) and over-fat (X^2 (1)= 8.211, $p=0.004$). All 12 girls with reported breast development were over-fat. Thus it is likely that the actual prevalence of breast development may be much lower than was reported though it could be that increased adiposity is related to earlier puberty (Novotny et al. 2011). The two girls with breast development 'definitely underway' were the only girls wearing bras when the Actiheart was placed on their chest, which supports the lower actual presence of breast development. In order to determine if there was an age difference between those who had reported breast development and those without, an independent t -test was performed. The girls with breast development (mean age=9.06, SD= 0.62) were significantly older than those without (mean age 8.17, SD= 0.54) $t(25)=0.89$, $p=0.001$.

Only girls with breast development reported as 'definitely underway' will be considered as in gonadarche for the purpose of this work. While this method has the

potential to misclassify girls in the early stages of breast development, it is likely to correctly classify more of the girls who are not yet in the early stages of gonadarche. Therefore the error introduced by the classification of the girls who are gonadarchal as pre-gonadarchal will be less than if those who are pre-gonadarchal are classified as gonadarchal.

The two girls with their growth spurt reported to be ‘definitely underway’ also reported that their breast development was ‘definitely underway’. Therefore these two girls were classified as gonadarchal. In order to assess what impact they had on the findings, each final analysis was performed including and excluding them. If the two gonadarchal girls altered the findings from the final analyses, then each step of the analysis was performed and explained without them.

B. Data analysis

The differences between interviewers AV and JT as well as anthropometry assessors AV, JT and myself were checked using independent *t*-tests and ANOVAs with *post hoc* comparisons (Appendix C). HA, SL and MLS were not included in these analyses because of the low number of assessments and interviews they performed. The anthropometric assessors were significantly different on mothers’ and children’s triceps skinfolds only. Therefore, measures of peripheral adiposity (AFI, AFA and 2SK) must be interpreted cautiously. This difference was not systematic between mothers and children. For the interviewers, in order to reduce the number of comparisons, only the SES indices were compared with assessor. There was a significant difference between AV and JT on the economic stability indices, which could be because they happened to get children with different economic levels of stability or it could be because of the different interview styles used. Therefore analyses that included economic stability were interpreted very cautiously.

C. Original study design

The original study design was focused on examining intergenerational influences of maternal under-nutrition during childhood on her offspring’s obesity risk. The hypothesis was based upon the phenotypic inertia hypothesis (Kuzawa 2005), which postulates that offspring receive signals from their mother (during gestation and breastfeeding) that interact with the offspring’s own environmental

experiences to influence metabolic programming. Combining environmental exposure from a previous generation with current exposures allows adaptation to changing environments to occur over several generations (Price and Coe 2000; Price et al. 1999). Such phenotypic inertia buffers the family line from quick, cyclical shifts in environment. When applied to quickly modernizing populations where caloric abundance replaces chronic under-nutrition, phenotypic inertia may predispose children to obesity.

The phenotypic inertia hypothesis is postulated to contribute to the high rates of obesity in countries and populations undergoing rapid economic and nutrition transition. The nutrition transition is occurring very rapidly in developing countries and in some populations can occur within one generation (Popkin 2002). This speed of transition can lead to a woman who experienced under-nutrition during her own childhood, while her offspring experience over-nutrition during their growing years. Thus the intergenerational influences predispose the offspring to storing energy. However the offspring experiences an obesogenic environment and the intergenerational signals predispose them to weight gain.

Our planned methodology involved using measures of maternal linear growth (stature and SHR) to predict the children's adiposity. Chapter 2, section X provides the theory and justification for using these measures as a proxy for chronic nutritional status during the growing years. Mothers who were shorter and also mothers with a higher SHR (relatively short legged) were predicted to have children with higher levels of adiposity. Multiple linear regressions were used with child adiposity indicators (BMI-for-age z-score, %BF, WC z-score, 2SF z-score) as the dependent variables. The independent variables were maternal SHR and child's height-for-age z-scores. Child's height-for-age z-scores were included as an indicator of the child's own chronic environment.

As shown in Tables 3.5 and 3.6, indicators of maternal chronic nutritional status during her childhood (stature and SHR) did not greatly impact her offspring's adiposity indicators. However after child's stature was included in the model, maternal stature negatively (though rarely significantly) predicted child's adiposity indicators. Maternal SHR was not related to child's adiposity indicators. Child's height-for-age z-scores were consistently significant, positive predictors of child's adiposity indicators.

Table 3.4 Multiple linear regression analysis of child's adiposity indicators as predicted by maternal stature and child's own stature

| | | Model 1 | | Model 2 | |
|-----------------------------------|------------------------------------|--------------------|----------|--------------------|----------|
| | | B (SD) | p | B (SD) | p |
| Weight z-score¹ | Constant | -5.856 (3.727) | 0.122 | 4.461 (3.118) | 0.158 |
| | Maternal Stature (cm) | 0.037 (0.025) | 0.152 | -0.027 (0.021) | 0.194 |
| | Child height z-scores ¹ | | | 0.767 (0.110) | <0.001 |
| | R² adj | 0.019 | | 0.469 | |
| BMI z-score¹ | Constant | 1.387 (4.089) | 0.736 | 8.833 (4.188) | 0.040 |
| | Maternal Stature (cm) | -0.006 (0.028) | 0.839 | -0.052 (0.028) | 0.068 |
| | Child height z-scores ¹ | | | 0.554 (0.148) | <0.001 |
| | R² adj | -0.017 | | 0.174 | |
| WC z-score¹ | Constant | 0.292 (3.387) | 0.932 | 7.187 (3.358) | 0.037 |
| | Maternal Stature (cm) | <0.001 (0.023) | 0.988 | -0.043 (0.022) | 0.063 |
| | Child height z-scores ¹ | | | 0.513 (0.119) | <0.001 |
| | R² adj | -0.018 | | 0.226 | |
| %BF² | Constant | 13.220 (31.016) | 0.672 | 78.276 (29.959) | 0.012 |
| | Child Age | 1.725 (1.157) | 0.142 | 1.741 (0.986) | 0.083 |
| | Sex ³ | 3.011 (1.851) | 0.110 | 4.001 (1.592) | 0.015 |
| | Maternal Stature (cm) | -0.007 (0.202) | 0.974 | -0.417 (0.194) | 0.036 |
| | Child height z-scores ¹ | | | 4.659 (1.009) | <0.001 |
| | R² adj | 0.049 | | 0.309 | |

¹Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)²Percent body fat calculated using American Indian specific formula including bioelectric impedance and anthropometry data (Lohman et al. 1999).³Boys set as reference

Table 3.5 Multiple linear regression analysis of child's adiposity indicators as predicted by maternal relative leg length and child's own stature

| | | Model 1 | | Model 2 | |
|-----------------------------------|------------------------------------|-------------------|----------|--------------------|----------|
| | | B (SD) | p | B (SD) | p |
| Weight z-score¹ | Constant | 3.082 (5.380) | 0.569 | 1.395 (3.941) | 0.725 |
| | Maternal SHR | -0.066 (0.100) | -0.514 | -0.019 (0.073) | 0.799 |
| | Child height z-scores ¹ | | | 0.713 (0.102) | <0.001 |
| | R² adj | -0.01 | | -0.460 | |
| BMI z-score¹ | Constant | 3.381 (5.779) | 0.561 | 2.310 (5.337) | 0.667 |
| | Maternal SHR | -0.052 (0.108) | 0.628 | -0.023 (0.100) | 0.821 |
| | Child height z-scores ¹ | | | 0.453 (0.138) | 0.002 |
| | R² adj | 0.014 | | 0.139 | |
| WC z-score¹ | Constant | 4.515 (4.785) | 0.350 | 3.527 (4.311) | 0.417 |
| | Maternal SHR | -0.078 (0.089) | 0.387 | -0.050 (0.080) | 0.535 |
| | Child height z-scores ¹ | | | 0.417 (0.112) | <0.001 |
| | R² adj | 0.004 | | 0.188 | |
| %BF² | Constant | 7.825 (42.773) | 0.856 | -2.820 (37.591) | -0.940 |
| | Child age | 1.735 (1.164) | 0.142 | 1.749 (1.021) | 0.093 |
| | Sex ³ | 3.160 (1.859) | 0.095 | 3.625 (1.634) | 0.031 |
| | Maternal SHR | 0.081 (0.775) | 0.918 | 0.357 (0.682) | 0.603 |
| | Child height z-scores ¹ | | | 3.882 (0.943) | <0.001 |
| | R² adj | -0.018 | | 0.053 | |

¹Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

²Percent body fat calculated using American Indian specific formula including bioelectric impedance and anthropometry data (Lohman et al. 1999).

³Boys set as reference

In hindsight, the lack of an intergenerational influence was not surprising since the intergenerational influence would be small and the study was inadequately powered to detect a small influence, after controlling for current factors. Also, targeting a low SES population for sampling did not lead to large variation in stature or SHR for the mothers. A test of the intergenerational influence of maternal early life under-nutrition on her offspring's risk of childhood obesity should have a larger sample size and target low and high income families.

This hypothesis was not included in this thesis with more detail due to the insignificant findings. The negative results were worrying for inclusion in the thesis since the relationship between maternal recall of her childhood and her attained linear growth measurements were weak to insignificant and thus the findings would be difficult to publish. Having two chapters whose results would be difficult to publish was not considered appropriate for a doctoral thesis.

There are plans in the works for a paper examining the relationship between maternal and offspring stunting, so as to not completely disregard the intergenerational aspect to this sample.

This chapter has outlined the data collection methods and the data cleaning techniques used for this thesis. The data analysis techniques specific to each of the aims are explained in detail in the relevant chapters.

The next chapter provides a description of the living conditions experienced by the Maya living in the south of Merida.

Chapter 4. *The current Maya of southern Merida*

This chapter aims to provide a contextual framework for the research project by describing the neighbourhood, living conditions and socioeconomic status of the Maya mother-child dyads. The variables that are necessary for later analyses are also shown in the relevant chapter.

This chapter provides the general descriptive statistics of the SES variables in the sample of urban Maya as well as a description of the south of Merida neighbourhood environment. This information is from a combination of the field-team's notes (primarily my own notes) and data collected from the interview. For information on the methods used to collect the data, see Chapter 3.

A. *Neighbourhood characteristics*

The wider context of the areas in which the families live also needs to be considered when assessing their SES. This section will describe the neighbourhood, school and house characteristics of the south of Merida.

The facilities in the south of Merida are basic, but present. In terms of health care, there was one public hospital and a woman's health clinic and a few other, smaller clinics. The available piped water was not suitable for drinking and several shops dedicated to selling clean drinking water are located in various areas around the *colonias*. There was, however, no sewage system or rubbish collecting service. The area did have some bus services to other parts of the city but the services were primarily for travel between *colonias*. The buses primarily serviced individuals travelling to the northern areas of the city.

The neighbourhood was dominated by pavement and concrete. Almost all of the streets and sidewalks were paved; many of the sidewalks in the more recently settled areas were currently being constructed and older ones being repaired. The state and city governments were approaching elections, which were held at the end of the fieldwork period, and the current government was investing money in visible projects in the less developed areas of the city in order to gain support. Many of the households were also using campaign posters as curtains and other window coverings.

1. *Schools*

Primary schools in the south of Merida were more common than secondary schools. Approximately seven primary schools were located in the south which only contains one secondary school. Children attended school for four hours a day and wear uniforms, which can be purchased at a variety of locations. Schooling cost a

nominal fee of 50 to 100 pesos per term (2-5 GBP), which could be waived for the lowest income families. The fee went toward the individual school, not toward the city or state education system. The teachers generally taught at one school in the morning and then changed locations to teach at another school in the afternoon. Only one of the schools in our sample had a computer lab for the children's use. Air conditioning was very rare, with only two directors' offices and no classrooms having an air conditioning unit. All of the schools had indoor, flush toilets with sinks and running water.

The schools consisted of several, single story buildings surrounding a central courtyard (Figure 4.1). Typically the play areas were concrete as during the rainy season exposed soil became very muddy. Many schools also had other paved areas, to maximise the usable outdoor space.



Figure 4.1 Central courtyard and surrounding classroom buildings at two south Merida schools

This central courtyard was the area for physical education and school meetings. During heavy rain, which is frequent between May and September, there was no indoor option for physical education, and therefore alternate activities were performed during the physical education period. Throughout the Yucatan, when there was not heavy rain, every school day began with 'Activation', a short (<10 minutes) physical activity routine simultaneously performed by the entire school, which was typically led by visibly unenthusiastic teachers.

2. Houses

The south of Merida had very low land costs due to the proximity of the airport and two state prisons (one male and one female). The houses were typically built by the owners, varied substantially in size and quality and tended to undergo several

phases of construction as determined by the family finances (Figure 4.2). When the families first moved to the south of the city (typically from rural areas of the Yucatan), they tended to construct temporary houses made primarily from sheet metal and cardboard soaked in used petrol for water-proofing (Figure 4.2.a). Only two families in this sample lived in such temporary houses. When possible, the families re-built their house into a permanent structure. These houses were of varying quality ranging from one room with gaps in mortar (Figure 4.2.b) to multi-room, fully sealed and painted houses (Figure 4.2.c). The fathers of half the sample are bricklayers by trade likely increasing the quality of the building. All houses in this sample had electricity, concrete floors and piped water into the property. Some of the high SES houses had a room with a tiled floor.



Figure 4.2 The main house types of the south of Merida: a) Temporary house constructed of cardboard and sheet metal b) Single room permanent house with gaps in the mortar c) Multi-room permanent house which is completely sealed and painted.

The Maya traditionally and currently use hammocks as beds. Hammocks were strung across the main living area at night (Figure 4.3.a). Up to five hammocks could fit into a typical room and two adults could share a large hammock. During the

day the hammocks were wrapped up and hung on the wall to accommodate activities (Figure 4.3.b) or extended to be used for seating, as needed. Storage space in these houses was limited, with most items being stored in one wardrobe. Most of the tables and chairs were plastic and provided for free by large drinks corporations such as Coca-Cola or beer companies.



Figure 4.3 Hammocks in urban Maya houses: a) in use; b) wrapped up and hung up on the wall

As can be seen from Figure 4.4 most of the house plots were quite large, with the average size being estimated at an acre or more. For some families, the extra space was rarely used (Figure 4.4.a) except for refuse burning and an outdoor toilet. Other families used the back garden as a large extension of their house, particularly for household chores (Figure 4.4.b).



Figure 4.4 Back gardens of differing utilisation in the Maya of Merida: a) unutilized back garden; b) highly utilized back garden with a 1-cooking area, 2-clothes drying, 3-washing area and 4-animal pen.

The lack of utilisation of outdoor space is an indicator of a reduction in the traditional lifestyle after migration to an urban environment. However the majority of urban Maya families still grew some edible plants. The most common are fruit trees (Figure 4.5.a) such as lime and avocado trees. Smaller plants were also grown in pots (Figure 4.5.b). These plants were grown for food (primarily as herbs), medicine and ornamental purposes. Some families also kept some fowl, such as chickens and turkeys, but never enough to meet their protein needs.

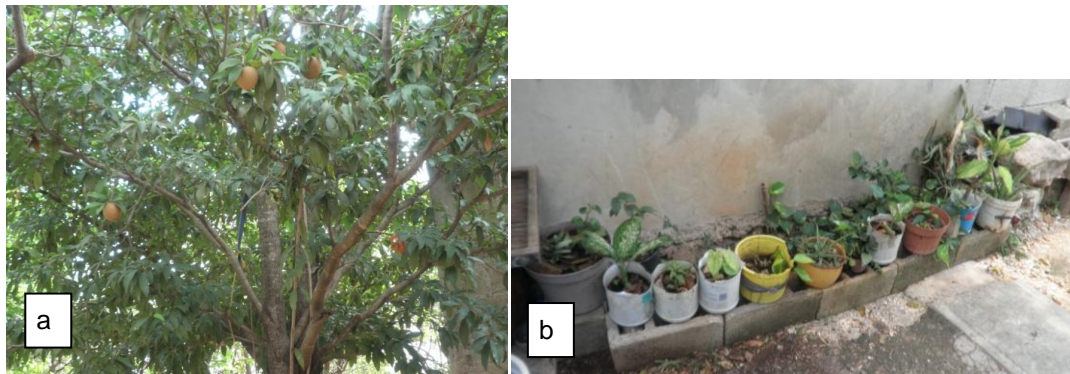


Figure 4.5 Examples of cultivated plants in the gardens of the Maya of Merida: a) zapote fruit tree; b) row of potted plants

The food was typically heated and cooked over an open fire or a gas stove. While this data was not collected for every house, it appeared as though the majority used a wood fire to cook (Figure 4.6.a). These fires were outdoors, typically against a permanent block wall and covered with a roof made of temporary materials. The other families used gas burners for cooking. These were either portable (Figure 4.6.b) or included an oven (Figure 4.6.c).

Though every house had piped water to the property, it did not always reach the house. Quantifiable data are not available on the frequency of piped water reaching the house, but from the ethnographic notes, it was uncommon. When piped water did not reach the house, buckets tended to be filled and left standing and uncovered throughout the day, which attracted flying insects fairly quickly.



Figure 4.6 The cooking methods of the Maya of Merida: a) an outdoor fire; b) a portable gas burners; c) a combination of gas burners and oven

B. *Cultural indicators*

1. *Maya language*

Traditionally, the Maya language is central to the Maya cultural and ethnic identification (Quijano 2007). However, even though the urban Maya sampled in this study identified as Maya, few predominately spoke the Maya language (Table 4.1). The vast majority of families used Spanish, while only 13 percent of families spoke Maya. Maya was the primary language of only three households in the current sample. The low prevalence of Maya spoken in urban Maya families indicates that these families have abandoned a portion of their traditional culture in order to assimilate to the dominant Mexican culture.

Table 4.1 Primary language spoken in the home in the Maya study sample

| Language spoken in household, n(%) | |
|---|-----------|
| Maya | 5.2 (3) |
| Spanish and Maya | 8.6 (5) |
| Spanish | 86.2 (50) |

2. *Family composition*

The Maya of this study tended to follow the family composition typical of Mexico in general. Most of the households consisted of the mother, father and children. The vast majority of these Maya women were currently married (67%) or in a civil union (22%) and lived with their partner, who contributed money to the household. Only 10% of the women were separated or never married, none of whom received financial support from the child's father.

It must be noted that this sample is likely over-representative of housewives, with 66% of the mothers did not work outside the home. Housewives were not specifically targeted for sampling however these were the mothers who could most easily accommodate the hour of interviewing and measurements required in this study. It is likely that this bias was introduced from the recruitment strategy adopted, which was holding information meetings at the school at the beginning of the school day. This strategy may have unintentionally excluded women who worked at that time. However it was deemed more efficient than visiting individual houses, which was the first sampling technique attempted which was highly unsuccessful. Therefore, a recruitment strategy that resulted in a larger, though potentially more biased sample, was adopted. The sample may also be biased toward slightly higher SES families who do not financially require dual incomes.

C. *Current child health variables*

The current child health characteristics and the pregnancy-related variables of their mothers are shown in Table 4.2. This sample of children had an average of 2.3 siblings, and 29% of these children were the first born. From the mother's recall, most children had healthy birth weights and received complementary foods at a mean of 5 months of age. Over 90% of the mothers reported to have received prenatal medical care and all but one of the children were born in a medical facility. Also, 97% of the children were breastfed.

Within this sample, four siblings of the children in the study were reported to have died before the age of 5 years out of a total of 190 children (including both siblings and the child measured). The under-5 mortality in this population can be extrapolated to 21.05 deaths per 1,000 live births. The average under-5 mortality in Mexico as a whole was 17 per 1,000 in 2009, down from 45 per 1,000 in 1990 (UNICEF 2010).

Table 4.2 Child health and birthing data in the Maya study sample

| | Mean (SD) | |
|---|-------------|-----------|
| Number of siblings | 2.28 (1.39) | |
| Birth order | 1.43 (1.48) | |
| Birth weight (kg) | 3.04 (0.60) | |
| Complementary foods (months) | 5.05 (3.74) | |
| Age at weaning (years) | 1.71 (1.21) | |
| | Yes, % (n) | No, % (n) |
| Sibling died in 1 st 5 years of life | 5.1 (3) | 94.9 (55) |
| Prenatal medical care | 91.4 (53) | 8.6 (5) |
| Birth in medical facility | 98.3 (57) | 1.7 (1) |
| First born | 29.3 (17) | 70.7 (41) |
| Breastfed | 96.6 (56) | 3.4 (2) |

a. Birth weight and gestational age validation

The mothers recalled the birth weight of their children. In order to determine if their recall was valid, we attempted to obtain a photograph of the child's birth certificate, with birth weight and length of gestation. This was possible for four of the children (Table 4.3) as few mothers had the birth certificate in the house. In these four cases, all of the recalls were higher than the recorded birth weights, which suggests that there may be a systematic bias in this dataset.

Table 4.3 Maternal recall of her child's birth weight versus the birth weight recorded on the birth certificate (g) in the Maya study sample

| Recall | Recorded |
|--------|-----------|
| 2750 | 2250 |
| 3200 | 2925 |
| 4250 | 4100 |
| 3200 | 2501-3500 |

In addition, the certificates also included gestational length, which had a mean of 8.83 months (0.64 SD). All of the four mothers with birth certificates for their child, correctly recalled their gestation as being full term (at least 37 weeks), though all were off by one week. This error was not systematically biased toward earlier or later births and would not have resulted in the incorrect classification of a gestation. Three of the mothers in the entire sample reported that their children were born preterm.

b. Children's infectious diseases

The majority of the mothers reported that their children (81.0%) had diarrhoea less than once a year with monthly or more frequent episodes occurring in 12.0% of

the children. Also, over 30% (n=18) of the children were reported to have been ill within the past two weeks. The majority of the illnesses were fairly minor, e.g. colds and coughs, however several were more serious, such as fever and parasites.

D. *Objective measures of current SES*

More complete data on objective measures of SES were collected through maternal interviews. These questions were based upon the Demographic Health Survey (DHS) (MeasureDHS 2011) and also included questions on family stability. The questions were mainly concerned with current SES, though some inquired about factors that had occurred within the life of the child.

1. *Current sanitation*

The sanitation of this community was basic. In this sample of urban Maya, 40% of families used their gardens for their toilet (Figure 4.7.a). These outdoor toilet areas were physically separated from the rest of the garden using temporary materials such as cloth and are occasionally rotated around the garden. Many (60%) in this sample of Maya from Merida had flush toilets in their homes (Table 4.4). The flush toilets of these families were most often in the condition shown in Figure 4.7.b though some were of higher quality (Figure 4.7.c). There was no city maintained sewer or water treatment system in the south of Merida. Almost always, toilets were directly drained into a man-made or natural cavern on the family's property.

Table 4.4 Basic sanitation variables for the Maya sample households, %(n)

| Variable | Yes | No |
|----------------------|------------|-----------|
| Flush toilet | 60.3 (35) | 39.6 (23) |
| Clean drinking water | 75.9 (44) | 24.1 (14) |



Figure 4.7 Toilet types in the houses of the Maya of Merida: a) outdoor toilet, separated from rest of garden; b) indoor toilet that must be flushed using a bucket; c) toilet linked into a main water supply

Almost one quarter of the families did not have clean drinking water (Table 4.4). There were no water treatment facilities in Merida to provide clean, piped water to houses and the piped water is not safe for drinking. This was well known throughout the city, in both the south and the affluent north. In response to this knowledge most families purchased purified water for drinking, including in this sample of Maya. The purified water was distributed primarily by large corporations, Coca-Cola and Pepsi, and costs 20 pesos (about 1 GBP) for a 20 litre jug and cheaper options are available. This clean drinking water was affordable for most families. Many of the families that did not purchase drinking water cited a preference for the taste of the piped water over purified.

2. *Current consumer durable ownership*

Current television ownership was universal in this sample and radio ownership is almost 85% (Table 4.5). The ownership of these media sources allows for a large potential influence of wider culture and may influence assimilation. This may be particularly evident in this sample of stay-at-home mothers who may have

had more time to consume such media while their children are in school. Mothers were often watching television when the researchers came to perform an interview during school hours.

Table 4.5 Reported current consumer durable ownership of the sample of Maya families, n(%)

| Variable | Yes | No |
|-------------------|-----------|-----------|
| Radio | 84.5 (49) | 13.8 (8) |
| Television | 100 (58) | 0 (0) |
| Refrigerator | 75.9 (44) | 24.1 (14) |
| Car | 12.1 (7) | 87.9 (51) |
| Telephone | 69.0 (40) | 31.0 (18) |
| Tricycle/ bicycle | 72.4 (42) | 27.6 (16) |

Refrigerators were also common in this sample, with the ownership levels at 76% (Table 4.5). Most families had a telephone however little information can be gleaned from this variable because the interview did not differentiate between a mobile or landline. Landlines were much more expensive than mobile phones in Mexico and far less common in low SES groups.

Car ownership was fairly low in this sample. From ethnographic notes, it appeared as though motorbikes were more common than cars but this was not assessed as part of the interview. The low prevalence of car ownership, combined with limited public transport (such as buses) in the area, results in high levels of active transport such as walking and bicycle riding.

Most families also owned a bicycle or tricycle. The tricycles were very heavy, made out of steel and have a storage area for transporting items (see Figure 4.8). Bicycles were also used for transport occasionally carrying large loads however large loads on a bicycle are more logistically challenging. For example, one woman was seen carrying four young children on a bicycle.

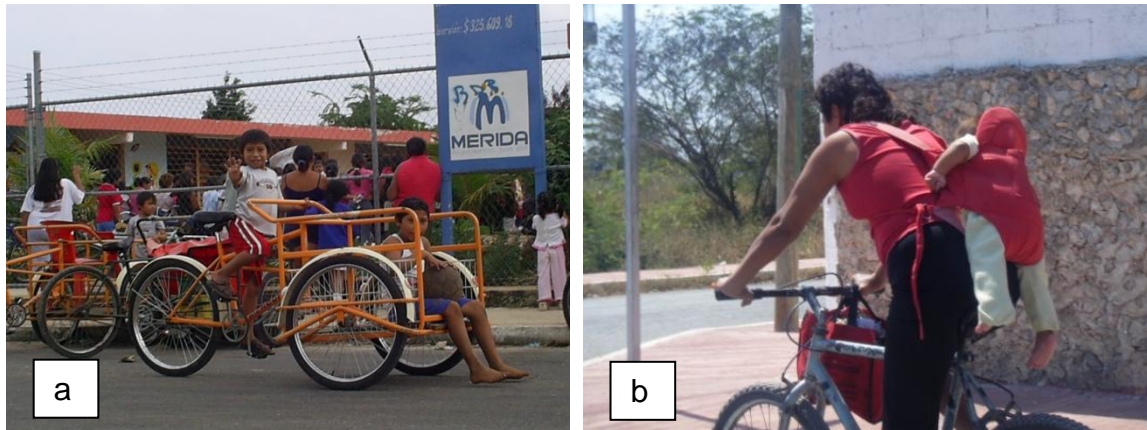


Figure 4.8 Tricycles and bicycles used by the urban Maya: a) A Maya boy using a tricycle and transporting another boy; b) a mother transporting her infant and a bag of goods on a bicycle

3. *Recent family stability*

The vast majority of women reported that their families had experienced difficulties during the life of the targeted child (Table 4.6). Only two families had not experienced a reported major difficulty. The most common difficulty reported to be faced by the mother's family was serious illness and a death in the family, which were often linked. Divorce of caregivers and migration which caused a problem within the family occurred within 17% and 10% of families, respectively. Over one fifth of the women reported other family problems, which was almost exclusively related to alcoholism of an adult male family member, primarily the father/ husband.

Table 4.6 Reported measures of current family stability of the sample of Maya families, n(%)

| Variable ¹ | Yes | No |
|--|-----------|-----------|
| Serious illness in family | 50 (29) | 50 (29) |
| Death in family | 43.1 (25) | 56.9 (33) |
| Divorce of caregivers ² | 17.2 (10) | 81.0 (47) |
| Migration that caused problems in family | 10.3 (6) | 89.7 (52) |
| Other family problems ³ | 22.4 (13) | 77.6 (45) |

¹Difficulties that have occurred during the lifetime of the child, aged 7-9 years.

² does not add to 100% because one mother was never married to the father of the child

³The majority of problems reported were related to alcoholism of a male family member

4. *Recent financial stability*

Financial instability was measured by significant job loss and pawning or selling of valuable items (Table 4.7). Roughly half of the sample had experienced a

significant employment loss in the child's life. Almost three quarters of families reported the temporary solution of pawning. From the data available, it does appear as though most of these families experienced short term financial short comings. Employment in the south of Merida was often not stabilised by contracts or unions.

Table 4.7 Reported measures of family economic stability of the sample of Maya families, %(n)

| Variable ¹ | Yes | No |
|------------------------------|-----------|-----------|
| Job loss of caregivers | 48.3 (28) | 51.7 (30) |
| Sale of valuable property | 31.0 (18) | 69.0 (40) |
| Pawning of valuable property | 74.1 (43) | 25.9 (15) |

¹Difficulties that have occurred during the lifetime of the child, aged 7-9 years.

5. *Subjective social status*

In a longitudinal study in the U.K., subjective social status was found to be more strongly related to health measures than objective measures of SES (Singh-Manoux et al. 2005). Therefore, current and past subjective social status was assessed in this sample of urban Maya. The women were not comfortable answering these questions and some became very visibly distressed. The subjective social status is intended to be left vague in its explanation to allow the participant to interpret what social status means to them (Singh-Manoux et al. 2003). However these women required a relatively detailed explanation that included money, schools, employment and health care. This explanation imposed an externally defined social status. The women were very rarely confident in their choice. Due to the difficulties in obtaining this measure, subjective social status is not used as an indicator of SES in this thesis.

E. *Mother's recalled early life environment*

This next section describes the mother's recall of her childhood living conditions.

1. *Recalled demographics*

These Maya women were raised in fairly large families (mean child number =7.6) (Table 4.8), as expected in a population with no or limited access to family planning methods (Cleland 2008). These populations often have high infant and

child mortality (*ibid*), which is also shown in this group of Maya women, over one third of whom had a sibling who died early in life, before 5 years of age.

Table 4.8 The sample of Maya women's childhood family demographics

| Variable | Mean (SD) | Range |
|--|-------------|-----------|
| Total sibling number | 6.66 (3.11) | 0-14 |
| Older sibling number | 3.03 (2.34) | 0-10 |
| Number of deceased siblings ¹ | 0.53 (0.93) | 0-5 |
| | Yes | No |
| First Born, %(n) | 13.8 (8) | 86.2 (50) |
| Sibling death ² , %(n) | 34.5 (20) | 65.5 (38) |

¹Number of siblings who died before the sibling reached 5 years of age.

²Women who reported having a sibling die before the sibling reached 5 years of age

2. *Recalled parental occupation*

Half of the women reported that their mothers were housewives/ stay-at-home mothers. The rest of the women had mothers who were housecleaners, made clothing or hammocks, laundresses, cooks, or sold various items such as fruit or firewood. One woman reported that her mother was a prostitute. The women's fathers primarily worked as manual labourers, primarily as bricklayers or farmers. Other fathers were reported to be in the military or police forces. Some were also vendors of various items. Overall, the jobs held by the parents of these women were not necessarily highly paid.

While data were collected on the occupation of the women's father, no data were collected on whether or not the father contributed money to the household. It was reported voluntarily by six women that the father was not in the family's life. In the Yucatan at this time, it was acceptable for men to have and support more than one family. However the divorce rate of caregivers was almost one third. So it is likely that at least a few of the divorced fathers were absent and did not contribute money to the family.

3. *Recalled sanitation and housing conditions*

Good sanitation measures were not frequently recalled by these women (Table 4.9). The vast majority (95%) used their back garden as their toilet. All of the women who had flush toilets in childhood lived in urban areas. Over one third of the

women recalled having their drinking water cleaned before consumption. Thus the majority of these women were very likely exposed to pathogens in their drinking water as children. Almost two thirds of these women reported that their houses had electric lights in their childhood. The primary alternate source of lighting was candles. As was the case with drinking water, urban residence was not related to electric lighting.

Table 4.9 Recalled housing and sanitation variables from the Maya women's early life, %(n)

| Variable | Yes | No |
|-----------------------|-----------|-----------|
| Flush toilet | 5.2 (3) | 94.8 (55) |
| Clean drinking water | 39.1 (23) | 60.9 (35) |
| Electric light source | 67.2 (39) | 32.8 (19) |

4. *Recalled agriculture*

The majority of families kept animals and grew some of their food (Table 4.10). The animals were primarily chickens, turkeys, and pigs. Some families also kept sheep, rabbits, ducks and horses. Most families grew many edible fruits, vegetables and herbs in their gardens. The fruits were the most numerous and included several types of oranges, limes, avocados, bananas, tomatoes, plums, coco, many types of melon and chillies. The vegetables grown in the families' vegetable gardens included several types of beans, sweet potatoes, members of the onion family and sweet corn. Few herbs were reported but some families grew cilantro (coriander) and mint. The high frequency of families growing food exemplifies how important plants and farming are to the Maya culture, which is noted in other discussions of Maya culture (Quijano 2007).

Table 4.10 Recalled agricultural factors of the sample of Maya mothers' early life. %(n)

| Variable | Yes | No |
|--------------------------------------|-----------|-----------|
| Animals for consumption or transport | 82.8 (48) | 17.2 (10) |
| Vegetable garden | 81.0 (47) | 19.0 (11) |
| <i>Milpa</i> ¹ | 54.4 (24) | 58.6 (34) |

¹a large vegetable garden with multiple types of plants grown primarily for subsistence.

5. *Recalled housing*

Almost half of the women were born in a city (48.3%, n=28). The women lived in houses made from a variety of materials (Table 4.11). These houses were constructed by the family and were primarily made out of temporary materials.

Table 4.11 Housing materials reported by the sample of Maya women for their childhood home

| | Materials | % (n) |
|-------|----------------------------|--------------|
| Roof | Palm leaves | 20.7 (12) |
| | Wood | 8.6 (5) |
| | Sheet metal | 10.3 (6) |
| | Cardboard soaked in petrol | 24.1 (14) |
| | Asbestos | 8.6 (5) |
| | Block | 8.6 (5) |
| | Cement | 13.8 (8) |
| Walls | Earth | 24.1 (14) |
| | Grass | 1.7 (1) |
| | Wood | 8.6 (5) |
| | Limestone | 12.1 (7) |
| | Cardboard soaked in petrol | 15.5 (9) |
| | Block | 20.7 (12) |
| | Cement | 17.2 (10) |
| Floor | Earth | 34.5 (20) |
| | Cement | 60.3 (35) |
| | Brick | 1.7 (1) |

6. *Recalled consumer durable ownership*

Ownership of consumer durables ranged from 3% for telephones to 76% for radios (Table 4.12). The relatively inexpensive items were owned far more frequently than the expensive items.

Table 4.12 Recalled ownership indicators of the sample of Maya mothers' early life. % (n)

| Variable | Yes | No |
|-------------------|------------|-----------|
| Radio | 75.9 (44) | 24.1 (14) |
| Telephone | 3.4 (2) | 96.6 (56) |
| Refrigerator | 12.1 (7) | 87.9 (51) |
| Car | 5.2 (3) | 94.8 (55) |
| Television | 55.2 (32) | 44.8 (26) |
| Tricycle/ bicycle | 48.3 (28) | 51.7 (30) |

7. *Recalled family stability*

Most of the women reported that their family had undergone difficulties in their childhood (Table 4.13). Only five women did not report any problems. While under-reporting due to poor recall is possible, the magnitude of these difficulties is likely to be well recalled, particularly for death and divorce.

Table 4.13 Recalled family stability indicators of the sample of Maya mothers' early life; %(n)

| | Yes | No |
|--|-----------|-----------|
| Serious illness in family | 46.6 (27) | 53.4 (31) |
| Death in family | 63.8 (37) | 36.2 (21) |
| Divorce of caregivers | 31.0 (18) | 67.2 (39) |
| Migration that caused problems in family | 24.1 (14) | 75.9 (44) |
| Other family problems ¹ | 46.6 (27) | 53.4 (31) |

¹Most commonly reported problem was related to alcoholism of a male family member

8. *Recalled economic stability*

Many of the women reported economic instability (Table 4.14). Half of the women reported that their caregivers had experienced job loss during the woman's youth. As children are often somewhat buffered from family economic strife, these families most likely experienced a very problematic job loss or job loss was a reoccurring theme in the household. The same is true for sale and pawning of valuable property, both of which were common but not universal. Overall these women appear to have been raised in situations that were not very financially stable.

Table 4.14 Recalled family stability indicators of the sample of Maya mothers' early life, %(n)

| | Yes | No |
|------------------------------|-----------|-----------|
| Job loss of caregivers | 50.0 (29) | 50.0 (29) |
| Sale of valuable property | 36.2 (21) | 63.8 (37) |
| Pawning of valuable property | 53.4 (31) | 46.6 (27) |

F. *Evidence for modernisation of behaviours*

By comparing the mother's recall of her early life to the family's current living conditions, it is clear that this population is undergoing modernisation. Firstly family size is decreasing. Total fertility rates in Mexico decreased substantially from 1960, with more than seven children born per woman, to roughly 2.5 in 2000 (UNICEF 2010). This trend is roughly seen within this sample, as the mothers grew up in

families with an average of 7.6 children while the current generation has an average of 3.3 children. It is likely that many of the women have not finished their childbearing, as most are within their prime childbearing years and many of the children are first born. It is therefore expected that the current generation will increase in family size as the mothers are likely to have more children. Divorce rates were more common among the grandparents than the mothers. Divorce among the grandmothers was reported at 31%, while only 17% of the mothers reported that they themselves were divorced, but again, this rate may increase as the mothers age.

It appears as though sanitation for this sample has improved since the mother's childhood. The percentage of families with clean drinking water has increased substantially from 39% to 76%. Clean drinking water may be partially responsible for the decrease in early life mortality among this sample, which decreased from almost 40% in the mothers' generation to 5% in the children's. The reduction of early life mortality was also aided by several country-wide public health campaigns that included universal vaccination (Frenk 2003).

Another factor that has changed from the mothers' youth is the sugar sweetened beverage consumption. The frequency of sugar sweetened beverage consumption has risen substantially, with 95% of participants currently consuming sugar sweetened beverage at least once a week (Table 4.15) as compared to 41% of mothers reporting frequent consumption in her childhood. This may be due to a recall problem from the mother's childhood. However, Mexico has increased its sugar sweetened beverage consumption during the latter half of the 20th century and is now the second largest consumer of sugar sweetened beverage in the world (ANAPRAC 2005).

Table 4.15 Urban Maya frequency of sugar sweetened beverage consumption, % (n).

| | Never | Rarely¹ | Frequently² |
|--------------------|--------------|---------------------------|-------------------------------|
| Mothers' recall | 13.8 (8) | 44.8 (26) | 41.4 (24) |
| Mothers' current | 3.4 (2) | 1.7 (1) | 94.9 (55) |
| Children's current | 8.6 (5) | 1.7 (1) | 89.7 (52) |

¹Rarely for the mother's recall was not defined by the interviewer. For current consumption, rarely is defined as less than once a week.

²Frequently for the mother's recall was not defined by the interviewer. For current consumption, frequently is defined as one or more times per week.

Current consumer durable ownership (Table 4.5) has also increased from the mothers' childhood (Table 4.12). Ownership of every item has increased, excluding a radio and bicycle. Notably, television ownership has increased from just over half to being ubiquitous. It is likely that an increase in income and decrease in price as well as a much greater desire for electronics and other consumer durables led to the increase in ownership.

G. *Diet*

The dietary data were collected as frequency of consumption, with no indication of the portion size, limiting the usefulness of the data. The description of the dietary data will cover the frequency and type of cereal, animal products, fats, sugars, fruit, vegetables and sugar sweetened beverage consumption.

The current consumption frequencies were very similar for the mothers and the children. This is likely to be due to a similarity in actual frequency of food intake and also the mothers being more likely to recall foods the children ate while at home. Mothers likely under-reported foods the child ate at school. As the children spend four hours at school and do not consume meals there, it is likely that this bias is mostly in the form of milk and packaged food, which are given daily at snack-time. The schools provide milk to students and there are small stores and carts selling packaged and fried food to students outside of the school. In these stores, water is twice as expensive as *Coca-cola*. Thus many of the children likely consume milk every school day and packaged food and sugar sweetened beverage regularly. Children also likely obtained snacks without their mothers being aware, either by purchasing the snacks from the street vendors or sharing with their friends.

1. *Cereals*

Corn tortillas formed the basis of the cereal consumption in these urban Maya, with 86% (n=50) of children and 94.9% (n=55) of mothers eating them 5-7 days per week. Corn tortillas are a staple food in the Yucatan. There is also a fairly high frequency of processed cereal consumption, including white rice, white bread (called French bread), sweet bread and pasta. The most frequently consumed of these processed cereals was French bread with 50% of the children and 58.6% (n=34) of the mothers eating it 5-7 times a week. These processed cereals contain a low fibre content compared to corn tortillas, oats and corn dough. Overall, the

sample consumed cereals with high fibre contents at a lower frequency than the processed, low fibre cereals, with the exception of the corn tortilla.

2. *Animal products*

The majority of mothers and children had a low frequency of meat consumption, at 1-2 days a week. Only one child and three mothers ate meat daily, which was chicken. The most frequently consumed meats were chicken and pork, likely due to their relatively low cost compared to beef and fish. Beef and fish were rarely consumed by these Maya, with just over half consuming each 1-2 times a week, and the remaining never consuming beef or fish. Eggs were consumed at low to medium frequencies, 1-4 times a week.

These children were reported to consume milk regularly, with half drinking it 5-7 times a week. The children almost exclusively (93%, n=55) consume whole milk. This frequency is almost certainly underreported as most of the schools give milk to the children as a snack during the school day. The schools provided chocolate and plain milk, both are full fat.

The mothers had a lower milk consumption than the children, with only 12% (n=7) consuming milk frequently, 5-7 times a week. The milk consumed by mothers was also predominately whole milk. Overall, the dairy consumption of this sample includes high levels of fat.

3. *Fats*

The main fat consumed is oil with 63.8% (n=37) of the children and 69% (n=40) of mothers consuming it 5-7 times a week. The oil used is primarily corn oil for frying. Frying is a very common cooking method for meat and tortillas. This oil is used multiple times before discarding. Lard, mayonnaise and margarine are also regularly eaten. The fat contained in these products is primarily saturated.

4. *Fruits and vegetables*

Frequency of fruit consumption was never or low (1-2 times a week) for every fruit except bananas, orange and watermelon, all of which are local fruits. The low frequency of fruit consumption indicates that micro-nutrients may be lacking in the diet of these urban Maya.

Frequency of vegetable consumption was higher than fruit consumption for these Maya. However the consumption was not high, with no more than 5.2% (n=3) children and 6.9% (n=4) of mothers reporting daily consumption of any vegetable.

The next chapter discusses the objective measurements of this sample of urban Maya: anthropometry and energy expenditure.

Chapter 5. *Descriptive results of anthropometry and energy expenditure*

This chapter presents the descriptive results for the anthropometry of the urban Maya mothers and children and the energy expenditure of the children.

A. Anthropometry

1. Anthropometry of the child

The anthropometry of the children shows that this is a small sample in all linear growth measures (Table 5.1); the children are relatively short with short relative leg lengths. The implications of these linear proportions in the use of BMI are assessed in Chapter 6. This sample exhibits linear growth that is almost always more than one standard deviation below the reference median. No significant differences between the sexes were found in linear measurements or z-scores.

Table 5.1 Anthropometric measures of this sample of Maya children's linear growth status as well as age divided by sex

| | Boys ² | Girls ² | All |
|---|-------------------|--------------------|---------------|
| N (%) | 31 (54.4) | 26 (45.6) | 57 (100) |
| Age | 8.23 (0.84) | 8.59 (0.72) | 8.43 (0.8) |
| Stature | 121.84 (5.95) | 122.54 (7.94) | 122.13 (6.82) |
| Stature z-score¹ | -1.12 (0.86) | -1.20 (0.89) | -1.15 (0.87) |
| Sitting height | 65.48 (2.69) | 65.75 (7.92) | 65.6 (3.43) |
| Sitting height z-score¹ | -0.97 (0.80) | -0.51 (0.97) | -0.76 (0.9) |
| SHR^a | 53.77 (1.28) | 53.68 (1.38) | 53.73 (1.31) |
| SHR z-score¹ | 1.02 (0.74) | 1.26 (0.89) | 1.13 (0.82) |
| ELL (cm)^b | 56.36 (3.81) | 56.79 (4.39) | 56.56 (4.05) |
| ELL z-score¹ | n/a | n/a | n/a |
| Knee height (cm) | 37.42 (2.61) | 38.07 (2.78) | 37.72 (2.68) |
| KHR^c | 30.69 (0.96) | 30.88 (0.85) | 30.77 (0.91) |

¹All sex- and age- specific z-scores calculated using Frisancho's Comprehensive reference (2008).

²Independent *t*-tests were performed to compare the sexes but no significant differences were found.

^aSHR= Sitting height ratio = (sitting height/stature)*100. ^bELL= Estimated leg length = stature (cm) – sitting height (cm). ^cKHR= Knee height ratio = (knee height/stature)*100.

In contrast to the linear growth measurements, the children's adipose indicators were found to be much closer to the median on the Comprehensive growth chart (Frisancho 2008) (Table 5.2). While the weight z-scores were found to be below the median, body mass index (BMI), waist circumference (WC), skinfolds, arm fat area (AFA) and arm fat index (AFI) were found to be above the median. It appears as though the muscle mass of these children is low. Upper arm muscle area (UMA) for both sexes is below the reference median and the average %BF is above the age and sex specific 85th percentile (McCarthy et al. 2006). Overall, this sample has high levels for adiposity indicators, which when coupled with their short stature place them at a risk for overweight in adulthood (Florencio et al. 2001).

Table 5.2 Anthropometric measures of this sample of Maya children's soft tissue and body composition by sex, mean (SD)

| | Boys | Girls | All |
|--|----------------|----------------|---------------|
| <i>N</i> (%) | 31 (54.4) | 26 (45.6) | 57 (100) |
| Weight (kg) | 25.76 (4.83) | 28.38 (7.92) | 26.87 (6.47) |
| Weight z-score ¹ | -0.64 (0.81) | -0.21 (0.93) | -0.44 (0.89) |
| BMI (kg/m ²) ^a | 17.24 (2.31) | 18.62 (3.70) | 17.83 (3.07) |
| BMI z-score ¹ | 0.48 (0.88) | 0.67 (1.03) | 0.56 (0.95) |
| WC (cm) ^b | 58.51 (5.86) | 61.4 (9.6) | 59.77 (7.8) |
| WC z-score ¹ | 0.14 (0.70)* | 0.58 (0.84)* | 0.34 (0.79) |
| Triceps skinfolds (mm) | 12.58 (6.44) | 15.30 (6.14) | 13.75 (6.36) |
| Triceps skinfold z-score ¹ | 0.45 (0.88) | 0.75 (0.93) | 0.58 (0.91) |
| Subscapular skinfold (mm) | 9.14 (5.43)* | 12.65 (7.23)* | 10.62 (6.46) |
| Subscapular skinfold z-score ¹ | 0.61 (0.79) | 0.84 (0.92) | 0.72 (0.85) |
| Sum of 2 skinfolds (mm) ^c | 21.71 (11.58)* | 27.42 (12.63)* | 24.37 (12.31) |
| Sum of 2 skinfolds z-score ¹ | 0.33 (0.99) | 0.82 (0.84) | 0.56 (0.95) |
| Supra-iliac skinfolds (mm) | 6.60 (1.19)* | 9.77 (1.88)* | 12.49 (8.61) |
| Mid-arm circumference (cm) | 19.05 (2.34)* | 20.94 (4.16)* | 19.88 (3.38) |
| Mid-arm circumference z-score ¹ | -0.56 (0.94) | 0.74 (1.17) | 0.03 (1.23) |
| TUA (cm ²) ^d | 29.3 (7.27)* | 36.22 (15.28)* | 32.33 (11.96) |
| TUA z-score ¹ | -0.07 (0.92) | 0.46 (1.09) | 0.17 (1.02) |
| UMA (cm ²) ^e | 18.31 (3.59) | 21.47 (9.16) | 19.71 (6.79) |
| UMA z-score ¹ | -1.18 (1.01) | -0.58 (1.59) | -0.91 (1.33) |
| AFA (cm ²) ^f | 10.98 (6.36) | 14.74 (8.11) | 12.62 (7.35) |
| AFA z-score ¹ | 0.54 (1.23) | 0.68 (1.18) | 0.6 (1.2) |
| AFI (%) ^g | 35.55 (12.51) | 39.67 (10.25) | 37.34 (11.54) |
| AFI z-score ¹ | 0.91 (1.22) | 1.26 (1.37) | 1.07 (1.29) |
| %BF ^h | 26.54 (0.65)* | 30.24 (7.22)* | 28.23 (7.03) |

¹All sex- and age- specific z-scores calculated using Frisancho's Comprehensive reference (2008).

*Significant differences between the sexes found using an independent *t*-test, *p*<0.05

** Significant differences between the sexes found using an independent *t*-test, *p*<0.01

^aBMI = Body mass index= (weight in kilograms/stature in metres²). ^bWC= Waist circumference. ^cThe skinfolds summed were triceps and subscapular. ^dTUA= Total upper arm area (Frisancho 2008).

^eUMA= Upper arm muscle area (Frisancho 2008). ^fAFA= Arm fat area (Frisancho 2008). ^gAFI= Arm fat index, percent of the upper arm that is fat (Frisancho 2008). ^h%BF= Percent body fat calculated from an bioelectric impedance equation specific to American Indian children (Lohman et al. 1999)

2. Anthropometry of the mother

These Maya women were very short with high levels of adiposity indicators (Table 5.3). The vast majority (>75%) were stunted (stature <150cm). While the stunted women had significantly smaller measures of linear growth than the non-stunted, there were no significant differences between the two groups in adiposity

indicators. The %BF and WC measurements show this sample to be at a high risk for nutrition-related non-communicable diseases (NR-NCDs).

Table 5.3 Urban Maya mother's descriptive statistics with two stunting classifications, Mean (SD)

| | <150 cm | | <145 cm | | All |
|---|-----------------|----------------|-----------------------------|-----------------------------|---------------|
| | Stunted | Non-stunted | Stunted | Non-stunted | |
| N (%) | 40 (75.5) | 13 (24.5) | 16 (30.2) | 37 (69.8) | 53 (100) |
| Age | 33.74 (6.74) | 36.61 (4.16) | 36.06 (8.52) | 33.74 (5.04) | 34.44 (6.3) |
| Stature (cm) | 145.15 (3.73)** | 152.66 (2.1)** | 141.39 (2.65) ^{††} | 149.41 (3.00) ^{††} | 147 (4.7) |
| Weight (kg) | 61.81 (9.74)* | 69.58 (9.28)* | 59.07 (8.49) [†] | 65.73 (10.21) [†] | 63.72 (10.12) |
| Body mass index (kg/m²) | 29.32 (4.31) | 29.85 (3.84) | 29.55 (4.23) | 29.40 (4.20) | 29.45 (4.17) |
| Sitting height (cm) | 77.97 (2.3)** | 81.32 (2.04)** | 76.21 (2.05) ^{††} | 79.91 (2.04) ^{††} | 78.79 (2.65) |
| Sitting height ratio | 53.73 (1.1) | 53.26 (0.87) | 53.91 (1.33) | 53.49 (0.91) | 53.61 (1.06) |
| Waist circumference (cm) | 87.98 (8.43) | 88.94 (9.2) | 88.06 (7.91) | 88.28 (8.91) | 88.22 (8.55) |
| Triceps skinfold (mm) | 28.42 (8.71) | 33.04 (7.05) | 25.42 (7.56) [†] | 31.34 (8.36) [†] | 29.55 (8.51) |
| Mid-arm circumference (cm) | 30.28 (3.64) | 31.68 (2.44) | 29.45 (3.98) | 31.13 (3.07) | 30.62 (3.42) |
| Total upper arm area (cm²) | 73.98 (17.57) | 80.26 (12.34) | 70.18 (18.62) | 77.83 (15.28) | 75.52 (16.56) |
| Upper arm muscle area (cm²) | 36.82 (8.5) | 36.19 (4.04) | 37.33 (9.27) | 36.38 (6.91) | 36.67 (7.62) |
| Arm fat area (cm²) | 37.16 (13.68) | 44.07 (11.32) | 32.85 (12.53) [†] | 41.45 (13.04) [†] | 38.86 (13.38) |
| Arm fat index (%) AFA) | 49.35 (10.39) | 54.24 (6.67) | 46.24 (9.93) [†] | 52.42 (9.24) [†] | 50.55 (9.79) |
| Body fat percentage | 42.93 (4.19) | 40.49 (4.27) | 44.09 (4.77) | 41.57 (3.91) | 42.33 (4.3) |

*Using an independent *t*-test to compare stunted (stature <150 cm) and non-stunted women (stature ≥150 cm), *p*<0.05

** Using an independent *t*-test to compare stunted (stature <150 cm) and non-stunted women (stature ≥150 cm), *p*<0.01

[†] Using an independent *t*-test to compare stunted (stature <145 cm) and non-stunted women (stature ≥145 cm), *p*<0.05

^{††} Using an independent *t*-test to compare stunted (stature <145 cm) and non-stunted women (stature ≥145 cm), *p*<0.01

3. Anthropometric classifications

a. Stunting

The short stature of these Maya children is severe enough for many to be classified as stunted. However the prevalence of stunting is highly dependent upon the reference and criteria used. Figure 5.1 shows that the prevalence of stunting ranges from 15.5% to 37.9% using the -2 z-scores of the Comprehensive reference

versus the 5th percentile of the WHO reference, respectively. These children seem to cluster around the cut-off points, and therefore the choice of criteria for stunting will have a substantial impact on these results and analyses (Varela Silva et al. 2011).

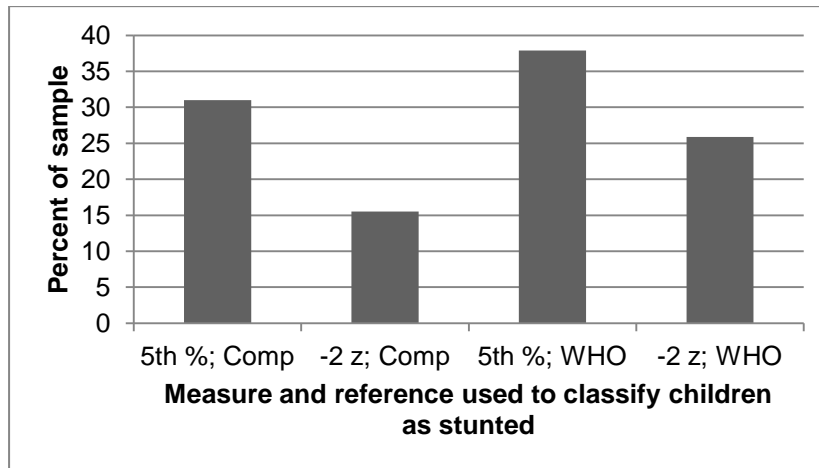


Figure 5.1 Prevalence of childhood stunting in this sample using different criteria and references

¹5th %= Stunted defined as a height-for-age below the 5th percentile on the sex-specific growth charts.

²-2 z= Stunted defined as a height-for-age below -2 z-scores on the sex-specific growth charts

³Comp= Frisancho's Comprehensive reference (Frisancho 2008).

⁴WHO= World Health Organisation references (Frisancho 2008).

The rest of this thesis will use the 5th percentile of the Comprehensive reference as the criteria for childhood stunting. The Comprehensive reference is used as it includes individuals of Mexican descent (Frisancho 2008), see Chapter 3, section E.2.

Using a stunting cut-off of the 5th percentile (150.3 cm) versus 150 cm changed the stunting classification of three women. When mothers are classified as stunted in this thesis, 150 cm will be the most frequently used criteria since it is the most common cut-off for adult women stunting in Mexico (Lara-Esqueda et al. 2004; Varela-Silva et al. 2009). The stunting cut-off of 145 cm will also be used in Chapter 6 to assess the influence of stunting cut-offs on the relationship between BMI and adiposity indicators.

It must be noted that classifying individuals as stunted or non-stunted may give the impression that non-stunted are, therefore, healthy. As the health risks related to short stature are linear (Song and Sung 2008), the classification of stunting

is not as useful in a very short population such as this one because such a high percentage of the sample are clustered around the cut-off points.

b. *Overweight and Obesity*

As can be seen from Figure 5.2 the prevalence of excess weight or adiposity is also highly dependent upon the reference used. The prevalence of high weight-for-age is quite low, which is almost certainly due to the short stature of the sample. The proportion of children classified as OW/OB using the Comprehensive, IOTF or WHO do not vary as much as the rates of stunting do. However there is a 7% difference between the OW/OB rates between the different charts. The WHO reference having the highest prevalence of obesity (34.5%) and IOTF having the lowest (27.5%). Therefore it appears as though when using BMI, the reference source is less important when grouping the OW/OB individuals together than when splitting them into separate categories for overweight and obese.

The adiposity levels of these children are much higher than is expected based upon BMI. Over 80% of the children have body fat percentages in the over-fat or obese range, though roughly 30% of the sample is OW/OB based upon BMI classifications. Thus this sample is at a much higher risk of negative health outcomes based upon their %BF than is expected when only the BMI is examined. The limitations of BMI are discussed more completely in Chapter 6.

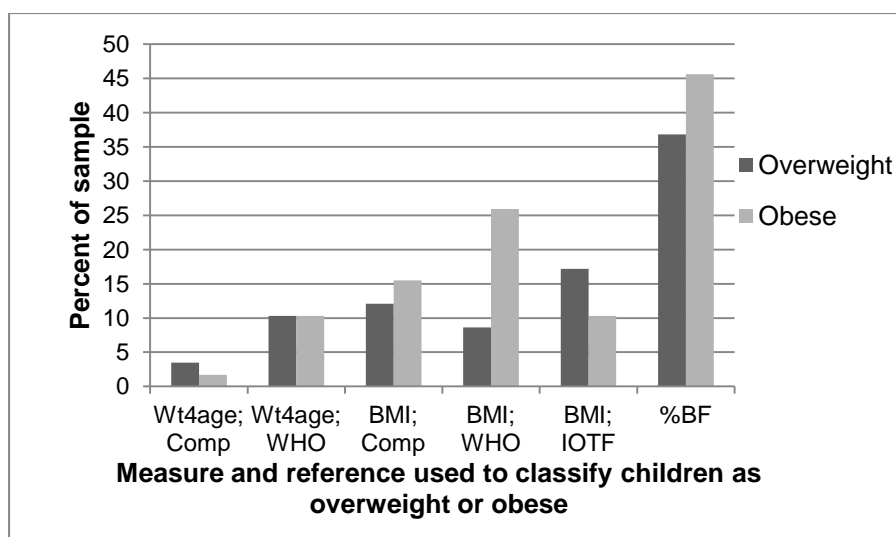


Figure 5.2 Children classified as overweight and obese using different measures and references

¹Overweight based upon sex-specific references (either Comprehensive or WHO) is a weight-for-age or BMI for age that is between the 85th and 94.99th percentile on sex specific references.

²Obese based upon sex-specific references (either Comprehensive or WHO) is a weight-for-age or BMI for age that is at least 95th percentile.

³IOTF= International Obesity Task Force, sex-specific cut-offs scaled to adult cut-offs for each age based upon an international survey (Cole et al. 2000).

⁴Comp= Frisancho's Comprehensive reference (Frisancho 2008).

⁵WHO= World Health Organisation references (Frisancho 2008).

⁶%BF= Percent Body Fat calculated using an equation specific to American Indian children using bioelectrical impedance analysis and anthropometry (Lohman et al. 1999). The cut-off used for over-fat and obese was the 85th and 95th percentile, respectively, of a body fat reference curve based upon White British children defined by McCarthy et al. (2006).

B. Double burden

A detailed description of what double burden is and the current state of the evidence is in Chapter 2, section H. Briefly, double burden is the co-existence of over- and under-nutrition within a population, household or individual. Each level of double burden is likely to have different causes and require different intervention strategies to eliminate each type of malnutrition.

1. Double burden at the sample level

Both over-nutrition and chronic under-nutrition are exceedingly common in this sample with 54% of the sample being stunted and 76% being over-fat (Figure 5.3). The rates of acute over-nutrition increase by over 15% when %BF is used as the criteria versus BMI.

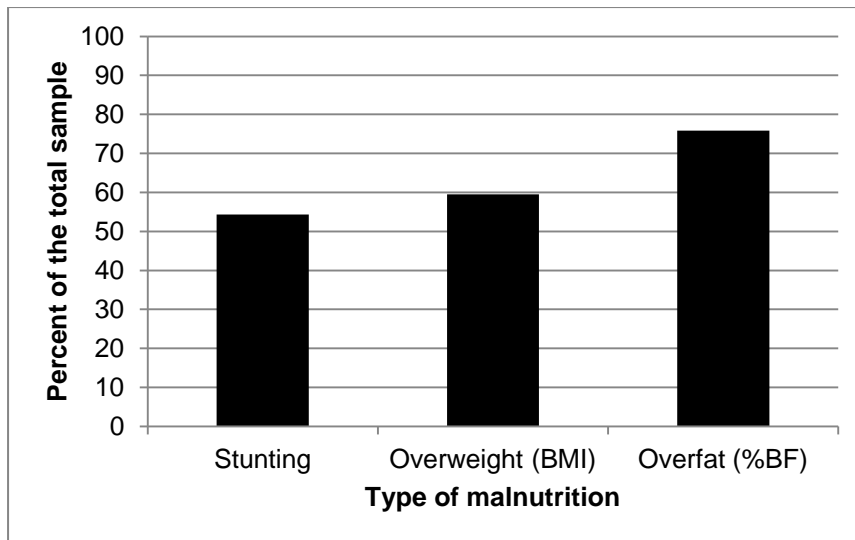


Figure 5.3 Prevalence of double burden within a sample of urban Maya children and mothers. A) Overweight is classified based upon BMI. B) Overfat classified based upon percent body fat.

¹ Stunting is classified as height-for-age below the 5th percentile of Frisancho's Comprehensive growth reference (2008) for children and stature below 150 cm for mothers.

² BMI-for-age z-score above the 85th percentile of Frisancho's Comprehensive growth reference (2008) for children and a BMI above 25 for mothers.

³ Overfat classified as percentage of body fat above the 85th percentile of sex- and age-specific body fat reference curves (McCarthy et al 2005) for children and above 34% for mothers.

2. Double burden at the household level

Double burden households are common in this sample as well. Using the Comprehensive reference for the stunting classification and OW/OB of the mother defined as a BMI > 25, 28% (n=16) of the households had an OW/OB mother and a stunted child (height-for-age < 5th percentile). When childhood stunting was defined as height-for-age < -2 z-scores, the prevalence of double burdened household was reduced to 15.5% (n=9) of the families. When the WHO reference is used to classify the children as stunted, 34.5% (n=20) families are double burdened when child stunting is defined as a height-for-age < 5th percentile. The prevalence reduced to 24.1% (n=14) when -2 z-scores is used for the criteria of childhood stunting.

3. Double burden at the individual level

The prevalence of individual double burden is very high among the mothers, with the prevalence being close to 70% (Figure 5.4.a). The children have a lower prevalence of individual double burden (5%) when over-nutrition is classified by BMI

yet this prevalence increases to 18% when over-nutrition is classified by %BF (Figure 5.4.b).

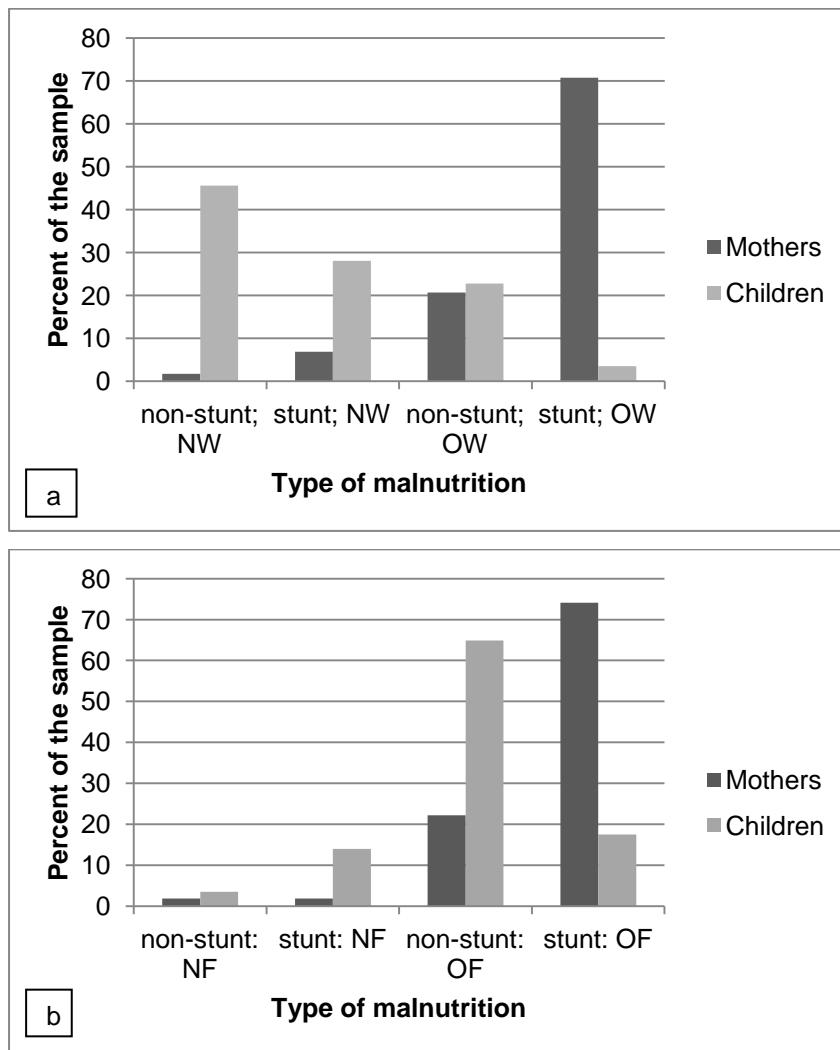


Figure 5.4 Prevalence of individual double burden within the sample. A) Overweight (OW) classified based upon BMI. B) Overfat (OF) classified based upon percent body fat

¹Stunted is defined as stature <150cm for the mothers and a height-for-age z-score below the 5th percentile of Frisancho's Comprehensive reference (2008) for the children.

²BMI used for the classification of overweight, Mother OW defined as a BMI >25, children's OW defined as a BMI-for-age z-score greater than the 85th percentile of Frisancho's Comprehensive reference (2008).

³NF= normal fat

⁴Overfat classified as percentage of body fat above the 85th percentile of sex- and age-specific body fat reference curves (McCarthy et al 2005) for children and above 34% for mothers.

C. *Energy expenditure of the child*

The anthropometry of the children with full Actiheart data was compared with those without complete data (Table 5.4). The two groups were not found to be

significantly different for anthropometry or anthropometric z-scores. The only measure in which the two groups were significantly different was fat free mass (FFM), with the excluded children having significantly more lean mass than those included.

Table 5.4 Comparison of body composition and anthropometric measurements of included and excluded urban Maya children

| | Included | Excluded | All |
|------------------------------------|-----------------|-----------------|---------------|
| N (%) | 33 | 25 | 58 (100) |
| Boys, n (%) | 17 (51.5) | 14 (56.0) | 31 (53.4) |
| Age | 8.34 (0.82) | 8.52 (0.75) | 8.42 (0.79) |
| Stature (cm) | 121.41 (6.96) | 123.08 (6.64) | 122.13 (6.82) |
| Stature z-score¹ | -1.19 (0.92) | -1.10 (0.79) | -1.15 (0.86) |
| Weight (kg) | 26.14 (6.96) | 27.85 (5.76) | 26.87 (6.47) |
| Weight z-score¹ | -0.52 (0.89) | -0.35 (0.88) | -0.45 (0.88) |
| BMI (kg/m²) | 17.48 (2.97) | 18.28 (3.19) | 17.83 (3.07) |
| BMI z-score¹ | 0.45 (0.90) | 0.68 (1.01) | 0.56 (0.95) |
| WC (cm) | 58.90 (7.87) | 60.93 (7.70) | 59.77 (7.80) |
| WC z-score¹ | 0.28 (0.75) | 0.43 (0.84) | 0.34 (0.79) |
| FFM (kg)² | 18.43 (2.96) | 19.58 (2.83) | 18.96 (2.95) |
| FM (kg)² | 7.71 (4.40) | 8.27 (3.32) | 8.00 (3.97) |
| %FFM² | 72.19 (7.53) | 71.38 (6.31) | 71.84 (6.98) |
| %BF² | 27.81 (7.53) | 28.62 (6.31) | 28.23 (7.03) |

¹Age and sex specific z-scores based upon Frisancho's Comprehensive reference (2008).

²Calculated using an equation for American Indian children including bioelectrical impedance and anthropometry

No significant differences found between included and excluded using independent t-tests

Independent *t*-tests were used to determine if these urban Maya pre-pubertal boys and girls had significantly different energy expenditures (Table 5.5). For both RMR and AEE, no significant difference was found in the unadjusted models. Energy expenditure was then adjusted for body size by dividing RMR and also AEE by body weight (kg) and also FFM (kg). When energy expenditure measures were adjusted for body size, the boys had significantly higher energy expenditures than the girls. The boys spent significantly more time, approximately 45 minutes, in moderate and high intensity activities than the girls.

Table 5.5 Energy expenditure variables for urban Mexican Maya children

| | | Stunted | | Non-Stunted | | All |
|--|------------------------------------|---------------------|---------------------|-----------------------|----------------------|----------------------|
| | | Boys | Girls | Boys | Girls | |
| Resting energy expenditure² | kJ/day^b | 4221.00 (315.23) | 3813.17 (384.53) | 4542.25 (437.84) | 4615.90 (615.36) | 4383.33 (547.15) |
| | kJ/kg/day^b | 188.89 (15.02) | 185.99 (18.34) | 176.31 (15.05) | 153.24 (19.44) | 172.98 (21.50) |
| Activity energy expenditure² | kJ/day^b | 2022.20 (257.92) | 2340.50 (425.36) | 3711.67 (955.11) | 3411.60 (799.06) | 3272.27 (879.21) |
| | kJ/kg/day^d | 135.97 (15.19) | 114.26 (13.51) | 143.10 (32.08) | 112.09 (18.51) | 127.38 (26.78) |
| Total energy expenditure² | kJ/day^b | 8060.40 (594.34) | 6859.83 (858.29) | 9,171.00 (1393.84) | 8919.50 (1487.21) | 8506.30 (1485.79) |
| | kJ/kg/day^d | 360.95 (32.28) | 333.62 (30.23) | 354.90 (42.56) | 294.81 (36.81) | 33373 (45.13) |
| MET (min)³ | Light (<3)^{a,c} | 1327.00 (29.65) | 1380.33 (40.81) | 1275.92 (70.63) | 1323.10 (52.93) | 1316.94 (65.46) |
| | Mod-Vig (≥3)^{a,c} | 113.60 (29.84) | 60.00 (40.09) | 164.25 (70.30) | 116.70 (52.74) | 123.21 (65.21) |
| | | | | | | |

¹Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference (2008).

²Calculated in the Actiheart software using simultaneous heart rate and accelerometry data and an external reference curve (Corder 2005).

³Average number of minutes per day spent at each MET level

^aSignificant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.05

^bSignificant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.01

^cSignificant difference found between the sexes using an independent *t*-test, *p*<0.05

^dSignificant difference found between the sexes using an independent *t*-test, *p*<0.01

In order to assess whether this sample exhibits a different level of AEE during the week compared to weekends, paired *t*-tests were performed. No significant differences were found between the weekdays and weekends (Table 5.6). These findings differ to developed countries where children are normally more active at weekends (Ward et al. 2005). This lack of significant differences between AEE on weekdays versus weekend days is likely due to the education structure of Mexico. In Mexico, students spend four hours a day in school, unlike in the U.K. where school hours are much longer. However when AEE was adjusted for body weight, there was a significantly lower AEE on weekends within each sex.

Table 5.6 Activity energy expenditure of urban Maya children calculated using the Actiheart

| | Boys | | | Girls | | | All | | | | |
|-----------|------|-----------------|----------|-------|----------|----------|-----|----------|---------|-----------|---------|
| | n | MJ ¹ | MJ/kg | n | MJ | MJ/kg | n | MJ | MJ/kg | | |
| Week-days | 18 | 3265.72 | 132.01 | 16 | 3027.35 | 122.23 | 34 | 3153.55 | 122.70 | | |
| | | (1270.94) | (29.06)* | | | (975.89) | | (17.27)* | | (1131.19) | (25.91) |
| Weekend | | 3217.78 | 133.76 | | | 2786.53 | | 104.46 | | 3014.84 | 119.97 |
| | | (766.62) | (32.55)* | | (852.36) | (22.55)* | | (825.06) | (31.58) | | |
| Combined | | 3241.75 | 132.89 | | 2906.94 | 108.35 | | 3084.19 | 121.34 | | |
| | | (857.11) | (24.68)* | | (875.35) | (18.03)* | | (869.20) | (24.82) | | |

[†]MJ= megaJoule*Significant difference found between the boys and girls using an independent *t*-test, *p*<0.05.[†]No significant difference found between weekdays and weekends using a paired *t*-test, *p*<0.05.

The next three chapters will present analyses that test hypotheses in order to address the thesis' aims 2-4. The next chapter examines aim 2, assessing the usefulness of BMI as estimate of adiposity in these very short Maya women and children.

Chapter 6. *The usefulness of BMI as an estimate of adiposity in a very short population*

This chapter examines the relationship between BMI and adiposity indicators in a sample of urban Maya women and children and assesses whether stunting status impacts this relationship.

A. *Introduction*

The nutritional double burden (the coexistence of stunting and obesity) is becoming a major health problem in middle income countries (Doak et al. 2005; Doak et al. 2000), such as Mexico (Rivera et al. 2002; Rivera et al. 2004). Linear growth stunting has been linked in theory (Leitch 1951) and in practice (Bogin and Beydoun 2007; Norgan 1994b) with altered body proportions. A proportionately larger torso can artificially increase weight-for-height, due to the higher proportion of mass in the torso compared to the limbs. Thus weight-for-height indices, such as body mass index (BMI), may not be appropriate to detect excess adiposity in stunted populations.

BMI is a very cost effective and quick way of estimating overweight and obesity (OW/OB) (Ellis 2001) and as such is very beneficial in settings with resource and time constraints, such as health researchers and public hospitals in middle income countries. Yet the BMI must be validated for use in estimating adiposity in order to use the tool with confidence in double burdened populations.

1. *Stunting*

Stunting has been described in detail in Chapter 2, section F. Briefly, when the environment experienced by an individual or population is poor, linear growth will falter and if the poor conditions continue, stunting (*i.e.* very short height-for-age) will result. Stunting is very common in much of the developing world, including Latin America (Garrett and Ruel 2005; Van de Poel et al. 2008) and indigenous groups (Azcorra et al 2009; Varela-Silva et al 2012) even in urban areas (Van de Poel et al. 2007) and particularly in the poor (Malina et al. 2008a; Van de Poel et al. 2008).

Middle-income countries are at high risk for the co-existence of over- and under-nutrition (*e.g.* obesity and stunting) (Doak et al. 2005; Doak et al. 2000; Jafar et al. 2008; Popkin 1996). The Maya of southern Merida are a double burdened group (Chapter 5, section B). The areas where stunting and poor conditions are common are rarely equipped to handle both health issues related to both under- and over-nutrition simultaneously (Chapter 2, section H.1). Thus an efficient and cost effective method of assessing malnutrition is necessary.

a. *Stunting cut-offs*

There is no biologic cut-off point between those who are stunted and non-stunted. A negative, linear relationship does appear to exist between adult mortality and stature (Langenberg et al. 2005; Pajunen et al. 2010; Song and Sung 2008), although there is no dramatic increase occurring below any particular stature. Due to the lack of biologically driven cut-offs, the chosen definition of stunting is decided by the researcher based upon epidemiological or statistical evidence and population characteristics. For children, statistical cut-offs based upon reference or standard growth charts are typically used. Childhood stunting is most commonly defined as a height-for-age below -1.65 (Frisancho 2008; Frisancho et al. 2001) or -2 z scores (de Onis and Blossner 2003). One can also use the 5th percentile of a growth chart (-1.65 z-scores), which is one of the least conservative cut-offs, classifying the most individuals as stunted. This sample of Maya children cluster around stunting cut-offs, with the prevalence of stunting in children ranging from 15% to 40% using different reference curves and cut-offs (Chapter 5, section A.3).

For adults, raw statures are preferred, with the cut-off chosen by the researcher. For example, the stunting definitions for adult women are most often set at below, 150 cm in Latin America (Lara-Esqueda et al. 2004; Lopez-Alvarenga et al. 2003; Varela-Silva et al. 2009) and 157 cm (the 10th percentile) in European women (Bosy-Westphal et al. 2009). This study will investigate the influence that different stunting definitions have on the relationship between BMI and adiposity in both women and children.

Likely due, at least in part, to this fluidity of stunting definitions, adult stunting is almost exclusively referred to as 'short stature'. This thesis will refer to adult short stature as stunting because short stature is caused by the same factors as childhood stunting and is, in fact, the end result of childhood stunting. Also using the same terminology for both mothers and children makes the discussion more succinct.

2. *BMI*

Body mass index (BMI) is often used to screen for over-nutrition, particularly in large scale studies in double burdened populations (Doak et al. 2000; Jinabhai et al. 2003; Kimani-Murage et al. 2010). BMI is advantageous when there are time constraints or scarce economic resources as it is very easy to measure, with minimal training, requiring only height and weight measurements (Ellis 2001). It also

correlates fairly well with total body adiposity (Ellis 2001) and body composition (Cameron et al. 2009). Also, adults have shown a direct relationship between BMI and CVD risk factors (Paeratakul et al. 2002), cancer (Bergstrom et al. 2001), mental health (Carpenter et al. 2000; Whitmer et al. 2005), and overall mortality (Flegal et al. 2005). BMI is recommended mainly for large, population based studies (Ellis et al. 1999).

The problem with BMI is that it does not distinguish between fat and fat-free mass. Individuals, both children and adults, with normal or overweight BMIs can also have a percent body fat (%BF) that is over the cut-off for over-fat (Ellis 2001; Frankenfield et al. 2001; Kontogianni et al. 2005). Therefore, when used as a screening tool for obesity, BMI is not precise, with a high specificity (ability to classify normal-fat individuals) but a low sensitivity (ability to classify over-fat individuals) (Ellis 2001). The problems with BMI may be especially true in non-Western populations that may have different body compositions (Norgan 1994b) or fat patterning (Fuke et al. 2007). For example, Australian Aborigines have been found to have higher skinfold thicknesses than predicted by their BMI (Norgan and Jones 1995). Populations who preferentially store fat in their abdomens, such as Asians, are at an increased risk of CVD at lower BMIs than Europeans (Fuke et al. 2007). For these reasons, the Center for Disease Control and Prevention (CDC) recommends BMI to be used in combination with other measures of adiposity, such as skinfolds, bioelectrical impedance (BIA) and dual X-ray absorptiometry (DXA) (CDC 2009).

Populations in transitioning societies are at high risk for obesity and NR-NCDs (Fowler-Brown and Kahwati 2004; Wang et al. 2006) and are often in countries without the ability to properly treat NR-NCDs (Walley et al. 2005). In such conditions, a simple and quick method of assessing nutritional status, such as BMI, is necessary. More complete assessments of NR-NCDs, *i.e.* lipid profiles, are more time consuming and costly than assessing nutritional status and may not be available in low SES communities. As BMI is so commonly used (Hall and Cole 2006; Popkin and Doak 1998; Popkin and Udry 1998) and has recognised problems it is necessary to understand how well BMI relates to other measures of adiposity in all of the populations to which it is applied.

3. *Stunting and BMI*

Stunted populations pose a theoretical problem to the use of BMI. First of all, as height is squared in the denominator of the BMI equation, a reduction in height will lead to an exaggerated increase in BMI, the effect of which will be greater in children than adults (Cameron et al. 2005a). Also body proportions may be altered in stunted individuals (Leitch 1951). Short legs and a long torso will result in a higher body weight due to the size differences between the limbs and torso (Bogin and Beydoun 2007; Norgan 1994b). The higher weight will result in a higher BMI, without any change to body composition that is likely to impact health. BMI has been questioned for use in non-Western populations in part due to differences in body proportions (Norgan 1994a). BMI has been shown to be less effective in predicting CVD risk factors in stunted compared to non-stunted Mexican adults (Lara-Esqueda et al. 2004). So far it is not clear whether the weaknesses of BMI outweigh its usefulness as an indicator of risk for later poor health outcomes in stunted populations at a high risk of later negative health outcomes, including overweight/obesity (OW/OB).

4. *Population*

This study examines a population of low SES Mexican Maya mothers and children living in the south of Merida. The Mexican Maya are adopting behaviours that increase their risk of obesity (Leatherman and Goodman 2005; Leatherman et al. 2000) and have been found to have high rates of stunting and obesity (Azcorra et al. 2009; Varela Silva et al. 2012). For more detail on the Maya, see Chapter 2, section B and Chapters 4 and 5. The high levels of stunting and adoption of behaviours associated with obesity make the Maya a very good population in which to examine the effects of stunting on BMI's ability to predict adiposity.

5. *Aim*

This section of the thesis focuses on Aim 2: to determine whether BMI is an appropriate estimate of adiposity indicators in stunted and non-stunted urban Maya women and children; to assess whether this relationship changes with different definitions of stunting; and also to determine if the power of BMI to predict adiposity indicators is influenced by body proportions.

B. Methods

1. Sample

The study design was cross-sectional. This sample is composed of 58 urban Maya mothers and their children (31 boys), aged 7 to 9 years old, living in Merida, Yucatan, Mexico. For more information on this sample see Chapter 3, section B.

2. Recruitment

Schools were approached that were located in *colonias* in the south of Merida with a high proportion of Maya students. Schools that agreed to participate provided the school lists and from these Maya children were identified by having two Maya surnames. The mothers were then invited to information sessions at their children's schools where the study was explained and information sheets were provided. For more information on the recruitment process, see Chapter 3, section B.3.

Written informed consent was obtained from the mothers and verbal assent from the children. Ethical clearance was obtained from the Loughborough University Ethics Committee in the U.K. and the Bioethical Committee of Cinvestav in Mexico. For more information on the ethics of this study see Chapter 3, section B.2.

3. Measurements

Mothers and children underwent anthropometric measurement (Lohman et al. 1988), which included height, weight, waist circumference (WC), mid-upper arm circumference (MUAC) and skinfolds (triceps for both and sub-scapular, for children only). For more information on the protocol for performing the anthropometric measures, see Chapter 3 section D.1. All calculations for derived variables of body size and composition were performed using Frisancho equations (2008). The sum of two skinfolds (triceps and sub-scapular) (2SF) was calculated for children. Body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in metres. Sitting height ratio (SHR) was calculated by dividing sitting height by stature and multiplying by 100. Upper arm muscle area (UMA) and upper arm fat area (UFA) were calculated. Arm fat index (AFI) was calculated by dividing UFA by upper arm area for an estimation of the percentage of the arm that is fat (see Chapter 3 section E.3 for more details of the calculations).

Body composition was measured for mothers and children using bioelectric impedance analysis (BIA) with a BioScan 916 by Maltron following standard methodology, Chapter 3, section D.2. Percent body fat (%BF) was calculated using the impedance and reactance values with equations specific for American Indian children (Equation 3.8) (Lohman et al. 1999) and for American Indian women (Equation 3.9) (Stolarczyk et al. 1994). Fat free mass as calculated by Equation 3.9 was converted to fat mass and then into %BF. For further information about these equations and their validation, see Chapter 3, Section E.2.

Frisancho's Comprehensive sex- and age-specific reference charts were used to classify children as stunted and OW/OB and calculate z-scores (2008). The Comprehensive reference was chosen as it was created using NHANES III data from the U.S., which includes Mexican-Americans. To the researchers' knowledge, this is the only large scale growth reference that includes Hispanics and, as such, is the most appropriate for use with a Hispanic population such as the Maya. Age- and sex-specific z-scores were calculated for the children's height, weight, WC, BMI, UMA, UFA, AFI and 2SF. No reference values were available for children's %BF as the references begin at age 12. Therefore, the analytic models with %BF included age and sex as predictors as the values were not age and sex standardised.

Participants were classified as stunted using multiple cut-offs for several different analyses to allow comparisons between the effects of the different stunting definitions on the relationship between BMI and adiposity indicators. Children were classified as being stunted if their height-for-age was below the 5th percentile and if their z-scores were below -2. Mothers were classified as stunted if their height was below 145 cm or 150 cm. Stature was also considered as a continuous variable for mothers and children.

4. Statistical analysis

Normality of the distribution of all variables was checked. Adiposity indicators for the children were %BF, and the z-scores for WC, 2SF, UMA, UFA and AFI. The adiposity indicators for the mothers were %BF, WC, UMA, UFA, AFI. Bivariate comparisons of adiposity indicators versus BMI, SHR and stunting were performed using Pearson's correlations, independent *t*-tests and Pearson's chi square. In order to compare with previous studies on BMI and SHR, a simple linear regression was also performed with BMI as the dependent variable and SHR as the predictor.

Multiple linear regressions using the enter method were performed with adiposity indicators as the dependent variables. The model building is shown in Table 6.1. The independent variables examined were BMI, stunting and SHR, which were included in the model in a stepwise fashion. Entering variables into the model in a stepwise fashion shows how the inclusion of variables influences the other variables in the model. This allows for a more complete understanding of the relationship between variables in the model compared to entering all variables in one step. To determine whether BMI interacted with stunting or SHR, an interaction between each was calculated by first centring the BMI and SHR by subtracting the BMI or SHR of each case from the mean. The interaction was calculated using the raw variables for mothers and the z-scores for children. Then the centred BMI was multiplied by stunting or centred SHR to create the interaction variable. Stunting was coded as 1= non-stunted and 2=stunted. The interaction variable was then included in a first step model that also included BMI and stunting or BMI and SHR. The final model included BMI, stunting, SHR and any interaction with a p -value <0.10 in a first step model. Stunting and SHR were both included in the final model to determine whether either had independent effects on adiposity indicators. Significance for the multiple linear regressions was set *a priori* at $p < 0.05$. Bonferroni adjustments were performed to adjust for the multiple comparisons within each model due to the number of steps. Therefore the p value was divided by the number of steps ($0.05 / 6$) resulting in the significance level for the multiple regression model being 0.008.

Table 6.1: Model building for linear regressions on Maya mothers and children assessing the relationship between adiposity indicators and body mass index, stunting and sitting height ratio

| Model | 1 | 2 | 3 | 4 | 5 | 6 |
|-----------------------------------|---|---|---|---|---|---|
| BMI | Y | Y | Y | Y | Y | Y |
| Stunted | | Y | Y | | | Y |
| BMI * Stunting¹ | | | Y | | | ? |
| SHR | | | | Y | Y | Y |
| BMI * SHR² | | | | | Y | ? |

¹Interaction between BMI and stunting

²Interaction between BMI and SHR

Y= yes, included in the model

?= included in the model if borderline significant ($p < 0.10$) in previous models

The dependent variables for the children's models were the adiposity indicators: %BF and the z-scores of WC, 2SF, UMA, UFA and AFI. In the children's %BF model, age and sex were also entered into the model due to the lack of z-

scores available for this age range. The dependent variables for the mother's models were the adiposity indicators: %BF, WC, UMA, UFA and AFI.

Each outcome variable was regressed three times, once with height as a continuous variable and twice with different stunting classifications. For the children the classifications for stunting used were 5th percentile and -2 z-scores using Comprehensive references (Frisancho 2008). The stunting classifications used for mothers were 145 cm and 150 cm.

All analyses were undertaken using SPSS v. 17.0 or SPSS (PASW) v. 18.0. Significance was set *a priori* at $p < 0.05$, although a more conservative cut-off ($p < 0.10$) was used to identify variables for inclusion in later multiple linear regression models from the initial bivariate analyses.

C. Results

One child (sitting height = 53.9 cm; SHR = 44.7) and one mother (sitting height of 43.5cm; SHR = 30.1) were excluded due to biologically improbable sitting height values. Also, four mothers were excluded due to missing BIA data (the equipment was not brought to the field). The final sample sizes were 57 children (31 boys) and 53 mothers.

1. Descriptive statistics

For the children, no significant difference in the prevalence of stunting between the sexes was found ($p > 0.05$). Overall, the children were quite small, with negative mean z-scores for weight, all linear growth measures and the estimate of muscle (Table 6.2). Significant differences between the sexes were found for WC, mid-arm circumference and %BF, with girls being larger and having more body fat than boys. The stunted children were consistently smaller than their non-stunted peers. The only measures in which stunted children were equal to their non-stunted peers were in measures of adiposity, primarily peripheral adiposity (triceps skinfolds, 2SF and AFI).

The mothers were very short, with 75% having heights below 150 cm, 30% having heights below 145 cm and had high levels of adiposity (mean %BF = 42%). Using both definitions of stunting, significant differences only existed between the stunted and non-stunted mothers in stature, body mass, and sitting height (Table

6.3). When stunting was defined as <145 cm, stunted women had significantly lower indicators of arm adiposity.

Table 6.2 Urban Maya children's descriptive statistics, Mean (SD)

| | Boys | Girls | Stunted ² | Non-stunted ² | All |
|---|----------------|---------------|----------------------------|----------------------------|--------------|
| N (%) | 31 (54.4) | 26 (45.6) | 18 (31.6) | 39 (68.4) | 57 (100) |
| Age | 8.23 (0.84) | 8.59 (0.72) | 8.33 (0.85) | 8.47 (0.78) | 8.43 (0.8) |
| Stature z-score¹ | -1.12 (0.86) | -1.20 (0.89) | -2.14 (0.47) ^{††} | -0.7 (0.58) ^{††} | -1.15 (0.87) |
| Weight z-score¹ | -0.64 (0.81) | -0.21 (0.93) | -1.09 (0.7) ^{††} | -0.14 (0.81) ^{††} | 0.44 (0.89) |
| Body mass index z-score¹ | 0.48 (0.88) | 0.67 (1.03) | 0.14 (0.94) [†] | 0.76 (0.9) [†] | 0.56 (0.95) |
| Sitting height z-score¹ | -0.97 (0.80) | -0.51 (0.97) | -1.42 (0.74) ^{††} | -0.45 (0.81) ^{††} | 0.76 (0.9) |
| Sitting height ratio z-score¹ | 1.02 (0.74) | 1.26 (0.89) | 1.57 (0.87) ^{††} | 0.93 (0.72) ^{††} | 1.13 (0.82) |
| Waist circumference z-score¹ | 0.14 (0.70)* | 0.58 (0.84)* | -0.02 (0.66) [†] | 0.51 (0.8) [†] | 0.34 (0.79) |
| Triceps skinfold z-score¹ | 0.45 (0.88) | 0.75 (0.93) | 0.27 (0.86) | 0.73 (0.9) | 0.58 (0.91) |
| Subscapular skinfold z-score^{1*} | 0.61 (0.79) | 0.84 (0.92) | 0.35 (0.8) [†] | 0.89 (0.83) [†] | 0.72 (0.85) |
| Sum of 2 skinfolds (triceps & subscapular) z-score¹ | 0.33 (0.99) | 0.82 (0.84) | 0.25 (0.88) | 0.7 (0.95) | 0.56 (0.95) |
| Mid-arm circumference z-score¹ | -0.56 (0.94)** | 0.74 (1.17)** | -0.39 (1.01) | 0.23 (1.28) | 0.03 (1.23) |
| Total upper arm area z-score¹ | -0.07 (0.92) | 0.46 (1.09) | -0.33 (0.84) [†] | 0.4 (1.03) [†] | 0.17 (1.02) |
| Upper arm muscle area z-score¹ | -1.18 (1.01) | -0.58 (1.59) | -1.53 (1.0) [†] | -0.62 (1.37) [†] | -0.91 (1.33) |
| Arm fat area z-score¹ | 0.54 (1.23) | 0.68 (1.18) | 0.12 (1.07) [†] | 0.82 (1.2) [†] | 0.6 (1.2) |
| Arm fat index (% arm fat area) z-score¹ | 0.91 (0.122) | 1.26 (1.37) | 0.79 (1.2) | 1.2 (1.33) | 1.07 (1.29) |
| Percent body fat | 26.54 (0.65)* | 30.24 (7.22)* | 25.09 (6.72) [†] | 29.68 (6.76) [†] | 28.23 (7.03) |

¹All z-scores calculated using sex- and age-specific Frisancho Comprehensive reference curves (2008)

²Stunted defined as height-for-age less than the 5th percentile of sex-specific Frisancho Comprehensive reference curves (2008).

*Using an independent *t*-test to compare the sexes, *p*<0.05

**Using an independent *t*-test to compare the sexes, *p*<0.01

[†]Using an independent *t*-test to compare stunted and non-stunted children (both sexes combined), *p*<0.05

^{††} Using an independent *t*-test to compare stunted and non-stunted children (both sexes combined), *p*<0.01

Table 6.3 Urban Maya mother's descriptive statistics with two stunting classifications, Mean (SD)

| | <150 cm | | <145 cm | | All |
|---|-----------------|----------------|-----------------------------|-----------------------------|---------------|
| | Stunted | Non-stunted | Stunted | Non-stunted | |
| N (%) | 40 (75.5) | 13 (24.5) | 16 (30.2) | 37 (69.8) | 53 (100) |
| Age | 33.74 (6.74) | 36.61 (4.16) | 36.06 (8.52) | 33.74 (5.04) | 34.44 (6.3) |
| Stature (cm) | 145.15 (3.73)** | 152.66 (2.1)** | 141.39 (2.65) ^{††} | 149.41 (3.00) ^{††} | 147 (4.7) |
| Weight (kg) | 61.81 (9.74)* | 69.58 (9.28)* | 59.07 (8.49) [†] | 65.73 (10.21) [†] | 63.72 (10.12) |
| Body mass index (kg/m²) | 29.32 (4.31) | 29.85 (3.84) | 29.55 (4.23) | 29.40 (4.20) | 29.45 (4.17) |
| Sitting height (cm) | 77.97 (2.3)** | 81.32 (2.04)** | 76.21 (2.05) ^{††} | 79.91 (2.04) ^{††} | 78.79 (2.65) |
| Sitting height ratio | 53.73 (1.1) | 53.26 (0.87) | 53.91 (1.33) | 53.49 (0.91) | 53.61 (1.06) |
| Waist circumference (cm) | 87.98 (8.43) | 88.94 (9.2) | 88.06 (7.91) | 88.28 (8.91) | 88.22 (8.55) |
| Triceps skinfold (mm) | 28.42 (8.71) | 33.04 (7.05) | 25.42 (7.56) [†] | 31.34 (8.36) [†] | 29.55 (8.51) |
| Mid-arm circumference (cm) | 30.28 (3.64) | 31.68 (2.44) | 29.45 (3.98) | 31.13 (3.07) | 30.62 (3.42) |
| Total upper arm area (cm²) | 73.98 (17.57) | 80.26 (12.34) | 70.18 (18.62) | 77.83 (15.28) | 75.52 (16.56) |
| Upper arm muscle area (cm²) | 36.82 (8.5) | 36.19 (4.04) | 37.33 (9.27) | 36.38 (6.91) | 36.67 (7.62) |
| Arm fat area (cm²) | 37.16 (13.68) | 44.07 (11.32) | 32.85 (12.53) [†] | 41.45 (13.04) [†] | 38.86 (13.38) |
| Arm fat index (%) | 49.35 (10.39) | 54.24 (6.67) | 46.24 (9.93) [†] | 52.42 (9.24) [†] | 50.55 (9.79) |
| Body fat percentage | 42.93 (4.19) | 40.49 (4.27) | 44.09 (4.77) | 41.57 (3.91) | 42.33 (4.3) |

*Using an independent *t*-test to compare stunted (stature <150 cm) and non-stunted women (stature ≥150 cm), *p*<0.05

** Using an independent *t*-test to compare stunted (stature <150 cm) and non-stunted women (stature ≥150 cm), *p*<0.01

[†] Using an independent *t*-test to compare stunted (stature <145 cm) and non-stunted women (stature ≥145 cm), *p*<0.05

^{††} Using an independent *t*-test to compare stunted (stature <145 cm) and non-stunted women (stature ≥145 cm), *p*<0.01

2. Children's results

For children, BMI was the largest contributor to each of the adiposity indicator models (Tables 6.4-9). The models using different cut-offs for stunting did not differ in any substantial way. The predictors explained between 30% (UMA) and 84% (WC) of the variance in adiposity indicators. BMI explained the majority of the variance in a positive relationship. The variance explained by the models varied little with the inclusion of covariates other than BMI. Stunting was statistically significant at the *p*<0.05 level in some models but never explained more than 2% of the variance. Stunting was never significant with the Bonferroni adjusted *p* value (*p*<0.008). SHR was not significantly associated with any outcome. Interactions between BMI and stunting and BMI and SHR were rarely significant.

Overall, for the WC models, BMI was a strong predictor that explained almost all of the variance. Stunting may have a small effect on WC but it was inconsistent between models and explains almost none of the variance and was never significant with the Bonferroni adjusted p value ($p < 0.008$). For the %BF models, variance in %BF was explained almost exclusively by BMI, age and sex. Stunting explains a very low amount of the variance ($< 2\%$). For the 2SF, UMA, UFA and AFI models: BMI was the only significant predictive variable. SHR was not significant and explained virtually none of the variance in any of the models.

Stunting and BMI

Table 6.4 Urban Maya children's relationships between percent body fat and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1 ^{1,3} | | Model 2 ^{1,3} | | Model 3 ^{1,3} | | Model 4 ^{1,3} | | Model 5 ^{1,3} | | Model 6 ^{1,3} | |
|---|------------------------|--------|------------------------|--------|------------------------|--------|------------------------|--------|------------------------|--------|------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI z-score² | 6.291 (0.437) | <0.001 | 6.106 (0.462) | <0.001 | 6.769 (1.419) | <0.001 | 6.275 (0.439) | <0.001 | 6.238 (0.454) | <0.001 | 5.988 (0.466) | <0.001 |
| Stunted 5th %^{4,5} | | | -1.139 (0.96) | 0.241 | -1.214 (0.979) | 0.221 | | | | | -1.687 (1.032) | 0.108 |
| BMI z-score * Stunted 5th % | | | | | -0.494 (0.999) | 0.623 | | | | | | |
| SHR z-score² | | | | | | | 0.405 (0.513) | 0.433 | 0.351 (0.538) | 0.517 | 0.751 (0.548) | 0.176 |
| BMI z-score * SHR z-score | | | | | | | | | 0.185 (0.505) | 0.716 | | |
| R² adjusted | 0.807 | | 0.808 | | 0.806 | | 0.806 | | 0.802 | | 0.812 | |
| BMI z-score² | 6.291 (0.437) | <0.001 | 6.167 (0.431) | <0.001 | 6.410 (1.402) | <0.001 | 6.275 (0.439) | <0.001 | 6.238 (0.454) | <0.001 | 6.108 (0.428) | <0.001 |
| Stunted -^{2z2,6} | | | -2.193 (1.134) | 0.059 | -2.234 (1.167) | 0.061 | | | | | -2.719 (1.176) | 0.025 |
| BMI z-score * Stunted -2z | | | | | -0.205 (1.122) | 0.856 | | | | | | |
| SHR z-score² | | | | | | | 0.405 (0.513) | 0.433 | 0.351 (0.538) | 0.517 | 0.766 (0.517) | 0.145 |
| BMI z-score * SHR z-score | | | | | | | | | 0.185 (0.505) | 0.716 | | |
| R² adjusted | 0.807 | | 0.817 | | 0.813 | | 0.806 | | 0.802 | | 0.821 | |
| BMI z-score² | 6.291 (0.437) | <0.001 | 5.883 (0.462) | <0.001 | 5.816 (0.463) | <0.001 | 6.275 (0.439) | <0.001 | 6.238 (0.454) | <0.001 | 5.766 (0.462) | <0.001 |
| Stature z-score² | | | 1.101 (0.504) | 0.034 | 0.985 (0.512) | 0.060 | | | | | 1.334 (0.520) | 0.013 |
| BMI z-score * Stature z-score | | | | | 0.538 (0.451) | 0.238 | | | | | | |
| SHR z-score² | | | | | | | 0.405 (0.513) | 0.433 | 0.351 (0.538) | 0.517 | 0.785 (0.510) | 0.130 |
| BMI z-score * SHR z-score | | | | | | | | | 0.185 (0.505) | 0.716 | | |
| R² adjusted | 0.807 | | 0.820 | | 0.821 | | 0.806 | | 0.802 | | 0.824 | |

¹Percent body fat calculated using American Indian specific formula including bioelectric impedance and anthropometry data (Lohman et al. 1999).

²Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

³All models included age and sex

⁴Non-stunted set as the reference.

⁵Stunted set as height for age z-score <5th percentile

⁶Stunted set as height-for-age z-score ≤-2 z-score

Stunting and BMI

Table 6.5 Urban Maya children's relationships between waist circumference z-score and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | | Model 5 | | Model 6 | |
|---|------------------|--------|-------------------|--------|-------------------|--------|-------------------|--------|-------------------|--------|-------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI z-score¹ | 6.291 (0.437) | <0.001 | 6.106 (0.462) | <0.001 | 6.769 (1.419) | <0.001 | 6.275 (0.439) | <0.001 | 6.238 (0.454) | <0.001 | 5.988 (0.466) | <0.001 |
| Stunted 5th %^{2,3} | | | -1.139 (0.96) | 0.241 | -1.214 (0.979) | 0.221 | | | | | -1.687 (1.032) | 0.108 |
| BMI z-score * Stunted 5th % | | | | | -0.494 (0.999) | 0.623 | | | | | | |
| SHR z-score¹ | | | | | | | 0.405 (0.513) | 0.433 | 0.351 (0.538) | 0.517 | 0.751 (0.548) | 0.176 |
| BMI z-score * SHR z-score | | | | | | | | | 0.185 (0.505) | 0.716 | | |
| R² adjusted | 0.807 | | 0.808 | | 0.806 | | 0.806 | | 0.802 | | 0.812 | |
| BMI z-score¹ | 0.764 (0.045) | <0.001 | .0.759 (0.045) | <0.001 | 0.839 (0.146) | <0.001 | 0.764 (0.045) | <0.001 | 0.777 (0.046) | <0.001 | 0.759 (0.046) | <0.001 |
| Stunted -2z^{1,4} | | | -0.086 (0.117) | 0.465 | -0.101 (0.121) | 0.405 | | | | | -0.089 (0.124) | 0.476 |
| BMI z-score * Stunted -2z | | | | | -0.067 (0.116) | 0.563 | | | | | | |
| SHR z-score¹ | | | | | | | -0.008 (0.053) | 0.881 | 0.010 (0.055) | 0.860 | 0.004 (0.056) | 0.940 |
| BMI z-score * SHR z-score | | | | | | | | | -0.059 (0.050) | 0.241 | | |
| R² adjusted | 0.838 | | 0.837 | | 0.835 | | 0.835 | | 0.836 | | 0.833 | |
| BMI z-score¹ | 0.764 (0.045) | <0.001 | 0.729 (0.048) | <0.001 | 0.724 (0.048) | <0.001 | 0.764 (0.045) | <0.001 | 0.777 (0.046) | <0.001 | 0.726 (0.049) | <0.001 |
| Stature z-score¹ | | | 0.094 (0.053) | 0.078 | 0.084 (0.053) | 0.123 | | | | | 0.101 (0.056) | 0.074 |
| BMI z-score * Stature z-score | | | | | 0.048 (0.045) | 0.293 | | | | | | |
| SHR z-score¹ | | | | | | | -0.008 (0.053) | 0.881 | 0.010 (0.055) | 0.860 | 0.022 (0.054) | 0.692 |
| BMI z-score * SHR z-score | | | | | | | | | -0.059 (0.050) | 0.241 | | |
| R² adjusted | 0.841 | | 0.850 | | 0.853 | | 0.835 | | 0.836 | | 0.842 | |

¹Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

²Non-stunted set as the reference.

³Stunted set as height for age z-score <5th percentile

⁴Stunted set as height-for-age z-score ≤-2 z-score

Stunting and BMI

Table 6.6 Urban Maya children's relationships between sum of 2 skinfolds z-score and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1² | | Model 2² | | Model 3² | | Model 4² | | Model 5² | | Model 6² | |
|---|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI z-score¹ | 0.839 (0.093) | <0.001 | 0.851 (0.077) | <0.001 | 1.029 (0.232) | <0.001 | 0.834 (0.073) | <0.001 | 0.842 (0.075) | <0.001 | 0.836 (0.079) | <0.001 |
| Stunted 5th %^{3,4} | | | 0.077 (0.156) | 0.624 | 0.049 (0.160) | 0.761 | | | | | 0.008 (0.170) | 0.962 |
| BMI z-score * Stunted 5th % | | | | | -0.134 (0.164) | 0.417 | | | | | | |
| SHR z-score¹ | | | | | | | 0.095 (0.085) | 0.265 | 0.107 (0.089) | 0.233 | 0.093 (0.093) | 0.321 |
| BMI z-score * SHR z-score | | | | | | | | | -0.039 (0.081) | 0.634 | | |
| R² adjusted | 0.702 | | 0.698 | | 0.696 | | 0.703 | | 0.699 | | 0.698 | |
| BMI z-score¹ | 0.839 (0.073) | <0.001 | 0.833 (0.074) | <0.001 | 0.902 (0.237) | <0.001 | 0.834 (0.073) | <0.001 | 0.842 (0.075) | <0.001 | 0.822 (0.074) | <0.001 |
| Stunted -2z^{1,5} | | | -0.111 (0.191) | 0.562 | -0.124 (0.197) | 0.531 | | | | | -0.194 (0.199) | 0.333 |
| BMI z-score * Stunted -2z | | | | | -0.058 (0.189) | 0.761 | | | | | | |
| SHR z-score¹ | | | | | | | 0.095 (0.085) | 0.265 | 0.107 (0.089) | 0.233 | 0.122 (0.089) | 0.177 |
| BMI z-score * SHR z-score | | | | | | | | | -0.039 (0.081) | 0.634 | | |
| R² adjusted | 0.702 | | 0.698 | | 0.692 | | 0.703 | | 0.699 | | 0.703 | |
| BMI z-score¹ | 0.839 (0.073) | <0.001 | 0.827 (0.080) | <0.001 | 0.819 (0.080) | 0.821 | 0.834 (0.073) | <0.001 | 0.842 (0.075) | <0.001 | 0.809 (0.081) | <0.001 |
| Stature z-score^{1,6} | | | 0.032 (0.088) | 0.715 | 0.015 (0.089) | 0.865 | | | | | 0.067 (0.091) | 0.464 |
| BMI z-score * Stature z-score | | | | | 0.076 (0.076) | 0.995 | | | | | | |
| SHR z-score¹ | | | | | | | 0.095 (0.085) | 0.265 | 0.107 (0.089) | 0.233 | 0.115 (0.089) | 0.202 |
| BMI z-score * SHR z-score | | | | | | | | | -0.039 (0.081) | 0.634 | | |
| R² adjusted | 0.702 | | 0.697 | | 0.697 | | 0.703 | | 0.699 | | 0.701 | |

¹Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

²The skinfolds summed were sub-scapular and triceps

³Stunted defined as height-for-age <5th percentile of Frisancho's Comprehensive reference (2008).

⁴Non-stunted set as the reference.

⁵Stunting defined as height-for-age below -2 z-scores of Frisancho's Comprehensive reference (2008).

⁶Stature entered as a continuous variable, height-for-age z-scores based upon Frisancho's Comprehensive reference (2008).

Stunting and BMI

Table 6.7 Urban Maya children's relationships between upper arm muscle are z-score and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|---|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI z-score² | 0.794 (0.155) | <0.001 | 0.725 (0.162) | <0.001 | 0.984 (0.488) | 0.049 | 0.799 (0.156) | <0.001 | 0.794 (0.164) | <0.001 | 0.723 (0.166) | <0.001 |
| Stunted 5th %^{3,4} | | | -0.459 (0.327) | 0.167 | -0.499 (0.337) | 0.144 | | | | | -0.468 (0.361) | 0.200 |
| BMI z-score * Stunted 5th % | | | | | -0.195 (0.346) | 0.575 | | | | | | |
| SHR z-score² | | | | | | | -0.090 (0.182) | 0.622 | -0.098 (0.191) | 0.611 | 0.013 (0.198) | 0.948 |
| BMI z-score * SHR z-score | | | | | | | | | 0.025 (0.175) | 0.889 | | |
| R² adjusted | 0.311 | | 0.322 | | 0.314 | | 0.301 | | 0.288 | | 0.310 | |
| BMI z-score² | 0.794 (0.155) | <0.001 | 0.772 (0.157) | <0.001 | 0.709 (0.503) | 0.165 | 0.799 (0.156) | <0.001 | 0.794 (0.162) | <0.001 | 0.776 (0.159) | <0.001 |
| Stunted -2z^{2,5} | | | -0.400 (0.404) | 0.327 | -0.388 (0.418) | 0.357 | | | | | -0.373 (0.428) | 0.388 |
| BMI z-score * Stunted -2z | | | | | 0.053 (0.400) | 0.895 | | | | | | |
| SHR z-score² | | | | | | | -0.090 (0.182) | 0.622 | -0.098 (0.191) | 0.611 | -0.040 (0.191) | 0.837 |
| BMI z-score * SHR z-score | | | | | | | | | 0.025 (0.175) | 0.889 | | |
| R² adjusted | 0.311 | | 0.310 | | 0.298 | | 0.301 | | 0.288 | | 0.298 | |
| BMI z-score² | 0.794 (0.155) | <0.001 | 0.699 (0.168) | <0.001 | 0.720 (0.168) | <0.001 | 0.799 (0.156) | <0.001 | 0.794 (0.162) | <0.001 | 0.702 (0.172) | <0.001 |
| Stature z-score^{2,6} | | | 0.263 (0.184) | 0.158 | 0.305 (0.187) | 0.108 | | | | | 0.258 (0.194) | 0.190 |
| BMI z-score * Stature z-score | | | | | -0.188 (0.159) | 0.242 | | | | | | |
| SHR z-score² | | | | | | | -0.090 (0.182) | 0.622 | -0.098 (0.191) | 0.611 | -0.015 (0.189) | 0.937 |
| BMI z-score * SHR z-score | | | | | | | | | 0.025 (0.175) | 0.889 | | |
| R² adjusted | 0.232 | | 0.348 | | 0.364 | | 0.301 | | 0.288 | | 0.311 | |

¹ Mid-upper arm area – ($\pi \times \text{triceps skinfold}/10$) (Frisancho 2008)

² Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

³ Stunted defined as height-for-age z-score <5th percentile of Frisancho's Comprehensive reference.

⁴ Non-stunted set as the reference.

⁵ Stunting defined as height-for-age below -2 z-scores of Frisancho's Comprehensive reference (2008).

⁶ Stature entered as a continuous variable, height-for-age z-scores based upon Frisancho's Comprehensive reference (2008).

Stunting and BMI

Table 6.8 Urban Maya children's relationships between upper arm fat area and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|---|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI z-score² | 1.060 (0.093) | <0.001 | 1.052 (0.098) | <0.001 | 1.207 (0.296) | <0.001 | 1.052 (0.092) | <0.001 | 1.058 (0.095) | <0.001 | 1.021 (0.098) | <0.001 |
| Stunted 5th %^{3,4} | | | -0.049 (0.199) | 0.805 | -0.073 (0.205) | 0.721 | | | | | -0.192 (0.214) | 0.373 |
| BMI z-score * Stunted 5th % | | | | | -0.116 (0.210) | 0.582 | | | | | | |
| SHR z-score² | | | | | | | 0.152 (0.107) | 0.162 | 0.160 (0.112) | 0.158 | 0.194 (0.117) | 0.103 |
| BMI z-score * SHR z-score | | | | | | | | | -0.029 (0.103) | 0.777 | | |
| R² adjusted | 0.699 | | 0.694 | | 0.690 | | 0.705 | | 0.699 | | 0.703 | |
| BMI z-score² | 1.060 (0.093) | <0.001 | 1.050 (0.094) | <0.001 | 1.165 (0.301) | <0.001 | 1.052 (0.092) | <0.001 | 1.058 (0.095) | <0.001 | 1.033 (0.093) | <0.001 |
| Stunted 2z^{2,5} | | | -0.182 (0.242) | 0.456 | -0.203 (0.250) | 0.419 | | | | | -0.314 (0.250) | 0.213 |
| BMI z-score * Stunted 2z | | | | | -0.096 (0.239) | 0.689 | | | | | | |
| SHR z-score² | | | | | | | 0.152 (0.107) | 0.162 | 0.160 (0.112) | 0.158 | 0.194 (0.112) | 0.087 |
| BMI z-score * SHR z-score | | | | | | | | | -0.029 (0.103) | 0.777 | | |
| R² adjusted | 0.699 | | 0.697 | | 0.692 | | 0.705 | | 0.699 | | 0.708 | |
| BMI z-score² | 1.060 (0.093) | <0.001 | 1.055 (0.102) | <0.001 | 1.049 (0.103) | <0.001 | 1.052 (0.092) | <0.001 | 1.058 (0.095) | <0.001 | 1.027 (0.102) | <0.001 |
| Stature z-score^{2,6} | | | 0.015 (0.112) | 0.897 | 0.004 (0.115) | 0.976 | | | | | 0.067 (0.116) | 0.564 |
| BMI z-score * Stature z-score | | | | | 0.050 (0.097) | 0.613 | | | | | | |
| SHR z-score² | | | | | | | 0.152 (0.107) | 0.162 | 0.160 (0.112) | 0.158 | 0.171 (0.113) | 0.134 |
| BMI z-score * SHR z-score | | | | | | | | | -0.029 (0.103) | 0.777 | | |
| R² adjusted | 0.699 | | 0.699 | | 0.689 | | 0.705 | | 0.699 | | 0.701 | |

¹ Upper arm area – upper arm muscle area (Frisancho 2008)

² Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

³ Stunted defined as height-for-age z-score <5th percentile of Frisancho's Comprehensive reference.

⁴ Non-stunted set as the reference.

⁵ Stunting defined as height-for-age below -2 z-scores of Frisancho's Comprehensive reference (2008).

⁶ Stature entered as a continuous variable, height-for-age z-scores based upon Frisancho's Comprehensive reference (2008).

Stunting and BMI

Table 6.9 Urban Maya children's relationships between arm fat index and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|---|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI z-score² | 0.804 (0.148) | <0.001 | 0.819 (0.157) | <0.001 | 0.765 (0.476) | 0.114 | 0.793 (0.148) | <0.001 | 0.797 (0.153) | <0.001 | 0.781 (0.159) | <0.001 |
| Stunted 5th %^{3,4} | | | 0.102 (0.318) | 0.749 | 0.111 (0.329) | 0.738 | | | | | -0.070 (0.346) | 0.838 |
| BMI z-score * Stunted 5th % | | | | | 0.041 (0.337) | 0.904 | | | | | | |
| SHR z-score² | | | | | | | 0.219 (0.172) | 0.207 | 0.225 (0.180) | 0.217 | 0.235 (0.189) | 0.221 |
| BMI z-score * SHR z-score | | | | | | | | | -0.020 (0.165) | 0.903 | | |
| R² adjusted | 0.336 | | 0.325 | | 0.313 | | 0.344 | | 0.332 | | 0.332 | |
| BMI z-score² | 0.804 (0.148) | <0.001 | 0.804 (0.151) | <0.001 | 0.744 (0.485) | 0.131 | 0.793 (0.148) | <0.001 | 0.797 (0.153) | <0.001 | 0.782 (0.151) | <0.001 |
| Stunted 2z^{2,5} | | | <0.001 (0.390) | 0.999 | 0.012 (0.403) | 0.977 | | | | | -0.164 (0.407) | 0.688 |
| BMI z-score * Stunted 2z | | | | | 0.050 (0.386) | 0.130 | | | | | | |
| SHR z-score² | | | | | | | 0.219 (0.172) | 0.207 | 0.225 (0.180) | 0.217 | 0.424 (0.182) | 0.190 |
| BMI z-score * SHR z-score | | | | | | | | | -0.020 (0.165) | 0.903 | | |
| R² adjusted | 0.336 | | 0.324 | | 0.311 | | 0.344 | | 0.332 | | 0.333 | |
| BMI z-score² | 0.804 (0.148) | <0.001 | 0.829 (0.163) | <0.001 | 0.821 (0.165) | <0.001 | 0.793 (0.148) | <0.001 | 0.797 (0.153) | <0.001 | 0.370 (0.317) | 0.249 |
| Stature z-score^{2,6} | | | -0.069 (0.179) | 0.703 | -0.083 (0.183) | 0.652 | | | | | 0.793 (0.165) | <0.001 |
| BMI z-score * Stature z-score | | | | | 0.066 (0.156) | 0.674 | | | | | | |
| SHR z-score² | | | | | | | 0.219 (0.172) | 0.207 | 0.225 (0.180) | 0.217 | 0.219 (0.182) | 0.234 |
| BMI z-score * SHR z-score | | | | | | | | | -0.020 (0.165) | 0.903 | | |
| R² adjusted | 0.336 | | 0.326 | | 0.315 | | 0.344 | | 0.332 | | 0.367 | |

¹(Upper arm fat area/ total upper arm area) * 100 (Frisancho 2008)

²Age and sex specific z-scores by Frisancho's Comprehensive reference (2008)

³Stunted defined as height-for-age z-score <5th percentile of Frisancho's Comprehensive reference.

⁴Non-stunted set as the reference.

⁵Stunting defined as height-for-age below -2 z-scores of Frisancho's Comprehensive reference (2008).

⁶Stature entered as a continuous variable, height-for-age z-scores based upon Frisancho's Comprehensive reference (2008).

3. *Mother's results*

For mothers, BMI was the strongest predictor in every model, explaining the majority of the variance in a positive relationship (Tables 6.10-14). BMI was the only variable to predict adiposity outcomes at the Bonferroni adjusted p value ($p < 0.008$). The models with different cut-offs for adult stunting did not differ in any substantial way. The models explained between 11% and 79% of the variance. The predictors explained the most variance in WC (79%), while UMA and AFI had the least variance explained (11% and 19%, respectively). The variance explained by the models varied little with the inclusion of covariates other than BMI, with the exception of the UFA model. Stunting was significant in some models but never explained more than 2% of the variance in the outcomes examined. In the simple regression of SHR to BMI (Table 6.14), SHR did significantly predict BMI but explained less than 9% of the variance. SHR did not impact any model though it did significantly interact with BMI in the simple %BF models, but was attenuated in the final models with the inclusion of stunting.

Overall, for the WC models, BMI was a strong positive predictor that explained almost all of the variance. SHR may have an effect on WC but it is inconsistent between models and explains almost none of the variance. For the %BF models, a relatively low amount of the variance in %BF was explained (about 30%), almost exclusively by BMI. Stature and %BF have a significant negative relationship. There appears to be an interaction between BMI and SHR in the %BF models, as the interaction is always positive and significant in the simple model. However the interaction was consistently attenuated in the final model with the inclusion of stunting. For the UMA models, a low amount of variance is explained and only BMI significantly contributes to the model. For the UFA models, a medium amount of variance is explained, mostly by BMI. Stature and UFA have a significant, inverse relationship which explains approximately 10% of the variance. For the AFI model, a low amount of variance is explained (approximately 20%) mostly by BMI. There is also a trend for stature and AFI to have a positive relationship with one another.

Stunting and BMI

Table 6.10 Urban Maya mother's relationship between percent body fat and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|----------------------------------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI | 0.585 (0.119) | <0.001 | 0.601 (0.113) | <0.001 | 0.718 (0.532) | 0.183 | 0.626 (0.125) | <0.001 | 0.639 (0.125) | <0.001 | 0.670 (0.117) | <0.001 |
| Stunted 150cm² | | | 2.758 (1.087) | 0.014 | 2.762 (1.098) | 0.015 | | | | | 2.635 (1.138) | 0.025 |
| BMI * Stunted 150cm | | | | | -0.650 (0.288) | 0.823 | | | | | | |
| SHR | | | | | | | -0.499 (0.494) | 0.318 | -0.231 (0.490) | 0.639 | -0.569 (0.492) | 0.253 |
| BMI * SHR | | | | | | | | | 0.247 (0.110) | 0.030 | 0.167 (0.111) | 0.141 |
| R² adjusted | 0.309 | | 0.376 | | 0.364 | | 0.309 | | 0.361 | | 0.413 | |
| BMI | 0.586 (0.119) | <0.001 | 0.581 (0.114) | <0.001 | 0.484 (0.346) | 0.168 | 0.626 (0.125) | <0.001 | 0.639 (0.125) | <0.001 | 0.647 (0.117) | <0.001 |
| Stunted 145cm² | | | 2.424 (1.025) | 0.022 | 2.437 (1.036) | 0.023 | | | | | 2.248 (1.053) | 0.038 |
| BMI * Stunted 145cm | | | | | 0.075 (0.252) | 0.768 | | | | | | |
| SHR | | | | | | | -0.499 (0.494) | 0.318 | -0.231 (0.490) | 0.639 | -0.498 (0.490) | 0.315 |
| BMI * SHR | | | | | | | | | 0.247 (0.110) | 0.030 | 0.182 (0.111) | 0.107 |
| R² adjusted | 0.309 | | 0.366 | | 0.354 | | 0.309 | | 0.361 | | 0.404 | |
| BMI | 0.586 (0.119) | <0.001 | 0.600 (0.114) | <0.001 | 0.599 (0.115) | <0.001 | 0.626 (0.125) | <0.001 | 0.639 (0.125) | <0.001 | 0.671 (0.117) | <0.001 |
| Stature | | | -0.238 (0.101) | 0.023 | -0.235 (0.103) | 0.027 | | | | | -0.232 (0.107) | 0.035 |
| BMI * Stature | | | | | -0.008 (0.025) | 0.741 | | | | | | |
| SHR | | | | | | | -0.499 (0.494) | 0.318 | -0.231 (0.490) | 0.639 | -0.590 (0.501) | 0.244 |
| BMI * SHR | | | | | | | | | 0.247 (0.110) | 0.030 | 0.170 (0.112) | 0.135 |
| R² adjusted | 0.309 | | 0.365 | | 0.354 | | 0.309 | | 0.361 | | 0.406 | |

¹Percent body fat calculated using an American Indian women predictive equation using bioelectric impedance (Stolarczyk et al 1994)

²Non-stunted set as the reference

Stunting and BMI

Table 6.11 Urban Maya mother's relationship between waist circumference and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | | Model 5 | | Model 6 | |
|----------------------------------|------------------|--------|-------------------|--------|-------------------|--------|-------------------|--------|-------------------|--------|-------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI | 1.815 (0.133) | <0.001 | 1.815 (0.134) | <0.001 | 2.594 (0.621) | 0.983 | 1.898 (0.137) | <0.001 | 1.890 (0.137) | <0.001 | 1.905 (0.139) | <0.001 |
| Stunted 150cm¹ | | | 0.003 (1.290) | 0.998 | 0.027 (1.282) | 0.983 | | | | | 0.550 (1.288) | 0.671 |
| BMI * Stunted 150cm | | | | | -0.432 (0.336) | 0.205 | | | | | | |
| SHR | | | | | | | -1.022 (0.539) | 0.064 | -1.171 (0.554) | 0.040 | -1.075 (0.557) | 0.059 |
| BMI * SHR | | | | | | | | | 0.137 (0.125) | 0.277 | | |
| R² adjusted | 0.781 | | 0.777 | | 0.780 | | 0.792 | | 0.793 | | 0.789 | |
| BMI | 1.815 (0.133) | <0.001 | 1.816 (0.134) | <0.001 | 2.313 (0.400) | <0.001 | 1.898 (0.137) | <0.001 | 1.890 (0.137) | <0.001 | 1.897 (0.138) | <0.001 |
| Stunted 145cm¹ | | | -0.499 (1.206) | 0.681 | -0.567 (1.198) | 0.638 | | | | | -0.085 (1.200) | 0.944 |
| BMI * Stunted 145cm | | | | | -0.383 (0.291) | 0.194 | | | | | | |
| SHR | | | | | | | -1.022 (0.539) | 0.064 | -1.171 (0.554) | 0.040 | -1.015 (0.554) | 0.073 |
| BMI * SHR | | | | | | | | | 0.137 (0.125) | 0.277 | | |
| R² adjusted | 0.781 | | 0.778 | | 0.781 | | 0.792 | | 0.793 | | 0.788 | |
| BMI | 1.815 (0.133) | <0.001 | 1.809 (0.133) | <0.001 | 1.814 (0.130) | <0.001 | 1.898 (0.137) | <0.001 | 1.890 (0.137) | <0.001 | 1.875 (0.137) | <0.001 |
| Stature | | | 0.107 (0.118) | 0.372 | 0.085 (0.116) | 0.465 | | | | | 0.048 (0.118) | 0.684 |
| BMI * Stature | | | | | 0.057 (0.029) | 0.053 | | | | | 0.048 (0.029) | 0.109 |
| SHR | | | | | | | -1.022 (0.539) | 0.064 | -1.171 (0.554) | 0.040 | -0.740 (0.568) | 0.199 |
| BMI * SHR | | | | | | | | | 0.137 (0.125) | 0.277 | | |
| R² adjusted | 0.781 | | 0.781 | | 0.793 | | 0.792 | | 0.793 | | 0.796 | |

¹Non-stunted set as the reference

Stunting and BMI

Table 6.12 Urban Maya mother's relationship between upper arm muscle area and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|------------------------------------|----------------------------|-------|----------------------------|-------|----------------------------|-------|----------------------------|-------|----------------------------|-------|----------------------------|-------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI | 0.733 (0.234) | 0.003 | 0.738 (0.236) | 0.003 | -0.277 (1.101) | 0.802 | 0.734 (0.250) | 0.005 | 0.720 (0.249) | 0.006 | 0.749 (0.253) | 0.005 |
| Stunted (150cm)² | | | 1.022 (2.271) | 0.655 | 0.990 (2.274) | 0.665 | | | | | 1.086 (2.352) | 0.646 |
| BMI * Stunted (150cm) | | | | | 0.563 (0.596) | 0.945 | | | | | | |
| SHR | | | | | | | -0.023 (0.984) | 0.981 | -0.323 (1.010) | 0.751 | -0.127 (1.017) | 0.901 |
| BMI * SHR | | | | | | | | | -0.277 (0.227) | 0.229 | | |
| R² adjusted | 0.145 | | 0.131 | | 0.129 | | 0.127 | | 0.136 | | 0.113 | |
| BMI | 0.733 (0.234) | 0.003 | 0.731 (0.236) | 0.003 | 0.667 (0.718) | 0.358 | 0.734 (0.250) | 0.005 | 0.720 (0.249) | 0.006 | 0.739 (0.252) | 0.005 |
| Stunted (145cm)² | | | 0.841 (2.127) | 0.694 | 0.850 (2.150) | 0.694 | | | | | 0.882 (2.187) | 0.689 |
| BMI * Stunted (145cm) | | | | | 0.050 (0.522) | 0.925 | | | | | | |
| SHR | | | | | | | -0.023 (0.984) | 0.981 | -0.323 (1.010) | 0.751 | -0.100 (1.010) | 0.922 |
| BMI * SHR | | | | | | | | | -0.277 (0.227) | 0.229 | | |
| R² adjusted | 0.145 | | 0.130 | | 0.113 | | 0.127 | | 0.136 | | 0.113 | |
| BMI | 0.733 (0.234) | 0.003 | 0.736 (0.237) | 0.003 | 0.734 (0.239) | 0.003 | 0.734 (0.250) | 0.005 | 0.720 (0.249) | 0.006 | 0.744 (0.254) | 0.005 |
| Stature | | | -0.054 (0.210) | 0.799 | -0.047 (0.213) | 0.828 | | | | | -0.059 (0.219) | 0.790 |
| BMI * Stature | | | | | -0.019 (0.053) | 0.715 | | | | | | |
| SHR | | | | | | | -0.023 (0.984) | 0.981 | -0.323 (1.010) | 0.751 | -0.093 (1.027) | 0.928 |
| BMI * SHR | | | | | | | | | -0.277 (0.227) | 0.229 | | |
| R² adjusted | 0.145 | | 0.129 | | 0.113 | | 0.127 | | 0.136 | | 0.111 | |

¹Upper arm muscle area= [upper arm circumference – (triceps skinfold x π)]² / (4 x π)

²Non-stunted set as the reference

Stunting and BMI

Table 6.13 Urban Maya mother's relationship between upper arm fat area and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|------------------------------------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|----------------------------|--------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI | 2.408 (0.294) | <0.001 | 2.375 (0.288) | <0.001 | 2.884 (1.351) | 0.038 | 2.574 (0.307) | <0.001 | 2.577 (0.310) | <0.001 | 2.509 (0.303) | <0.001 |
| Stunted (150cm)² | | | -5.658 (2.766) | 0.046 | -5.642 (2.790) | 0.049 | | | | | -4.843 (2.815) | 0.092 |
| BMI * Stunted (150cm) | | | | | -0.282 (0.731) | 0.702 | | | | | | |
| SHR | | | | | | | -2.061 (1.210) | 0.095 | -2.000 (1.260) | 0.119 | -1.600 (1.217) | 0.195 |
| BMI * SHR | | | | | | | | | 0.056 (0.284) | 0.843 | | |
| R² adjusted | 0.556 | | 0.582 | | 0.575 | | 0.572 | | 0.563 | | 0.588 | |
| BMI | 2.408 (0.294) | <0.001 | 2.424 (0.264) | <0.001 | 2.898 (0.799) | 0.001 | 2.574 (0.307) | <0.001 | 2.577 (0.310) | <0.001 | 2.531 (0.278) | <0.001 |
| Stunted (145cm)² | | | -8.977 (2.377) | <0.001 | -9.042 (2.373) | <0.001 | | | | | -8.436 (2.410) | 0.0014 |
| BMI * Stunted (145cm) | | | | | -0.365 (0.581) | 0.522 | | | | | | |
| SHR | | | | | | | -2.061 (1.210) | 0.095 | -2.000 (1.260) | 0.119 | -1.327 (1.113) | 0.239 |
| BMI * SHR | | | | | | | | | 0.056 (0.284) | 0.843 | | |
| R² adjusted | 0.556 | | 0.647 | | 0.643 | | 0.572 | | 0.563 | | 0.560 | |
| BMI | 2.408 (0.294) | <0.001 | 2.408 (0.296) | <0.001 | 2.355 (0.255) | <0.001 | 2.574 (0.307) | <0.001 | 2.577 (0.310) | <0.001 | 2.430 (0.272) | <0.001 |
| Stature | | | 0.982 (0.227) | <0.001 | 0.958 (0.227) | <0.001 | | | | | 0.930 (0.235) | <0.001 |
| BMI * Stature | | | | | 0.064 (0.056) | 0.262 | | | | | | |
| SHR | | | | | | | -2.061 (1.210) | 0.095 | -2.000 (1.260) | 0.119 | -0.958 (1.100) | 0.388 |
| BMI * SHR | | | | | | | | | 0.056 (0.284) | 0.843 | | |
| R² adjusted | 0.556 | | 0.690 | | 0.672 | | 0.572 | | 0.563 | | 0.669 | |

¹Upper arm fat area= Total upper arm area – upper arm muscle area

²Non-stunted set as the reference

Stunting and BMI

Table 6.14 Urban Maya mother's relationship between arm fat index and body mass index, stunting and sitting height ratio in multiple linear regressions using multiple stunting criteria

| | Model 1¹ | | Model 2¹ | | Model 3¹ | | Model 4¹ | | Model 5¹ | | Model 6¹ | |
|------------------------------------|----------------------------|--------|----------------------------|--------|----------------------------|-------|----------------------------|-------|----------------------------|-------|----------------------------|-------|
| | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p | B (SE) | p |
| BMI | 1.041 (0.294) | <0.001 | 1.017 (0.291) | 0.001 | 1.496 (1.364) | 0.278 | 1.128 (0.311) | 0.001 | 1.148 (0.309) | 0.001 | 1.075 (0.311) | 0.001 |
| Stunted (150cm)² | | | -4.356 (2.793) | 0.125 | -4.341 (2.818) | 0.130 | | | | | -4.002 (2.883) | 0.171 |
| BMI * Stunted (150cm) | | | | | -0.266 (0.739) | 0.720 | | | | | | |
| SHR | | | | | | | -1.076 (1.227) | 0.385 | -0.672 (1.256) | 0.595 | -0.695 (1.246) | 0.580 |
| BMI * SHR | | | | | | | | | 0.373 (0.283) | 0.194 | | |
| R² adjusted | 0.181 | | 0.204 | | 0.190 | | 0.178 | | 0.190 | | 0.193 | |
| BMI | 1.041 (0.294) | <0.001 | 1.053 (0.280) | <0.001 | 0.765 (0.851) | 0.373 | 1.128 (0.311) | 0.001 | 1.148 (0.309) | 0.001 | 1.097 (0.298) | 0.001 |
| Stunted (145cm)² | | | -6.343 (2.522) | 0.015 | -6.303 (2.547) | 0.017 | | | | | -6.121 (2.588) | 0.022 |
| BMI * Stunted (145cm) | | | | | 0.222 (0.619) | 0.721 | | | | | | |
| SHR | | | | | | | -1.076 (1.227) | 0.385 | -0.672 (1.256) | 0.595 | -0.543 (1.196) | 0.652 |
| BMI * SHR | | | | | | | | | 0.373 (0.283) | 0.194 | | |
| R² adjusted | 0.181 | | 0.259 | | 0.246 | | 0.178 | | 0.190 | | 0.247 | |
| BMI | 1.041 (0.294) | <0.001 | 1.005 (0.281) | 0.001 | 1.006 (0.284) | 0.001 | 1.128 (0.311) | 0.001 | 1.148 (0.309) | 0.001 | 1.038 (0.306) | 0.001 |
| Stature | | | 0.608 (0.250) | 0.019 | 0.607 (0.254) | 0.021 | | | | | 0.587 (0.264) | 0.031 |
| BMI * Stature | | | | | 0.001 (0.063) | 0.019 | | | | | -0.004 (0.065) | 0.957 |
| SHR | | | | | | | -1.076 (1.227) | 0.385 | -0.672 (1.256) | 0.595 | -0.395 (1.268) | 0.756 |
| BMI * SHR | | | | | | | | | 0.373 (0.283) | 0.194 | | |
| R² adjusted | 0.197 | | 0.253 | | 0.238 | | 0.178 | | 0.190 | | 0.224 | |

¹Arm fat index= (Upper arm fat area/ total upper arm area) x 100

²Non-stunted set as the reference

D. Discussion

In this sample of Maya children, BMI significantly predicted and explained a high proportion of the variance in measures of visceral and total body adiposity but not peripheral adiposity. The significance and beta of BMI were not affected by the stunting status of the child, whether stunting was added as a mediator or moderator in the models. SHR was neither significant nor altered the predictive power of BMI. Using different cut-offs for stunting (*i.e.* z-scores versus percentiles) did not alter the findings. BMI therefore appears to be an appropriate tool to estimate total and central adiposity in this population of 7-9 year old children regardless of SHR.

In mothers, BMI significantly predicted visceral adiposity but not peripheral or total body adiposity. Stunting independently predicted a higher %BF and did not change the predictive power of BMI in any model. SHR was neither significant nor altered the predictive power of BMI. Using different cut-offs for stunting (*i.e.* 145 cm versus 150 cm) did not alter the findings. BMI therefore appears to only be appropriate for use in these short adult women to predict visceral adiposity.

1. Predictive power of BMI

a. Percent body fat

Maternal %BF (assessed by BIA) was not well predicted by BMI as it only explained 30% of the variance. This sample's BMI values mostly fall within the range of BMIs (mean = 29 kg/m²) that have been previously shown to be the worst at classifying %BF (25-29.99 kg/m²) (Ellis 2001). Therefore, it is not surprising that BMI was such a poor predictor of %BF in this population. The %BF of these women was very high (mean = 42%) and as such they are at a very high risk of CVD (Poirier et al. 2006). Body composition, especially adiposity, appears to be more strongly related to CVD risk factors than BMI in adult women (Shea et al. 2011). As such, the results of this research suggest that it may not be appropriate to use BMI to predict %BF and consequent CVD risk in this population of Maya women and a more direct estimate of body fat would be advisable.

In contrast to the findings in the mothers, for children in this study, BMI was highly associated with %BF, explaining just over 80% of the variance in %BF. Similar studies have been done in children to determine the relationship between BMI and

%BF. Ellis and colleagues (1999) compared BMI to body fat estimation by DXA in Black, Hispanic and non-Hispanic White children aged 3 to 18 years in the U.S. They found the two estimates of body fat to be significantly correlated, with the relationship stronger in the girls ($r^2=0.70$) than boys ($r^2=0.34$).

Hoffman and colleagues (2006) performed a case-control study of stunted and non-stunted children in the shantytowns of São Paulo, Brazil (Hoffman et al. 2006). They found that BMI significantly predicted %BF but the model explained much less of the variance (12.5%) than this study (80.7%, Table 6.3, model 1). The large difference in the amount of variance explained may be due in large part to methodological differences and the differing levels of adiposity found. Hoffman's %BF estimates are from a more precise method ($H_2^{18}O$ dilution) than the BIA estimations used in this study. The correlation between %BF as determined by $H_2^{18}O$ and BIA has been found to be 84% in non-over-fat pre-menarcheal girls in control laboratory conditions (Bandini et al. 1997). The BIA measurements taken in this study were not under tightly controlled laboratory conditions and therefore contain more error. Thus some, but not all, of the differences between the two studies' findings may be attributable to methodological differences.

This large difference may also be due to the low levels of adiposity found by Hoffman. The living conditions of participants in this study may have led to a truly higher %BF. It is well established that BMI better predicts %BF at higher BMIs (Ellis 2001; Frankenfield et al. 2001). The mean %BF for Hoffman's study was found to be 0.559 z-scores below this study. However neither sample had high levels of OW/OB. It is possible that the differences in adiposity levels are due to more favourable living conditions in this study's Maya sample. All of our families had permanent housing and access to running water inside their property and almost all drank purified water (Chapter 4 section D.1). Hoffman's sample was drawn from shantytowns, where conditions are typically much worse. Therefore, when measuring Maya children who are poor, but who have access to basic sanitation and are at risk for double burden, BMI may be a more appropriate tool for estimating body fat compared to groups living in worse conditions.

However BMI must be interpreted with caution as certain populations may have an increased BMI due to lean mass, not body fat (Wells et al 2002). Trowbridge and colleagues' examined Peruvian children aged 6 months to 5 years, who had a low mean stature (15th percentile) and high mean weight-for-height (60th percentile)

(1987). Skinfold thicknesses (triceps and sub-scapular) and AFI were below the reference median but UMA was more similar to the reference median. Thus, it did not appear as though the relatively high weight-for-height in the Peruvian children was due to increased adiposity, but lean tissue.

However the limitations of BMI (Ellis 2001) suggest that it be used in conjunction with other measures to gain an accurate and complete picture of health.

2. *Waist circumference*

The literature suggests that abdominal adipose tissue increases the risk of CVD. The abdominal adipose tissue is primarily stored around the intestines and, compared to subcutaneous adipose tissue, is more metabolically active and labile (Gabrielsson et al. 2003; Lau et al. 2005). Therefore, abdominal adiposity needs to be monitored, especially in adults since the body tends to store more adipose tissue in the abdomen with age (Wells et al. 2010). The best, cost effective and simple measure of abdominal adiposity is WC (Rankinen et al. 1999).

Waist circumference was the adiposity indicator best predicted by BMI with nearly 80% of the variance in WC explained by BMI alone in both mothers' and children's models. BMI has been previously shown to correlate well with central adiposity in children of this age range (Cameron et al. 2009) and adults (Harris et al. 2000). In adults, BMI and WC have been shown to be broadly similar in the prediction of adult CVD risk (Huxley et al. 2010; Satoh et al. 2010). Most studies involving individuals of non-European descent have found that WC better predicts CVD risk than BMI, though the difference in magnitude is small enough that the clinical relevance is questionable (Huxley et al. 2010).

Centralisation of body fat primarily occurs during adolescence (Cameron et al. 1992). As such, it can be assumed that BMI and WC would be roughly equivalent in assessing abdominal obesity in children, but WC would be superior in adults. This study was not designed to assess such a question, but if that expectation was met, BMI would likely explain less variance in WC in mothers than the children. Since BMI explained about the same variance in WC, it does not appear that the relationship between BMI and WC changes during maturation in this sample of urban Mayans.

a. *Peripheral adiposity*

BMI was not a good predictor of peripheral adiposity. UFA, AFI and UMA had the lowest amount of variance explained by BMI. The explained variances of UMA and AFI were substantially lower than UFA. This indicates that BMI is comparatively a better estimate of total peripheral adiposity than lean mass or peripheral composition. This finding is not surprising as the torso contributes a higher proportion of weight for every centimetre increase in length than the limbs. Therefore BMI will be more closely related to central adiposity than peripheral. Peripheral adiposity does not contribute as much as central adiposity to CVD risk (Matsuzawa 2008) and is important mainly for determining nutritional status in those who are under-nourished (Frisancho 2008). For these reasons, the relationship between BMI and peripheral adiposity has not been well studied.

3. *Stunting impact overall*

Stunting was related to adiposity indicators in opposite directions in children and mothers, though stunting never reached statistical significance in multiple regression models. In the simple *t*-tests, stunted children had significantly lower adiposity indicators than non-stunted children while stunting had no effect on in the mother's bivariate analyses. Stunting was negatively associated with adiposity indicators in children and positively associated with them in mothers in the multiple regression models.

Stunting did not alter the statistical significance of BMI in its relationship to the other adiposity indicators. Also, stunting was rarely significant as a predictor and had a low beta value in every model. This result suggests that BMI can be used in stunted populations in the same way it is used in non-stunted populations. As long as BMI is used in conjunction with other measures of nutritional status, such as height and waist circumference, it appears to be a useful indicator of adiposity. Multiple measures of nutritional status offer a more complete picture of an individual's health to better assess disease risk.

It is possible that due to the limitations of this sample, stunting does influence the ability of BMI to predict adiposity outcomes but that this effect was not detected here. This sample had a short overall stature, clustering around stunting cut-offs and relatively low variation in stature and small sample size, all of which limit the power to detect differences. The short overall stature coupled with the low variation is

indicative of a group in which all individuals have experienced chronically poor conditions (Bogin 1999). This is the case regardless of whether they have grown adequately as to be considered clinically normal or not. Thus there is widespread limitation on growth. Even the taller individuals are still at high risk relative to a chronically well nourished population for negative health outcomes since the relationship between short stature and mortality is linear (Song and Sung 2008) and this sample is short overall. The apparent environmentally imposed limit on height may be over-riding any impact of stunting or body proportions that this study was attempting to investigate. Thus a population that has experienced more heterogeneous conditions would need to be studied to establish further evidence for whether stunting impacts BMI.

From a statistical point of view, the low inter-individual variation in height may have reduced the power of the analysis, which was powered to detect a medium to large effect size. An analysis on a sample with a higher amount of variance may find a statistically significant impact of stunting on the predictive power of BMI.

Alternatively, a larger sample might have the same effect.

Future studies examining Maya in Merida may wish to expand into more prosperous neighbourhoods in an attempt to increase the variation in stature. This may allow the comparison of stunted women (<150 cm) with taller women (>160 cm).

These results suggest that BMI can be used in stunted and non-stunted groups without correcting for an individual's stunting status in Maya groups similar to this sample with limited variation on height.

a. *Stunting impact on children's BMI*

Stunting had opposite impacts on children and mothers, though these never reached statistical significance in regression models. In the simple *t*-tests, stunted children had lower adiposity indicators than non-stunted children while stunting had no effect in the mother's bivariate analyses on adiposity indicators. Stunting was negatively associated with adiposity indicators in children and positively associated with them in mothers in the regression models. The interaction between stunting and BMI was significant for WC when stunting was defined as the 5th percentile (Table 6.5) but in no other model. Because only one model yielded a statistically significant finding for the interaction and multiple comparisons were made, the chances of a

type 1 error (false positive) occurring were high. The lack of strong evidence to suggest an influence of the interaction between stunting and BMI indicates that stunting does not impact the relationship between BMI and adiposity indicators.

The consistently negative relationship in children between adiposity indicators and stunting indicates that stunted children have consistently experienced poorer conditions than their peers. While we do not possess the data to determine the timing of these children's stunting, the international data are overwhelmingly consistent and robust as to the timing of stunting. The data shows that the majority of stunting occurs in children before 5 years of age (Behrman and Skoufias 2004; Stein et al. 2010). Therefore it can be relatively safely assumed that these children experienced growth limiting conditions during their first 5 years of life. After 5 years of age, if stunted children experience improved conditions, the increase in calories is mostly shunted toward adipose storage, not linear growth (Martins et al. 2004). Therefore, greater or equal adipose indicators would be expected to be found if the stunted children had experienced improved conditions since 5 years of age. Since they tend to have lower adipose stores, their environmental conditions can be assumed to have remained relatively poor.

It is a matter of debate as to whether these stunted children are *currently* under-nourished. These children, with their normal BMIs will not be targeted for any interventions for acute under-nutrition directed by the Mexican government. In the past few decades, the Mexican government has been focused on reducing malnutrition, primarily wasting and, more recently, obesity (Frenk et al. 2003). The BMI z-scores of both stunted and non-stunted children in this study are over the median of the references. Thus, from the gross index of weight-for-height (which is likely the main clinical diagnostic that will be used in these neighbourhoods) these children are not under-nourished. They have a history of under-nutrition, but there is very little, if anything, that can be done at this stage to eliminate the stunting. Attempts to reduce the stunting may even result in obesity due to the altered metabolism of stunted children (Hoffman et al. 2000a; Hoffman et al. 2000b; Martins et al. 2004).

Some of these children have been told to eat less by medical personnel because their BMIs are close to the 85th percentile (ethnographic data). However this may lead to health concerns as the diet of these children has little variety and is low in micro-nutrients (Chapter 4 section G). An added problem is that their digestive

abilities may be compromised. A link has been established between linear growth faltering and intestinal lining abnormalities (Campbell et al. 2003; Goto et al. 2009; Panter-Brick et al. 2009). If these chronically under-nourished children have compromised intestinal linings, this may impede the gut's ability to completely digest food, leading to micro-nutrient deficiencies. BMI and other anthropometric measures are not able to detect such deficiencies and therefore these cannot be assessed in this sample. These issues exemplify how BMI cannot be used in isolation and other factors must be considered when assessing these children's nutritional status and health.

b. *Stunting impact on mother's BMI*

There are many difficulties in using this sample of women, who show a very narrow range of heights, to assess whether BMI is related to adiposity indicators. Overall, maternal stunting did not impact the relationship between BMI and the adiposity indicators in the regression models. This finding mirrors results from the INCAP study in Guatemala, which found that stunting in childhood had no impact on adult women's BMI or percent body fat (Schroeder et al. 1999).

There is increasing evidence that stunted adults are at higher risk of obesity due to increased substrate utilisation, with a preferential storage of fat and burning of carbohydrates for fuel (Baur et al. 2006; Florencio et al. 2003; Frisancho 2003; Leonard et al. 2009; Martins et al. 2004; Sawaya et al. 2003). All of these women are of relatively low SES. It appears that once the threat of starvation or wasting is lowered or eliminated, individuals of low SES are at the highest risk of OW/OB (Monteiro et al. 2002; Ziol-Guest et al. 2009). Since no impact of stunting was found for these mothers, it is likely that the more behavioural and cultural aspects related to adiposity indicators are masking any biological effect of stunting. The negative consequences of adult stunting have been discussed in Chapter 2, section F.

4. *Influence of different cut-offs for stunting*

The different permutations of stature and stunting had little to no impact on the ability of BMI to predict adiposity indicators. This lack of difference between stunting cut-offs and stature entered as a continuous variable in analyses supports the lack of an impact of stature on the relationship between BMI and adiposity indicators in this sample of Maya mothers and children.

On the whole, this sample is very short, and clusters between stunting cut-offs. In this sample, 40% of children were classified as stunted based upon the 5th percentile of Frisancho's Comprehensive reference while only 17% were stunted when using the -2 z-score of the same reference. Since no clear biological cut-off exists between stunting and disease the choice of cut-off is at the discretion of the researcher. The 5th percentile of growth charts is a more inclusive cut-off while -2 z-scores is more conservative. Policy makers who want to push for more funding may be more likely to use the 5th percentile cut-off as more children will be classified as stunted than using -2 z-scores. Just like adult cut-offs, the population being studied and the context of the investigation will determine which cut-off will be chosen.

5. *Influence of body proportions on BMI*

The hypothesis that BMI should not be used for stunted populations because they will have disproportionally large torsos is not supported by this study. SHR does not significantly predict BMI in either mothers or children. SHR did not impact the power of BMI to predict any of the adiposity indicators in either mothers or children. This is a surprising finding as the theoretical basis of this assumption is quite strong since the torso is heavier for every cm increase in height than the legs. Also previous research has found a correlation between SHR and BMI (Bogin and Beydoun 2007; Norgan and Jones 1995).

In the simple regression of SHR to BMI, SHR did significantly predict BMI in this sample but explained less than 9% of the variance. This preliminary finding is what would be expected in a small sample based upon previous research. SHR has been found to be significantly correlated with BMI using large datasets of Western populations (Bogin and Beydoun 2007; Norgan and Jones 1995). Norgan and Jones (1995) examined a large British sample and found that adjustment for SHR changed the BMI of 39% of women and 11% of men by more than 1 unit. Bogin and Beydoun (2007) found that SHR had a significant relationship with BMI using the NHANES III database from the U.S. However the overall standardised beta was quite low (0.12-0.15 for women). SHR does appear to significantly impact BMI, and can do so quite substantially in certain groups however it may be a relatively small effect when considered alongside other factors.

For these urban Maya mothers, SHR did not impact the power of BMI to predict adiposity indicators. Thus, though SHR does impact BMI itself, it does not

appear to significantly influence BMI as an estimate of nutritional status in this sample. SHR has also been shown to be positively correlated with %BF in adults (Deurenberg et al. 1999; Frisancho 2007; Varela-Silva et al. 2007) and children (Frisancho 2007). SHR appears to influence adiposity in some populations however these findings are not supported by this research.

Previous research on body proportions and weight-for-height in Latin America has observed conflicting findings. Trowbridge and colleagues (1987) examined 139 Peruvian pre-school children who had a low mean height-for-age but a relatively high mean weight-for-height. SHR did not correlate with the weight-for-height in the Peruvian children. Using almost 200 children, Post and Victora (2001) used analysis of covariance to compare low and high socioeconomic status preschool Brazilian children (Post and Victora 2001). The low socioeconomic children had lower weight-for-height however these differences were attenuated after SHR was included in the model. Taking these studies together with this study, where children's SHR did not predict BMI in a simple regression model (Table 6.14) it does not appear as though SHR is a large contributor to BMI.

It must be noted that this study lacks an estimate of gluteo-femoral fat. Sitting height can be potentially increased through the non-compressible nature of adipose tissue (Bogin and Varela-Silva 2008). If this sample had significant amounts of buttock fat, their SHR would increase; therefore their legs would be estimated as shorter than they really are. Thus an influence of body proportions on BMI would be more likely to be detected, when none actually exists, a potential false positive. From visually assessing this population, the women measured were generally 'apple-shaped', with the majority of their fat stored in their torsos, not their hips and thighs. Therefore, it is possible that buttock fat does not provide as large of a bias as it would be in a mainly 'pear-shaped' (gluteo-femoral patterning) sample. However the lack of hip circumference measurements or other indicator of buttock fat prevents this potential bias from being properly and objectively estimated here.

These results suggest that the effect of SHR is small, and therefore there is no reason to cite it as a reason to not use BMI as an estimate of nutritional status. Until the evidence for the association between SHR and BMI becomes clearer, it would be sensible for researchers to consider SHR in short stature populations in analyses that model BMI. This recommendation is based on the strong theoretical

evidence for the association between SHR and BMI and the statistical evidence of a small- modest effect size for the association from previous studies.

E. *Conclusion*

This portion of the thesis has found that BMI is appropriate for use to estimate adiposity indicators in this population of 7-9 year old urban Maya children regardless of stunting status but not in this population of adult urban Maya women. It has also found that the ability of BMI to predict adiposity indicators is not impacted by stunting status or SHR. It is recommended that if BMI is used, it should be in conjunction with other adiposity indicators due to its many known limitations.

Chapter 7. *Mother's recalled early life and linear growth*

This chapter examines whether early life environment as recalled by an adult Maya woman is able to significantly predict her adult measures of linear growth.

A. *Introduction*

Childhood environment relates to growth and health, both in childhood (Cameron et al. 2005b; Case et al. 2005; Gunnell et al. 1998b) and in adulthood (Case and Paxson 2010; Cohen et al. 2010; Gunnell et al. 1998b; Pollitt et al. 2005). Stature is often used as an aggregate measure of chronic conditions during the growing years (Bogin and Keep 1999; Komlos 1999; Steckel 2009), circumventing the difficulties in assessing environmental exposures and socio-economic status (SES) (Chapter 2 section I.1).

Due to the cephal-caudal gradient in growth prior to puberty (Leitch 1951), relative leg length may be a stronger indicator of chronic environmental conditions than stature (Gunnell et al. 1998b; Wadsworth et al. 2002). Examining the body segments separately may enable the differentiation between ‘genetically short’ and ‘environmentally short’ individuals as these groups will have different body proportions. ‘Environmentally short’ individuals will have relatively shorter legs than ‘genetically short’ individuals, even at the same stature. Chapter 2, section I.1.a contains more information on leg length and chronic environment.

Few longitudinal studies have examined the relationship between leg length and childhood conditions, all of them in the U.K. (Gunnell et al. 1998b; Wadsworth et al. 2002). Population differences disallow applying findings from one group to another without validation. Unfortunately, longitudinal studies conducted in developing countries do not measure leg length or sitting height (from which leg length can be estimated). Therefore, there are no data on the relationship between childhood conditions and body proportions in developing countries. This study will use maternal recall of her childhood as a measure of childhood conditions.

Childhood environment relates to growth and health, both in childhood (Cameron et al. 2005b; Case et al. 2005; Gunnell et al. 1998b) and in adulthood (Case and Paxson 2010; Cohen et al. 2010; Gunnell et al. 1998b; Pollitt et al. 2005). This biosocial phenomenon is known as the Developmental Origins of Health and Disease (DOHaD) (Barker 1995a) (Chapter 2 section C.1). Since the body is growing and developing rapidly during the early years of life, it is more sensitive to influences of the environment than in the far more biologically static stage of adulthood (Cameron and Demerath 2002). SES can remain low throughout the lifetime and likely interacts with the lasting health effects from childhood (Pollitt et al. 2005). For

example, individuals of low SES have poorer health in childhood and in adulthood, which limits economic opportunities (Case and Paxson 2006), creating a cycle of poverty throughout generations. Individuals who experienced a poorer childhood SES are at a modestly increased risk for adult cardiovascular disease (Pollitt et al. 2005). This relationship strengthens when total years spent in the poor environment is included in the model (*ibid*). It appears as though SES throughout the life course impacts health in independent and interlinked ways.

The limitation of SES measurement must be considered at all stages of research to avoid incorrect conclusions. SES measurement relies heavily on self-reported data, which has the potential to be biased. Also, cultural differences in the interpretation of wealth and status must be considered when designing and interpreting tools to assess SES. Finally, the measurement must be sensitive enough to reflect enough variance in the sample in order for statistical tests to be performed.

All of these difficulties in measuring SES make easily collectible and objective measurements difficult to obtain, a problem magnified when attempting to assess past SES. One of the most widely used objective measurements of environment and health is growth, particularly stature to indicate chronic conditions (Bogin 1999; Tanner 1986). Poor growth is an indicator of poor health and environment in early childhood (Bogin 1999). Growth is one of the three main areas in which calories are expended in childhood (Kuzawa 2005). The other two areas are maintenance and activity, with any remaining calories stored as fat. In this way, health and growth are tightly linked in children. Improving the diet through supplementation has been shown to improve children's linear growth (Stein et al. 2010). Linear growth is very useful as a measure of aggregate chronic conditions during the growing years.

Overall, stature can and is used to assess the overall health of a population and determine the level of hardship that has been faced (Bogin 1999; Komlos 1998; Steckel 2009) (Chapter 2 section I.1). It can be a better indicator of population health than economic productivity, as when economic development is occurring the population may not reap the benefits immediately or equally (Komlos 1985; Komlos 1998; Komlos 2008; Komlos and Coclanis 1997; Komlos and Lauderdale 2007). Thus even in apparently prosperous economic conditions, the chronic environmental conditions experienced by the population can be stagnant or deteriorating. This is apparent in urban areas, which tend to display better measures of linear growth than rural areas, primarily due to an increase in access to health care, sanitation and

calories (Coly et al 2006, Mukuddem-Petersen and Kruger 2004, Oyhenart et al 2008). However urban areas in developing countries also exhibit large amounts of heterogeneity. In some countries, stunting rates are higher in urban areas than rural, due to the very poor conditions experienced by the urban poor (van der Poel et al 2007).

Although stature is often highly correlated with environmental conditions, it may not be the strongest growth measure to use to assess chronic environmental conditions. Stature is a composite measure of several body segments, all of which grow at different rates throughout the growing years (Bogin 1999; Cameron 2002). Legs are the body segment that grows most rapidly before adolescence and therefore will be more affected by chronic early life conditions than the torso, resulting in shorter legs relative to stature (Leitch 1951). Examining the body segments separately may enable the differentiation between 'genetically short' and 'environmentally short' individuals as these groups will have different body proportions. 'Environmentally short' individuals will have relatively shorter legs than 'genetically short' individuals, even at the same stature. Longitudinal studies in the U.K. have shown that leg length is more influenced by childhood diet than torso length (Gunnell et al. 1998b; Wadsworth et al. 2002). Also, measures of childhood SES have been shown to be more strongly associated with adult leg length than adult sitting height (Li et al. 2007). Leg length is also more strongly correlated to CVD mortality than torso length (Gunnell et al. 1998a).

That early life impacts growth and later health is uncontroversial. Less clear is the influence and relative importance of various particular factors. Various factors have been implicated in influencing growth. In developed nations, longitudinal studies have shown that family income is positively related to children's growth and that household factors, such as crowding and family size, are inversely related to growth (Li et al. 2007). Similar findings have been observed in developing countries, with children from higher SES families having improved growth compared to their lower SES peers (Bogin et al. 2002; Johnston et al. 1985; Reyes et al. 2010). Family size (Bogin and MacVean 1981) and higher birth order (Sparks 2011) have also been shown in developing countries to be positively related to growth. Sanitation (Jones et al 2008, Motarjemi et al 1993, Plate et al 2004), including access to clean drinking water and flush toilets, is also shown to have a substantial influence on the

health and growth of children, particularly in developing countries where sanitation is not universal.

B. *Aim*

The aim of this study is to determine whether the women's recalled early life environment is related to her adult linear size and also to establish which factors of recalled early life are the most important to adult size.

C. *Methods*

1. *Sample*

The study design was cross-sectional. This sample is composed of 58 urban Maya mothers living in the south of Merida, Yucatan, Mexico. For more information on this sample see Chapter 3, section B.

2. *Recruitment*

The Maya women were recruited through their children. Schools were approached that were located in *colonias* in the south of Merida with a high proportion of Maya students. Schools that agreed to participate provided the school lists and from these Maya children were identified by having two Maya surnames. The mothers were then invited to information sessions at their children's schools where the study was explained and information sheets were provided. For more information on the recruitment process, see Chapter 3, section B.3.

Written informed consent was obtained from the mothers and verbal assent from the children. Ethical clearance was obtained from the Loughborough University Ethics Committee in the U.K. and the Bioethical Committee of Cinvestav in Mexico. For more information on the ethics of this study see Chapter 3, section B.2.

3. *Measurements*

Mothers underwent anthropometric measurement using standard techniques (Lohman et al. 1988), which included stature and sitting height in centimetres. For more detail on anthropometric measurements and technique, see Chapter 3, section D.1. Sitting height ratio (SHR) and estimated leg length (ELL) were both calculated using stature and sitting height (Equations 5.1 and 5.2).

Equation 5.1: $\text{SHR} = (\text{sitting height} / \text{stature}) * 100$

Equation 5.2: $\text{ELL} = \text{stature} - \text{sitting height}$

*All measures for both calculations are in centimetres.

SHR was not normally distributed, due to kurtosis, and therefore was transformed by taking the square root. The square root of SHR was normally distributed and was used in conjunction with the untransformed SHR in bivariate analyses. Both the transformed and untransformed SHR were used in bivariate analyses because the residuals for SHR in linear regression models were normally distributed, allowing the final multiple regression models to be performed on the untransformed SHR.

4. Interview

The women were interviewed in their homes, primarily, or in the school by a local female fieldworker. The interview included questions about the women's childhood SES and environmental conditions. The interview is described in detail in Chapter 3 section C. An English version of the interview can be found in Appendix B.1.

The questions were focused on six categories: Consumer durable ownership, Economic stability, Family stability, Housing/ sanitation, Modernisation, and Demography. Each variable included in the categories is shown in Table 7.1.

Questions regarding ownership of consumer durables (e.g. radio, television) were based on the Demographic and Health Survey (DHS) questionnaire (MeasureDHS 2011). An additional ownership question related to ownership of animals for consumption or transport as included, as ownership of these would be considered material wealth in the more traditional Maya families.

Economic stability questions were designed in order to assess whether the woman was able to perceive financial instability during her childhood (e.g. job loss of caretaker). It was assumed that the women who did perceive economic instability were more likely to be from families where the children were less buffered from financial strain or from families who had more financial strain.

Family stability questions were designed to capture the social aspect of SES and focused upon common problems experienced by the Maya (e.g. problematic migration of a family member) and in all societies (e.g. family death).

Housing/ sanitation questions were based upon DHS questions relating to the family's living conditions (e.g. electric lighting in the house) and hygiene (e.g. flush toilet). The housing materials were used to compile an aggregate score based upon the permanency, traditionalism and health effect of the building materials (see Chapter 3, section E.4).

Modernisation aimed at assessing the woman's exposure to non-traditional culture by assessing place of birth (urban versus rural) and purchased food consumption (e.g. sugar sweetened beverage).

Demographic questions were also based upon the DHS, focusing on family size and sibling mortality. These questions included birth year and the total number of siblings, older siblings and siblings who died before they reached 5 years of age.

No age cut-offs were given to define childhood when the mothers were interviewed. Questions were asked about the age at acquisition of consumer durable goods, and from these, we can estimate the age range these women perceived as their childhood. Most of these answers clustered between the ages of 5 and 14 years (range 2 to 17). Previous research done in this community in 2006 did specifically ask the Maya women to define childhood (Azcorra 2007). The majority responded that it encompassed the ages of primary school (6 to 12 years old). Therefore, in this analysis the women's recalled early life refers to the women's childhood and adolescence. It is not targeted on an exact period of early life.

Table 7.1 Maya women's recalled early life variables by category

| Category | Variables | | |
|----------------------------|---------------------------------|--------------------------------------|--------------------------------------|
| Ownership | Radio | Telephone | Refrigerator |
| | Car | Television | Animals for consumption or transport |
| Economic stability | Job loss of caretaker | Pawning of valuable items | Sale of valuable items |
| Family stability | Serious illness of in family | Death in the family | Divorce of caretakers |
| | Migration in family | Other problems | |
| Housing/ Sanitation | House materials Flush toilet | Electric lights | Clean water |
| Modernisation | Urban born | Sugar sweetened beverage consumption | Packaged food consumption |
| Demography | Birth year | Sibling number | Sibling death before 5 years of age |
| | Birth order | | |

¹A similar version of this table appears in Chapter 3, section E.4

5. SES index

The construction of the SES indices is discussed in detail in Chapter 3, section E.4. Briefly, the variables of the women's recalled early life were grouped into theoretically based indices (see Table 7.1). Values for each index were calculated. Responses were assigned a 0 for the least beneficial category and a positive integer for the beneficial categories. Bivariate variables were coded as 0 and 1. For example, having clean water was assigned a 1 while not having clean drinking water was assigned a 0. Also not having a serious illness in the family was assigned a 1 and having a serious family illness was assigned a 0. Variables with three categories were coded as 0, 1 and 2. For example, consumption of sugar sweetened beverages (as an indicator of modernisation) was coded as 0 for never, 1 for rarely and 2 for frequently. Then each variable included in the index was summed to obtain a final index score.

For the Modernisation index clarification of the coding is needed since modernisation is not intrinsically linked with improved health. The response most indicative of modernisation (which is often linked with improved growth status) was coded as the highest number (e.g. 1 for urban born and 2 for frequent sugar sweetened beverage consumption and 2 for frequent packaged food consumption). Also, due to the small number of variables in the Modernisation index, the PCA index was not used due to the limitations of PCA using small numbers of variables (Field 2005). The range of the Modernisation PCA index had a mean of 0 with a range of -

1.9 to 1.4. The mean additive index score was 1.3 with a range of 0 to 2.33. Neither Modernisation index was normally distributed.

6. **Statistical analysis**

Bivariate analysis to assess the impact of each socio-economic variable on linear growth outcomes was performed to determine the SES variables to be included in the final analysis. Stature and ELL were normally distributed and therefore parametric analyses were performed upon them. SHR was square root transformed; the square root of SHR was normally distributed. SES indices and individual SES variables were compared to stature, ELL, SHR and square root of SHR using Spearman's correlations, independent *t*-tests, Mann Whitney U tests, ANOVA or Kruskal Wallace tests. Those with a *p*-value <0.10 were entered into the final regression models. The test used and *p* value for each variable/ index are shown in Appendix D. Due to the large number of bivariate comparisons made, only the significant variables are included in this chapter. Appendix D also contains the results of all individual SES variable bivariate comparisons with linear growth measures.

a. **Model building**

The main analyses were conducted using forced entry multiple linear regressions with height, SHR and ELL as the dependent variables. The predictor variables were SES variables and indices which were tending toward significance ($p < 0.10$) in bivariate analyses. Indices were preferred over individual SES variables as they provided a more complete reflection of SES than a single measure and to maximise the model's power by keeping the number of parameters low. Individual SES variables were used only when the originally tested index did not include variables that influenced linear growth measures in the direction. Since there is no way to predict *a priori* which factors have the most effect or occurred first in the women's life, all combinations of these significant variables were tested within the regression models. Table 7.2 shows this model building. Entering variables into the model in this shows how the inclusion of variables influences the other variables in the model. This allows for a more complete understanding of the relationship between variables in the model compared to entering all variables in one step.

This analysis should be considered an exploratory analysis to determine which variables are related to linear growth in Maya women. This is because the sample size means that the analysis only has the power to detect large effect sizes with the sample size available (Cohen 1992).

All analyses were performed using SPSS v. 17.0. Significance was set *a priori* at $p < 0.05$ for multiple linear regressions. Due to multiple comparisons within the multiple linear regressions, Bonferroni adjustments were made. Significance was divided by the number of steps within the model ($0.05/4$). The final significance was set at $p < 0.025$.

Table 7.2 Multiple regression model building for Maya women's SES versus her recalled early life

| | | Model | | | |
|-------------------------|---|-------|---|---|---|
| | | 1 | 2 | 3 | 4 |
| Predictors ¹ | A | X | X | | X |
| | B | X | | X | X |
| | C | | X | X | X |

¹Predictors determined using bivariate analyses

D. Results

The anthropometric descriptive results are displayed in Table 7.3. Overall the women were very short and overweight with relatively short legs.

Table 7.3 Anthropometric variables for adult Maya women

| | Mean (SD) |
|--------------------------------------|---------------|
| Age (yrs) | 34.44 (6.3) |
| Stature (cm) | 146.83 (4.54) |
| Weight (kg) | 64.10 (10.46) |
| Body mass index (kg/m ²) | 29.69 (4.40) |
| Sitting height (cm) | 78.90 (2.67) |
| Sitting height ratio | 53.73 (1.19) |
| Estimated leg length (cm) | 67.97 (3.02) |

The variables used to construct each index are found in Table 7.4. A radio was the most commonly owned item, with a car being the least commonly owned item. The majority of families had a death in the family and other family problems,

with the most commonly reported other problem was related to alcoholism of a male family member (see Chapter 4 section D.4). Half of the women reported job loss of caregivers as well as pawning of valuable property. Flush toilets were very uncommon, although 75% had clean drinking water. Slightly more women were born in rural areas than urban. Over one third of women had a sibling die before they reached five years and over 10% were the first born child (Table 7.5). The mean sibling number was over six and on average these women were the fourth born. The highest number of children in one family who died before they reached five years was five.

Variables that had a $p < 0.10$ in bivariate comparisons are shown in Table 7.7. The predictive variables in the models for ELL and SHR were similar.

The identified predictor variables for stature were the consumer durable ownership PCA index, birth decade and sibling death. For ELL they were the sibling number (tertiles) and birth order (first born vs. later). For SHR they were the sibling number (tertiles) and birth order (continuous).

Table 7.4 Maya women's recall of ownership and family and economic stability in childhood. %(n)

| Category | Variable | Yes | No |
|----------------------------|--|-----------|-----------|
| Consumer durable ownership | Radio | 75.9 (44) | 24.1 (14) |
| | Telephone | 3.4 (2) | 96.6 (56) |
| | Refrigerator | 12.1 (7) | 87.9 (51) |
| | Car | 5.2 (3) | 94.8 (55) |
| | Television | 55.2 (32) | 44.8 (26) |
| | Tricycle/ bicycle | 48.3 (28) | 51.7 (30) |
| | Animals for consumption or transport | 82.8 (48) | 17.2 (10) |
| Family stability | Serious illness in family | 46.6 (27) | 53.4 (31) |
| | Death in family | 63.8 (37) | 36.2 (21) |
| | Divorce of caregivers | 31.0 (18) | 67.2 (39) |
| | Migration that caused problems in family | 24.1 (14) | 75.9 (44) |
| | Other family problems | 46.6 (27) | 53.4 (31) |
| Economic stability | Job loss of caregivers | 50.0 (29) | 50.0 (29) |
| | Sale of valuable property | 36.2 (21) | 63.8 (37) |
| | Pawning of valuable property | 53.4 (31) | 46.6 (27) |
| Housing/ sanitation | Flush toilet | 5.2 (3) | 94.8 (55) |
| | Clean drinking water | 74.1 (43) | 25.9 (15) |
| | Electric light source | 67.2 (39) | 32.8 (19) |
| Modernisation | City born | 48.3 (28) | 51.7 (30) |
| Demography | First born | 13.8 (8) | 86.2 (50) |
| | Sibling death | 34.5 (20) | 65.5 (38) |

Table 7.5 Maya women's reported sibling and family size

| Variable | Mean (SD) | Range |
|----------------------------------|-------------|-------|
| Total sibling number | 6.66 (3.11) | 0-14 |
| Older sibling number | 3.03 (2.34) | 0-10 |
| Dead sibling number ¹ | 0.53 (0.93) | 0-5 |

¹Number of siblings who died before the age of 5 years old.

Table 7.6 Maya women's recall of purchased food consumption in childhood, %(n)

| Variable | Never | Rarely | Frequently |
|--------------------------|-----------|-----------|------------|
| Sugar sweetened beverage | 13.8 (8) | 44.8 (26) | 14.4 (24) |
| Packaged foods | 25.9 (15) | 39.7 (23) | 34.5 (20) |

Table 7.7 Outcomes with a $p < 0.10$ of the bivariate analyses of women's recalled early life SES and measures of linear growth and body proportions

| Variable | | Test | p-value | Included in final analysis |
|-----------------------------|--------------------------------------|----------|---------|----------------------------|
| Height | Consumer durable ownership PCA index | Spearman | 0.031 | Y |
| | Birth decade*: | | | |
| | 1960-1970 vs. 1971-1980 | t-test | 0.11 | Y |
| | 1981-1987 vs. 1971-1980 | | 0.012 | Y |
| | Sibling died before 5 yrs old | t-test | 0.087 | Y |
| SHR square root | Sibling number | ANOVA | 0.068 | |
| SHR | Sibling number | Spearman | 0.086 | Y |
| | Birth order | Spearman | 0.035 | Y |
| Estimated Leg Length | Consumer durable ownership PCA index | Spearman | 0.019 | |
| | Economic wealth PCA index | Spearman | 0.064 | Y |
| | Birth decade | ANOVA | 0.05 | Y |
| | Number of siblings | ANOVA | 0.055 | Y |
| | First born | t-test | 0.048 | Y |

Though SHR was not normally distributed, the residuals for each regression model were normally distributed, thus all of the required assumptions for linear regression were met (Field 2005). Therefore, the linear regression models for the square root of SHR are not discussed further, see Appendix D for the outcomes of the regression. Further, the bivariate analyses of SHR and SHR square root found the same variables to be significant. Sibling number was significant in two forms: the

tertiles were found to tend toward significance for SHR square root ($p=0.068$) while the continuous data tended toward significance for SHR ($p=0.086$). As sibling number did not have a linear relationship with SHR, the tertiles were chosen for inclusion in the multiple linear regression model.

1. Main analysis

Birth decade was the only significant predictor of stature (Table 7.8) in the multi-variate regression models and predicted the majority of the variance. Specifically, women born between 1981 and 1987 were significantly shorter [$\beta = -3.57$ (SE=1.35)] than those born in earlier decades. Consumer durable ownership and sibling death were insignificant in every model and explained less than 5% of the variance.

Table 7.8 Stature of mothers predicted by recalled early life factors in multiple linear regression models

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|----------------------------------|------------------------------|----------|--------------------|----------|--------------------|----------|--------------------|----------|
| | B (SE) | <i>p</i> | B (SE) | <i>p</i> | B (SE) | <i>p</i> | B (SE) | <i>p</i> |
| Constant | 148.262 (0.798) | <0.001 | 147.471 (0.722) | <0.001 | 148.992 (0.880) | <0.001 | 148.888 (0.876) | <0.001 |
| Consumer durable ownership index | 0.955 (0.569) | 0.099 | 0.816 (0.595) | 0.176 | | | 0.798 (0.569) | 0.167 |
| Birth decade* | 1960-70 -2.346 (1.366) | 0.099 | | | -1.802 (1.426) | 0.212 | -1.831 (1.414) | 0.201 |
| | 1981-87 -3.344 (1.399) | 0.018 | | | -3.597 (1.365) | 0.011 | -3.566 (1.353) | 0.011 |
| Sibling death** | | | -1.856 (1.242) | 0.141 | -2.306 (1.231) | 0.066 | -2.010 (1.238) | 0.110 |
| R² adjusted | 0.105 | | 0.049 | | 0.116 | | 0.132 | |

Stature regression with birth decade as a binary variable

*1971-1980 set as reference.

**No set as reference

For SHR (Table 7.9), neither variable was significant at the Bonferroni adjusted p value (0.025) nor consistently significant at the $p<0.05$ level. Birth order had a negative relationship with SHR indicating a higher birth order was related to shorter legs and had the larger absolute B.

Table 7.9 Sitting height ratio of mother predicted by recalled early life factors in multiple linear regression models

| | Model 1 | |
|-------------------------------|-------------------|----------|
| | B (SE) | <i>p</i> |
| Constant | 53.365 (0.393) | <0.001 |
| Sibling number ¹ | 0.065 (0.051) | 0.209 |
| Birth order ¹ | -0.576 (0.487) | 0.242 |
| R² adjusted | 0.035 | |

¹Entered as a continuous variable

No variable consistently predicted ELL at the Bonferroni adjusted level ($p < 0.025$), though having at least 8 siblings was significant at this rigorous level in model 1 (Table 7.10). Having at least 8 siblings also had the most consistently low p value, predicting that women with large numbers of siblings tended to have shorter legs. Economic wealth was never significant but contributed the majority of the variance explained. Being the first born child predicted longer ELL however the affect was attenuated with the inclusion of sibling number. As the variables 'sibling number' and '2nd born or later' are both measures of family size and sibling number, an independent t -test was performed to determine whether the two were statistically related. Being first born was tended toward significance with sibling number ($t(56) = 1.936$, $p = 0.058$).

Table 7.10 Estimated leg length of mother predicted by recalled early life factors in multiple linear regression models

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---------------------------------|-------------------|----------|-------------------|----------|-------------------|----------|-------------------|----------|
| | B (SE) | <i>p</i> | B (SE) | <i>p</i> | B (SE) | <i>p</i> | B (SE) | <i>p</i> |
| Constant | 69.108 (0.676) | <0.001 | 67.692 (0.407) | <0.001 | 68.772 (0.686) | <0.001 | 68.744 (0.722) | <0.001 |
| Economic wealth index | 0.643 (0.389) | 0.104 | 0.697 (0.382) | 0.074 | | | 0.634 (0.386) | 0.107 |
| Sibling number * 0-4 | -1.081 (1.007) | 0.288 | | | -0.881 (1.010) | 0.387 | -1.043 (0.999) | 0.302 |
| ≥8 | -2.035 (0.900) | 0.028 | | | -1.843 (0.941) | 0.056 | -1.673 (0.932) | 0.079 |
| 2 nd or later born** | | | 2.235 (1.164) | 0.060 | 1.677 (1.230) | 0.179 | 1.643 (1.211) | 0.181 |
| R² adjusted | 0.097 | | 0.091 | | 0.056 | | 0.111 | |

*5-7 siblings set as reference

**First born set as reference

a. *Birth decade and linear growth*

A significant effect of birth decade on stature was not expected in a sample this small. Birth decade can be used as a proxy for the more distal SES aspects that influence an individual's health (Komlos 1998; Steckel 2009). Therefore, the absolute measures of linear growth were compared to the women's birth decade and whether they were born in a rural or urban area using multiple linear regressions.

The women born in the 1970s are the tallest in the sample, with the 1960s and 1980s born women 2.5 and 3.5 cm shorter, respectively (Table 7.11). The women born in rural areas follow this same trend, although the urban born women are equally tall whether born in the 1960s or the 1970s. Both urban and rural dwellers experience a decrease in stature in the 1980s compared to the 1970s. The same inverted U-shape trend was found for the ELL of the total sample and the rural dwellers (Table 7.12). However the urban dwellers experienced a negative secular trend in ELL throughout the decades.

Table 7.11 Maternal stature by birth decade and urban versus rural environment in childhood. Mean (SD)

| | Urban | | Rural | | Total | |
|-------------------|---------------|----|---------------|----|---------------|----|
| | Mean (SD) | N | Mean (SD) | N | Mean (SD) | N |
| 1960-1970 | 149.58 (5.08) | 5 | 143.81 (4.60) | 9 | 145.87 (5.40) | 14 |
| 1971-1980 | 147.69 (2.76) | 16 | 149.00 (5.27) | 13 | 148.28 (4.06) | 29 |
| 1981-1987* | 144.91 (3.44) | 7 | 144.95 (4.44) | 8 | 144.93 (3.86) | 15 |
| Total | 147.33 (3.63) | 28 | 146.36 (5.27) | 30 | 146.83 (4.54) | 58 |

*The range of birth years for this sample of women is 1960 to 1987.

Using a multiple linear regression with city versus decade as an interaction, no significant interaction was found. $p=0.101$.

Table 7.12 Estimated leg length (cm) of Maya women by birth decade and urban versus rural environment in childhood. Mean (SD)

| | Urban | | Rural | | Total | |
|-------------------|--------------|----|--------------|----|--------------|----|
| | Mean (SD) | n | Mean (SD) | N | Mean (SD) | N |
| 1960-1970 | 70.20 (1.88) | 5 | 66.14 (3.03) | 9 | 67.59 (3.29) | 14 |
| 1971-1980 | 68.43 (1.93) | 16 | 69.02 (3.78) | 13 | 68.71 (2.90) | 29 |
| 1981-1987* | 67.37 (1.96) | 7 | 66.57 (3.47) | 8 | 66.95 (2.80) | 15 |
| Total | 68.49 (2.08) | 28 | 67.51 (3.63) | 30 | 67.97 (3.02) | 58 |

*The range of birth years for this sample of women is 1960 to 1987.

Using a multiple linear regression with city versus decade as an interaction, no significant interaction was found.

A linear regression was performed to assess whether stature or ELL were predicted by birth decade and urban versus rural dwelling (Table 7.13). It was found

that birth decade explained a small but significant percentage of the variance in both stature and ELL. The secular trend occurred between the 1970s and 1980s births in stature and in ELL, tending toward significance ($p < 0.10$).

Table 7.13 Multiple linear regression of stature and estimated leg length (ELL) prediction by birth decade and rural versus urban living

| | Constant | | Birth decade ¹ | | | | Rural dwelt ² | | Model | |
|----------------|--------------------|----------|---------------------------|----------|-------------------|----------|--------------------------|----------|-----------------------|----------|
| | B (SE) | <i>p</i> | 1960-1970 | | 1981-1987 | | B (SE) | <i>p</i> | R ² adj | <i>p</i> |
| | | | B (SE) | <i>p</i> | B (SE) | <i>p</i> | | | | |
| Stature | 148.276 (0.811) | <0.001 | -2.404 (1.421) | 0.096 | -3.343 (1.389) | 0.019 | | | 0.076 | 0.043 |
| | 147.287 (1.990) | <0.001 | -2.280 (1.488) | 0.121 | -3.288 (1.401) | 0.023 | -0.637 (1.170) | 0.588 | 0.064 | 0.088 |
| ELL | 68.707 (0.561) | <0.001 | -1.114 (0.972) | 0.257 | -1.760 (0.951) | 0.070 | | | 0.030 | 0.165 |
| | 67.393 (1.344) | <0.001 | -0.962 (0.681) | 0.332 | -1.701 (0.951) | 0.079 | -0.856 (0.795) | 0.287 | 0.033 | 0.193 |

¹1971-1980 set as reference because it was the decade with the tallest statures.

²Urban set as the reference because it had the tallest statures.

E. Discussion

Variables of recalled early life significantly predicted SHR and ELL, but not stature, in this sample of urban Mexican Mayan women. The complete multi-variate regression model explained up to 5%, 9% and 11% of the variance in stature, SHR and ELL, respectively. SHR and ELL were both significantly predicted by the same variables: the Modernisation index and birth order/ first born. For ELL, first born tended toward significance ($p < 0.10$). Ownership index, birth decade, sibling death and sibling number were the variables which were significant in bivariate models, but not the multi-variate models.

Firstly it must be stated that there is the very real possibility that a statistical error occurred. Both a type I (false negative) and a type II error (false positive) are possible. The type I error is likely as many of the social factors have been shown to play a role in childhood growth in previous studies (Chapter 2, section F.2). Also a type II error is possible due to the potential for memory distortion in recalling the early life SES measures.

A low proportion of significant findings using this method was also found in a sample of elderly Chinese adults who were children during the People's Revolution

and under Mao's rule (Schooling et al. 2008a). When men and women were grouped together ($n=9,998$), the only significant predictor of ELL or stature was parental literacy (Schooling et al. 2008b). When only women were examined ($n=7,273$), the only predictor of her stature or ELL was her age at menarche and her education (Schooling et al. 2008b). The People's Revolution and Mao's regime are both associated with far more socio-political flux than the current epidemiological transition in Mexico. Therefore, it is possible that the conditions experienced by the Chinese adults in childhood were not stable and long lasting enough to impact linear growth. However the evidence from Chinese adults, taken with the well documented memory distortions that occur over time in humans (Schmolck et al. 2000a), suggest that it is very likely that using recall of childhood conditions is not an appropriate measure of actual conditions in early life.

Therefore, the factors discussed in this chapter will be used as a platform from which to discuss early life influences on linear growth, placed in a Maya context. This discussion will focus on the early life variables which were included in the regression models.

1. *Consumer durable ownership*

Consumer durable ownership is a very common method of assessing SES in developing countries (Bollen et al. 2001) and has been shown to relate to other measures of SES (Filmer and Pritchett 2001). The relationship between consumer durable ownership and stature has been observed previously (Bogin and MacVean 1983). The relationship has been most frequently measured in young children (Bomela 2009, Fotso and Kuate-Defo 2005, Mohsena et al 2010, Sichieri et al 2003), as this is the time that stunting is most likely to occur (Stein et al 2010).

The relationship between recalled consumer durable ownership in childhood and adult linear growth measures has also been observed previously. Adult recall of childhood consumer durable ownership has been positively and significantly related to adult stature in Polish and Russian adults (Webb et al 2007). A previous study in China on recalled consumer durable ownership in childhood found ownership to be significantly related to stature and leg length (Schooling et al 2008). The Chinese study was much larger ($n=9,998$) than this study on the Maya ($n=58$) and therefore had more power to detect small differences within the data. It is possible that a similar relationship between early life consumer durable ownership exists in these

Maya women but this study was under-powered to detect it. As the relationship between consumer durable ownership and linear growth is well established, the remainder of this chapter will focus on the other aspects of SES.

2. Modernisation

In this sample of urban Maya women, birth decade tended to be related to stature and leg length. This will be used as the basis for discussing the relationship between modernisation and growth. Just like most of the developing world, the Maya (Leatherman et al. 2010) and the Mexican population (Rivera et al. 2002) in general are undergoing rapid nutrition transition and modernisation. In this study, we focused upon the impact on linear growth of two transition factors: urbanisation and diet. The population of Merida increased by 26% during the 1990s (Azcorra 2007). During this period, the diet has also changed in the Yucatan, toward more purchased and processed foods (Leatherman and Goodman 2005).

The majority of the world's population currently lives in urban environments (UNPF 2007). Urban environments and lifestyles mark a substantial deviation from the traditional, rural lifestyle of the Maya. Individuals living in urban areas in developing countries tend to have better growth (Barquera et al. 2007; Crooks 1994; Oyhenart et al. 2008) and health than those living in rural areas, mostly due to increases in SES (Fotso 2007; Smith et al. 2005; Van de Poel et al. 2007). In this study of Maya women, those born in a city were taller, but not significantly so, than the women born in rural areas. The lack of a significant difference was also found by Azcorra and colleagues (2009), who compared young urban migrant children (aged 4-6 years old) and urban born children in Merida for growth status and found no difference (Azcorra et al. 2009). Higher rates of stunting in the urban poor than rural areas have been found in several Latin American countries (van der Poel et al 2007).

Lifestyles are different in rural and urban areas (Popkin 1999), particularly when making a comparison between very traditional rural cultures, such as the Maya, with an urban lifestyle. Farming is central to the culture and the economics of the Maya, with maize (corn) at its heart. Also before the 1970s, the Yucatan had a monopoly on the *sisal* industry, which was one of the main sources of rope for worldwide industry. Many of the rural born women, particularly the older ones, were likely born into families who were heavily dependent upon the *sisal* industry for income. However few appeared to work directly for the industry as only 2 women

lived on a *hacienda*, where the *sisal* was grown. The urban women were from families primarily supported by the construction industry and who followed a less traditional lifestyle. Chapter 4, section E.2 discusses these women's parents' occupations.

Modern and urban diets have been shown to include more saturated fat and less fibre than traditional, rural diets (Walker et al. 2001). Such foods have been repeatedly linked to obesity (Kelles and Adair 2009). The reduced emphasis on farming is coupled with an increased reliance on purchased foods. For example, in this sample of Maya women *milpas* (very large vegetable gardens, with many different types of plants) are less common in women who were born in a city, with only 2 of 28 urban born women having a *milpa* versus 22 of 30 rural born women ($X^2(1) = 26.158$, $p < 0.001$). Shifting from farming to purchasing foods allows for a relatively stable food supply, and allows money to be made in other occupations, outside of the home. While food that is purchased is not necessarily of low quality, many of the purchased foods are processed and packaged. Highly processed foods are often calorie-dense and nutrient poor (Sodjinou et al. 2009), not optimal for growth. See Chapter 2, section F.3 for a discussion on diet and linear growth.

a. *Sugar sweetened beverage and sugar sweetened beverages*

A dietary change that is very prominent in Mexico is a dramatic increase in sugar sweetened beverage consumption. Mexico has shown an increase of sugar sweetened beverage purchases over the past several decades with a concurrent decrease in milk purchases, while both have decreased in cost (Barquera et al. 2008). Daily caloric intake from sugar sweetened beverage consumption has increased while calories from non-sugar sweetened beverage beverages have remained stable (*ibid*). Mexico is the second largest consumer of Coca Cola in the world, after the United States. In what has been dubbed 'The Cola Wars', Pepsi Cola and Coca Cola compete aggressively for the dollars of Mexicans (Bell 2006). Coca Cola have an incredibly efficient distribution system that includes real time data, which is immediately fed back into the distribution system, they employ local entrepreneurs and aggressively market to local people (Gates 2010).

Sugar sweetened beverage has become a very important part of Mexican culture, even being fed to bottle fed infants (field notes). In the south of Merida, a bottle of sugar sweetened beverage costs more than a bottle of purified water from

the small, local *tiendas* (*ibid*). Increased consumption of sugar sweetened beverage in rural Maya communities in the Yucatan has been linked with tourism (Leatherman and Goodman 2005).

Sugar sweetened beverage is a drink that is calorie dense and nutrient poor and is associated with obesity (DiBello et al. 2009; Rivera et al. 2002). However it is also a source of clean water and calories, which in undernourished, marginalised populations are often considered very important for health (UNICEF and WHO 2009). In part this may be why modernisation is associated with longer legs in this sample of Maya women. Supplementing the traditional diet with calorie-dense sugar sweetened beverage may have added calories to the diet of a group that was calorie deficient, improving growth. This is not the first study to observe that modernisation, in the form of tourism, is associated with an increase in stature in other Maya groups in the Yucatan (Leatherman et al. 2010). This increase in stature is unfortunately also accompanied by an increase in obesity (*ibid*). Therefore the modernisation of the diet of the Maya appears to be a double edged sword. This double edged sword is found in the modernisation of many diets, particularly those that were traditionally quite limited.

3. *Macroeconomics and birth decade*

Though a significant secular trend in stature was found by birth decade, the small sample along with unequal group numbers does not allow for firm conclusions to be made based upon this finding. Therefore, this discussion is intended to be a case study to highlight the influences of regional and national factors, specifically macroeconomic trends, on the well being of low income populations.

The economic trends of Mexico follows the same basic pattern as was experienced by many countries in the world during this period. Overall, countries experienced steady growth during the post World War II period followed by an extended economic downturn in the 1980s (Santaella 1998). The Post War period was a time of relative prosperity and growth for many nations. Mexico and Brazil were growing at over 3% of their GDP (Gross domestic product) annually during this period. The growth of the Mexican economy was three times larger than their increase in population, more than enough to offset the population growth. Thus the overall, average standard of living and per capita GDP were improving in time with the growth in the national GDP (*ibid*). The Yucatan state maintained a stable

percentage of the national GDP, so the Yucatan economy has risen and fallen with the national trends. The economic sectors which grew the most were tourism and construction, both showing annual growth of over 7%. Agriculture showed more modest growth of over 3%. This growth in agriculture was outstripped by the service industries and the contribution of agriculture to the GDP shrunk by over 60% during this time frame. This shift in the country's economy is mirrored in the growth of Merida. In 1950, Merida was home to 140,000 people, which grew to 400,000 by 1980 (Fuentes 2005). This growth was accelerated in the 1970s with the collapse of the sisal industry, driving many to the city in search of employment.

In 1981, an extended economic downturn occurred, negatively impacting the economies of almost all countries outside of East Asia (Santaella 1998). Latin America was hit particularly hard by the downturn. Mexico's annual growth in GDP slowed to 2%. The per capita GDP change in Mexico decreased more than the national GDP, resulting in stagnating or a deterioration of living conditions for much of the population. The industries most affected by the economic downturn in Mexico were the construction and tourism industries. Both were reduced to approximately 1.5% annual growth from 1981 to 1995, a dramatic reduction from the booming 7.5% annual growth seen in the previous decades (*ibid*). Even though the construction industry experienced an extended downturn during the 1980s, the population growth of Merida continued unabated, increasing by another 24% over the decade. In 1990 the population of Merida was 520,000 (Fuentes 2005).

The data from these Maya women follow the same overall trends as the macro-economic pattern would indicate. The average stature increased by 2.5 cm for those women born between 1960 and 1980, in time with the economic growth. These data suggest that all women experienced a substantial improvement in living conditions between 1960 and 1979. This is consistent with the increase per capita GDP and agriculture growth seen during the same period. The mean average stature decreased by more than 3 cm in stature during the economic downturn of the 1980s. From the apparent negative secular trend, it appears as though the boom in standard of living over the previous decades was not enough to buffer these families from the poor economy. A relationship between macro-economic trends and stature seems to be detected in a small sample that was not designed to examine the relationship. This exemplifies how substantial the impact between macro-economic trends and children's health is. However this does need to be interpreted within the

context of the fact that even in the best of economies, the women were stunted as the mean stature for women born in the 1970s was 148.28 cm.

4. Family structure

Multiple factors associated with family demographics are related to these Maya women's linear growth, including sibling death, sibling number and birth order.

Early sibling death was negatively related to Maya women's stature. It is likely that a family which had a child who died before his 5th birthday, on average, had poorer caring practices and/ or reduced access to health care and/ or poorer living conditions. This is primarily true of marginalized groups who do not have easy access to health care, particularly in developing countries where health care is limited to all but the wealthiest groups. Contaminated drinking water and poor sanitation are implicated in high rates of stunting and infectious diseases (UNICEF and WHO 2009). Thus if a family is consuming contaminated water, there is an increased risk of infant death and poor growth outcomes for the surviving children.

Sibling number was complexly related to ELL and SHR. The relationship between SHR and sibling number was linear and positive. However ELL had an inverted U shape relationship to sibling number. The group with the longest legs was those women with 5-7 siblings, while both small sibling number (0-4) and large sibling number (≥ 8) had shorter ELL. Family size in childhood has been shown to be negatively related to adult absolute leg length in Britain (Li et al. 2007). Earlier birth order was related to longer legs (positively to ELL and negatively to SHR) in this sample of Maya women.

This relationship between linear growth and family size and birth order can be explained by resource allocation. Lower sibling number is related to increased access to and reduced competition for resources. Therefore, first born children will benefit from this lack of competition during infancy, in the postnatal period of most rapid growth, even if their sibling number as they grow older is very high. Thus lower sibling number is associated with improved growth (Lawson and Mace 2008; Li et al. 2007). The same relationship has been found in the statures of children (Bogin and MacVean 1981; Li et al. 2007; Sparks 2011). Once a very high level of population-wide wealth and access to health care is reached, this affect seems to be marginal or disappear. Using data from the Avon Longitudinal Study of Parents and Children (ALSPAC) children born in Britain in 1990, a difference of 0.15cm was found

between the stature of children from large families and only children (Lawson and Mace 2008). The relationship disappeared in Swedish individuals in the 1950s, in concurrence with the full implementation of their universal health care system (Cernerud 1993).

It is possible that the inverted U relationship between sibling number and ELL is due to limitations placed upon fertility that also limit growth. As these women were born into a developing country that did not have universal health care, access to family planning, vaccinations or clean water, it is highly unlikely that every birth in the family was planned. Thus the smallest families were likely small due to another factor that limited maternal (or paternal) fertility. Thus the small families may have poor growth because of an over-reaching negative factor. This factor could be that the mother was un-partnered, which resulted in reduced resources for the family, as men tend to earn more money than women in this context.

F. *Conclusion*

The recall of the Maya women's early life environment cannot be considered as accurate as data collected prospectively to measure the early life environment. However measures of early life that have been well established in the literature were associated with the linear growth and body proportions of these Maya women. Modernisation significantly predicted increased leg length, without increasing stature. Changes from the traditional lifestyle by being born in a city and frequently consuming packaged food and sugar sweetened beverage are not inherently associated with improved stature. However they appear to be related to more rapid lower limb growth. This population was greatly impacted by macro-economic trends of development (1960-1980) and recession (1981-1987). The stature of these Maya women increased with birth decade until 1981, where a secular decrease of over 3cm was observed. Also family size/ sibling number, birth order and sibling death all were associated with measures of linear growth.

Chapter 8. *Child's energy expenditure, stunting and body composition*

This chapter examines the relationship between urban Maya children's energy expenditure and stunting on anthropometric z-scores and body composition outcomes.

A. *Introduction*

Middle-income countries, such as Mexico and Brazil, are currently experiencing a rise in obesity levels among their poor (Mendez et al. 2005; Popkin et al. 2012). It is possible that this rise in obesity is following the pattern shown in populations in high income countries as globalized diets and lifestyles are adopted by the populations of lower income nations (Adair and Popkin 2005; Barquera et al. 2008; Caballero 2007; Popkin et al. 2012). See Chapter 2, section D for a more detailed discussion of the nutritional changes associated with the nutrition transition. However, additional causes for this rise in obesity may exist. Trends toward urbanisation, including rural-to-urban migration, with associated changes in physical activity are a possible cause (Katzmarzyk and Mason 2009; Monda et al. 2007; Parra et al. 2009) (Chapter 2, section G.1). In addition, the most impoverished segments of lesser-developed nations may have experienced a relatively rapid and acute shift from general protein-energy under-nutrition to a more complex mix of under-supply of specific amino acids, vitamins and minerals along with concurrent over-supply of energy (Popkin 2002) (Chapter 2, section D.1). There is indirect evidence from research with adults indicating that this change in the nature of malnutrition is accompanied by metabolic shifts, such as reduced fat oxidation with increased risk of overweight/obesity (Florencio et al. 2003; Leonard et al. 2009).

Low SES, urban, Maya children are the focus of this study. Maya living in the Mexican Yucatan Peninsula are currently undergoing nutrition transition (Leatherman and Goodman 2005; Leatherman et al. 2010) and are a double burdened population, with simultaneously high rates of stunting and overweight and obesity (Azcorra et al. 2009; Dickinson 1997; Varela Silva et al. 2012). See Chapter 2, section B and Chapter 4 for a more extensive description of the population and sample.

1. *Stunting and energy expenditure*

Stunted and short height-for-age individuals may be at an increased risk of overweight or obesity (OW/OB) during the growing years compared with their non-stunted peers (Bogin and Loucky 1997; Corvalan et al. 2008; Hoffman et al. 2007; Mardones et al. 2008; Martins et al. 2004; Smith et al. 2003; Walker et al. 2007). Changes in resting metabolic rate (RMR) occur during the first 5 years of life (Grillo et al. 2005) the same period that stunting most often occurs (Fox and Hillsdon 2007). A decreased metabolism could be an efficient method of adapting to energy

requirements in response to low caloric availability. Yet a slowing of the metabolism places individuals at an increased risk of weight gain in conditions of caloric abundance. The preliminary evidence suggests that metabolic shifts may be associated with the risk for obesity in stunted children (Grillo et al. 2005; Hoffman et al. 2000b; Soares-Wynter and Walker 1996; Wren et al. 1997) but the exact conditions this relationship requires have yet to be fully understood. The present study attempts to objectively estimate urban Maya children's RMR and determine whether it is influenced by stunting status. Chapter 2, section F.4 provides a more complete discussion of the relationship between under-nutrition and energetics.

2. *Modernisation and activity energy expenditure*

The changes in behaviour that occur as part of urbanisation and modernisation may play a substantial role in the nutrition-related, non-communicable diseases. The behavioural transition, associated with modernisation and the nutrition transition, is characterised by an increase in sedentary activity (Onywera 2010) and reduced occupational physical activity (Huneault et al. 2011; Popkin et al. 2002) in adults (see Chapter 2, section F.4 and G.4). Low levels of activity energy expenditure (AEE) and physical activity are risk factors for obesity (Fox and Hillsdon 2007; Gomez et al. 2007; Monda et al. 2008) (see Chapter 2, section G.4).

The relationship between physical activity and adiposity or OW/OB in children living in developing countries has been less studied compared to adults in the same environments and children in developed countries (DepartHealth 2004) (see Chapter 2, section G.4). Research with Latin American and Mexican children has found an increase in sedentary behaviour, such as television viewing (Hernandez et al. 1999; Malina et al. 2008b; Sauri Bazán 2003). However frequently participating in sedentary activities does not necessarily result in low physical activity levels (Biddle et al. 2004; Malina et al. 2008b). Thus, it is not clear what the relationship is between children's physical activity and adiposity or OW/OB in the nutrition transition. This present study presents objective measurements of the physical activity and AEE of urban Maya children and classifies them for risk of negative health outcomes.

3. *Actiheart*

This is one of the first studies to objectively assess energy expenditure using combined heart rate monitoring and accelerometry, the Actiheart, in free-living

children in developing countries. A previous study has been performed in adolescents in urban Brazil (Victora et al. 2008). The Actiheart allows for habitual physical activity to be assessed objectively and accurately (Assah et al. 2011; Corder et al. 2005) for up to 3 weeks at a time (Delisle 2008). The Actiheart was designed to minimize high discomfort and burden on the participant (*ibid*) and has been validated against gold standard techniques for energy expenditure such as double-labelled water (Catalano and Ehrenberg 2006) and indirect calorimetry (Brage et al. 2005; Corder et al. 2005). This makes it a very good tool for use in free-living participants, particularly ones whose activity patterns are largely unknown.

B. Aims

The purpose of this article is to report the physical activity levels of these low SES, urban Maya children and to assess whether chronic under-nutrition (stunting) impacts their energy expenditure. We hypothesize that stunting significantly reduces both resting and activity energy expenditure, independently of lean mass.

C. Methods

1. Sample

The study design was cross-sectional and conducted between March and July 2010. This sample is composed of 7.00-9.99 year old urban Maya school children, living in Merida, Yucatan, Mexico (Chapter 3, section B.1).

2. Recruitment

Schools located in the low SES neighbourhoods of southern Merida, known to contain a relatively high proportion of Maya families, were targeted. Children of Maya ethnicity, aged 7-9 years-old, were identified from school lists by having two Maya surnames, one from their mother and one from their father. The mothers were then invited to group information sessions at their children's schools where the nature of the study was verbally explained, in Spanish, and information sheets, in Spanish, were provided. The Maya living in the south of Merida were highly acculturated and all were comfortable speaking Spanish. See Chapter 3, section B.3 for full recruitment details.

Written informed consent was obtained from the mothers and verbal assent from the children. Ethical clearance was obtained from the Loughborough University Ethics Committee in the U.K. and the Bioethical Committee of CINVESTAV in Mexico. See Chapter 3, section B.2 for full ethical considerations.

3. *Anthropometry*

Children underwent anthropometric measurements for stature (Gordon et al. 1988), weight (*ibid*), waist circumference (WC) and skinfolds (triceps and sub-scapular) using standard techniques. See Chapter 3, section D.1 for complete list of anthropometric measures and technique. Body mass index (BMI) was calculated by dividing the child's weight in kilograms by their statures in metres squared. See Chapter 3, section E.2 for full details on derived variables.

The Comprehensive sex- and age-specific reference charts (Frisancho 2008) were used to calculate z-scores for BMI and WC. Children were classified as being stunted if their height-for-age was below the 5th percentile. See Chapter 3, section E.2 for full explanation of reference and classification choice.

4. *Bioelectrical impedance analysis*

Body composition was measured using bioelectric impedance analysis (BIA) with a BioScan 916 by Maltron (Chapter 3, section D.2). Percent body fat (%BF) was calculated using the impedance and reactance values with equations specific for American Indian children (Equation 1) (Lohman et al. 1999), see Chapter 3, section E.1. %BF was converted into kilograms of body fat, which was used to calculate fat free mass (FFM).

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

Definitions: Sex coded 1 for girls, 0 for boys. Weight is in kg. Skinfold thicknesses are in mm. Resistance and reactance are in ohms. Stature is in m.

5. *Energy expenditure*

Physical activity of the children was measured for 7 days using an Actiheart, a combined heart rate and accelerometer (Corder et al. 2007; Corder et al. 2005; Wilson et al. 2011). See Chapter 3, section D.3 for a more complete description of the Actiheart methodology. Children were included in the analysis if they had at least 3 days usable data that included a minimum of 2 weekdays and 1 weekend day. A day was defined as a minimum of 10 hours of usable data during waking hours (Ward et al. 2005) (Chapter 3, section E.3). Data was collected and analysed in minute-long epochs or sampling interval.

Energy expenditure was estimated using branched equation modelling in Actiheart software v.4.52. Data were cleaned by removing extended periods (>5 minutes) in which the heart rate data was missing and also when there was a mismatch in the heart rate and accelerometry data, for example, extended periods of high accelerometry counts and low heart rates (but not the reverse). The software performed straight line interpolation for periods of missing heart rate data lasting up to 5 minutes, allowing energy expenditure to be estimated for these periods. The period of data removal was extended if 5 minutes of missing heart rate data was preceded or followed by one minute of heart rate data and then another period of missing heart rate data. Chapter 3, section E.3 has a complete description of the data cleaning methodology used for the Actiheart data.

Resting energy expenditure (REE), activity energy expenditure (AEE) and total energy expenditure (TEE) were the key variables used in these analyses. All variables were calculated using predictive equations with an external reference curve created using 13 year old British children (Corder et al. 2005). REE was calculated using the Schofield equation (Schofield 1985). Sleeping heart rate was individually calculated for each child using the average heart rate during extended periods (>2 hours) of negligible accelerometry counts during night hours (12 pm to 9 am). AEE was calculated when the heart rate was above sleeping heart rate. TEE was calculated as the additive combination of REE, AEE and diet induced thermogenesis.

For assessment of the level of physical activity in which the children engaged, metabolic equivalent (MET) were calculated by the Actiheart software. The time at each MET were used to classify the activity level of each minute-long epoch as low (MET<3) or moderate-to-vigorous (MET ≥3).

6. *Statistical analysis*

Independent *t*-tests were used to compare chronic nutritional status (stunted v. non-stunted) and sex with anthropometric, body composition and energy expenditure variables. Energy expenditure variables were linearly regressed onto BMI z-score and WC z-scores and body composition variables.

Stepwise multiple linear regressions using the enter method were performed with measures of energy expenditure (REE, AEE and TEE) as the dependent variables. The independent variables were added in three steps: 1) FFM, 2) height-for-age z-score or stunting and 3) sex. Entering variables into the model in a stepwise fashion shows how the inclusion of variables influences the other variables in the model. This allows for a more complete understanding of the relationship between variables in the model compared to entering all variables in one step. Normality of the residuals were checked.

All analyses were undertaken using PASW (SPSS) v.18.0. Significance was set *a priori* at $p < 0.05$. For the multiple regression models, Bonferroni adjustments were performed to reduce the risk of a false positive by dividing the criteria *p* value by the number of steps in the model ($0.05/3$). Significance was finalized at $p < 0.02$.

D. *Results*

From the 58 children recruited, 33 were included in the final analysis. Children excluded were excluded for incomplete Actiheart data ($n=24$) and health problems ($n=1$). The reason for missing Actiheart data was primarily due to poor electrode adherence caused by high rates of sweating in the hot, humid climate of Merida resulting in missing heart rate data or the device falling off of the skin (Wilson et al. 2011). Also several Actihearts were broken during the course of the week. Also, the children were unlikely to notice when the electrode pads became loose, reducing the odds that the problem would be fixed. See Chapter 3, section E.3 and Appendix E for a more complete description of the logistical difficulties encountered when attempting to use Actihearts in children in a tropical climate. The included children were not statistically significantly different from excluded children for any measures of anthropometry or body composition (Chapter 5, section C).

Overall these urban Mexican Maya children were short and except for the non-stunted girls have low weight-for-age z-scores (Table 8.1). Stunted children

were significantly smaller in stature and weight, with less estimated FFM and FM than their non-stunted peers. BMI z-scores did not significantly differ between the stunted and non-stunted children.

Table 1 Anthropometric and body composition variables for urban Mexican Maya children

| | Stunted | | Non-Stunted | | All |
|--------------------------------------|---------------|---------------|---------------|---------------|---------------|
| | Boys | Girls | Boys | Girls | |
| N (%) | 5 (15.2) | 6 (18.2) | 12 (36.4) | 10 (30.3) | 33 (100) |
| Age | 8.24 (0.93) | 8.27 (0.96) | 8.20 (0.86) | 8.60 (0.71) | 8.34 (0.82) |
| Stature (cm)^a | 114.46 (4.25) | 115.23 (7.91) | 123.47 (4.22) | 126.12 (5.02) | 121.41 (6.96) |
| Stature z-score^{2,a} | -2.38 (0.53) | -2.02 (0.45) | -0.71 (0.68) | -0.66 (0.57) | -1.19 (0.92) |
| Weight (kg)^a | 22.56 (3.27) | 20.89 (4.77) | 26.15 (5.11) | 31.05 (8.37) | 26.14 (6.96) |
| Weight z-score^{2,a} | -1.17 (0.58) | -1.17 (0.41) | -0.49 (0.85) | 0.17 (0.79) | -0.52 (0.89) |
| BMI (kg/m²) | 17.13 (1.50) | 15.60 (1.91) | 17.06 (2.53) | 19.28 (3.79) | 17.48 (2.98) |
| BMI z-score² | 0.48 (0.58) | -0.25 (0.73) | 0.44 (0.91) | 0.87 (0.95) | 0.45 (0.90) |
| FFM^{3,a} | 16.51 (1.84) | 15.88 (3.13) | 18.98 (1.79) | 20.24 (3.12) | 18.43 (2.96) |
| %FFM³ | 73.81 (7.49) | 76.37 (3.71) | 73.79 (7.18) | 66.96 (7.69) | 7.71 (4.40) |
| FM^{3,a} | 6.05 (2.46) | 5.01 (1.81) | 7.17 (3.75) | 10.81 (5.45) | 72.19 (7.53) |
| %BF³ | 26.19 (7.50) | 23.63 (3.71) | 26.21 (7.18) | 33.04 (7.69) | 29.81 (7.53) |

¹Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference (2008).

²Age and sex specific z-scores based upon Frisancho's Comprehensive reference (2008).

³Calculated using an equation for American Indian children including bioelectrical impedance and anthropometry

^aSignificant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.01

Stunted children had lower levels of, REE, AEE and TEE (Table 8.2) compared to non-stunted children. Stunted children spent significantly more time in light physical activity and less time per day in moderate-to-vigorous physical activity (MVPA) than non-stunted children. No significant differences in REE, AEE or TEE were found between the sexes.

Overall, this sample of children was highly active, spending an average of 120 minutes per day in MVPA. However girls and stunted children spent significantly less time in MVPA compared to boys and non-stunted children, respectively. Of the 5 children who did not spend 60 minutes per day in MVPA, all were girls and 4 were stunted. The variation in time spent at each level of physical activity was high, with a range of 20-312 minutes per day spent in moderate-to-vigorous physical activity.

Table 2 Energy expenditure variables for urban Mexican Maya children

| | | Stunted | | Non-Stunted | | All |
|--|------------------------------------|---------------------|---------------------|-----------------------|----------------------|----------------------|
| | | Boys | Girls | Boys | Girls | |
| Resting energy expenditure² | kJ/day^b | 4221.00 (315.23) | 3813.17 (384.53) | 4542.25 (437.84) | 4615.90 (615.36) | 4383.33 (547.15) |
| | kJ/kg/day^b | 188.89 (15.02) | 185.99 (18.34) | 176.31 (15.05) | 153.24 (19.44) | 172.98 (21.50) |
| Activity energy expenditure² | kJ/day^b | 2022.20 (257.92) | 2340.50 (425.36) | 3711.67 (955.11) | 3411.60 (799.06) | 3272.27 (879.21) |
| | kJ/kg/day^d | 135.97 (15.19) | 114.26 (13.51) | 143.10 (32.08) | 112.09 (18.51) | 127.38 (26.78) |
| Total energy expenditure² | kJ/day^b | 8060.40 (594.34) | 6859.83 (858.29) | 9,171.00 (1393.84) | 8919.50 (1487.21) | 8506.30 (1485.79) |
| | kJ/kg/day^d | 360.95 (32.28) | 333.62 (30.23) | 354.90 (42.56) | 294.81 (36.81) | 33373 (45.13) |
| MET (min)³ | Light (<3)^{a,c} | 1327.00 (29.65) | 1380.33 (40.81) | 1275.92 (70.63) | 1323.10 (52.93) | 1316.94 (65.46) |
| | Mod-Vig (≥3)^{a,c} | 113.60 (29.84) | 60.00 (40.09) | 164.25 (70.30) | 116.70 (52.74) | 123.21 (65.21) |
| | | | | | | |

¹Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference (2008).

²Calculated in the Actiheart software using simultaneous heart rate and accelerometry data and an external reference curve (Corder 2005).

³Average number of minutes per day spent at each MET level

^a Significant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.05

^b Significant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.01

^c Significant difference found between the sexes using an independent *t*-test, *p*<0.05

^d Significant difference found between the sexes using an independent *t*-test, *p*<0.01

In multiple linear regression models (Table 8.3), kilograms of FFM was the largest predictor of all measures of energy expenditure (REE, AEE and TEE), explaining between 33 and 81% of the variance in energy expenditures (Table 4). Boys expended significantly more energy of all types (REE, AEE and TEE) than girls. Neither height-for-age z-scores nor stunting were significant predictors of REE. Height-for-age z-score but not stunting was a significant predictor for AEE and TEE.

Table 3 Estimated measures of energy expenditure predicted by absolute fat free mass, stature and sex in urban Mexican Maya children using multiple linear regression

| | Resting energy expenditure (kJ/day) | | | | | | Activity energy expenditure (kJ/day) | | | | | | Total energy expenditure (kJ/day) | | | | | |
|-----------------------------------|-------------------------------------|--------|---------------------|--------|---------------------|--------|--------------------------------------|--------|----------------------|-------|---------------------|-------|-----------------------------------|--------|----------------------|--------|----------------------|--------|
| | Model 1 | | Model 2 | | Model 3 | | Model 1 | | Model 2 | | Model 3 | | Model 1 | | Model 2 | | Model 3 | |
| | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P |
| Constant | 1297.51 (264.34) | <0.001 | 1675.07 (367.88) | <0.001 | 1716.65 (338.84) | <0.001 | -10.10 (798.22) | 0.990 | 1526.13 (1078.69) | 0.167 | 1646.29 (994.72) | 0.109 | 1430.70 (1087.01) | 0.198 | 3556.42 (1465.73) | 0.021 | 3736.13 (1323.30) | 0.009 |
| FFM (kg)^{1,2} | 167.48 (14.17) | <0.001 | 152.23 (17.46) | <0.001 | 154.77 (16.09) | <0.001 | 178.15 (42.79) | <0.001 | 116.08 (51.18) | 0.031 | 123.42 (47.33) | 0.014 | 384.02 (58.27) | <0.001 | 298.14 (69.55) | <0.001 | 309.11 (62.84) | <0.001 |
| Height z-score³ | | | 81.35 (56.13) | 0.158 | 78.29 (51.65) | 0.140 | | | 330.99 (164.57) | 0.053 | 322.15 (151.93) | 0.043 | | | 458.00 (223.62) | 0.049 | 444.79 (201.71) | 0.036 |
| Sex⁴ | | | | | -189.81 (74.76) | 0.017 | | | | | -548.45 (219.92) | 0.019 | | | | | -820.22 (29.98) | 0.009 |
| R² adj | 0.813 | | 0.819 | | 0.847 | | 0.338 | | 0.397 | | 0.486 | | 0.570 | | 0.610 | | 0.683 | |
| Constant | 1297.51 (264.34) | <0.001 | 1322.66 (341.02) | 0.001 | 1310.45 (314.92) | <0.001 | -10.10 (798.22) | 0.990 | 851.06 (997.89) | 0.400 | 818.43 (937.75) | 0.390 | 1430.70 (1087.01) | 0.198 | 2415.07 (1371.99) | 0.089 | 2365.24 (1263.20) | 0.071 |
| FFM (kg) | 167.48 (14.17) | <0.001 | 166.35 (17.24) | <0.001 | 171.50 (16.05) | <0.001 | 178.15 (42.79) | <0.001 | 139.302 (50.45) | 0.010 | 153.08 (47.80) | 0.003 | 384.02 (58.27) | <0.001 | 339.62 (69.36) | <0.001 | 360.65 (64.39) | <0.001 |
| Stunted^{5,6} | | | -12.74 (106.43) | 0.906 | 22.48 (99.29) | 0.822 | | | -436.32 (311.43) | 0.171 | -342.24 (295.65) | 0.256 | | | -498.75 (428.18) | 0.253 | -355.09 (398.26) | 0.380 |
| Sex | | | | | -194.98 (78.38) | 0.019 | | | | | -520.80 (233.40) | 0.034 | | | | | -798.56 (314.40) | 0.017 |
| R² adj | 0.813 | | 0.806 | | 0.835 | | 0.338 | | 0.358 | | 0.433 | | 0.570 | | 0.575 | | 0.640 | |

¹Fat free mass expressed as kilograms of body weight

²Calculated using an equation for American Indian children including bioelectrical impedance and anthropometry

³Height-for-age z-scores calculated using the age and sex specific curves of Frisancho's Comprehensive reference (2008).

⁴Boys set as reference

⁵Non-stunted set as reference

⁶Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference (2008).

E. Discussion

1. Stunting and energy expenditure

a. Stunting and resting energy expenditure

Our hypothesis that stunting, *per se*, is associated with lower estimated energy expenditure (resting + activity) in children is not supported. Rather children with short stature, as a continuous variable, and lower fat free mass (FFM) have lower total energy expenditure (TEE) compared to taller children with higher FFM. Stunted children had significantly lower absolute FFM than non-stunted children. For these Maya children the lower absolute amount of FFM in the stunted children may mitigate other factors and result in lower resting energy expenditure (REE).

Other studies find that stunting *per se* has effects. Soares-Wynter and Walker (1996) found lower REE in stunted Jamaican children aged 7-8 years old, when controlling for lean body mass. Our findings are similar to those of Grillo et al. (2005) who found in a case-controlled study in the shantytowns of Sao Paulo, Brazil that stunted girls had lower resting energy expenditure than age- and weight-for-height matched, non-stunted girls. Yet, in the same Brazilian cohort, Martins et al. (2004) report that the same stunted Brazilian have a significantly increased %BF and reduced %FFM compared to their non-stunted, age- and weight-for-height matched peers. Our new results find a trend toward just the opposite for body composition in girls.

The dominance of FFM over REE in this study is not surprising as FFM is the primary determinant of resting metabolic rate (RMR) (Malina et al 2004). The relationship between body composition and short stature is complex and depends on the complex interaction between the individual and population nutritional history and current diet and energy balance. In populations where under-nutrition is an acute problem as well as a chronic problem for the children (the expected and historically common case), it is likely that the fat mass (FM) in the stunted children will be lower because they are likely to be both chronically and acutely under-nourished. However the rapid speed of the nutrition transition in currently developing countries (Popkin 2002) can lead to a very quick rise in food availability in a local area or for a family. Thus children who are nutritionally stunted in infancy and early childhood (before 5

years of age) (Stein et al 2010) may experience an excess of available calories in childhood (6-9 years of age). The body composition of a stunted child in the latter situation will almost certainly be very different to that of a stunted child in the former situation.

This complex interplay between the individual's nutritional history and current nutritional situation is the likely reason for the inconsistent relationship between body composition and stunting found in the literature. A study in Sao Paulo, Brazil found that in mildly stunted, low income adolescents, the only clear difference in body composition by stunting status was abdominal fat, with stunted children having almost 10% more fat stored around their abdomens than non-stunted children. The body composition of the stunted, pre-pubertal girls was borderline significantly higher in %BF than non-stunted girls but there were no differences in body composition for the boys or pubescent girls (Grotti Clemente et al 2011).

b. *Stunting and activity energy expenditure*

The stunted children also have lower activity energy expenditure (AEE), a potential a behavioural modification. The differences in behaviour according to stature are clearly revealed by the Actiheart data, which show that the stunted children engage in significantly less moderate-to-vigorous activity than the non-stunted children.

The Maya findings also stand in contrast to the same stunted Brazilian children, which found equivalent AEEs in stunted and non-stunted children (Hoffman et al. 2000b). This finding indicates that chronic under-nutrition (stunting) does not impact the metabolic reserves of the children. It appears as though the Mexican Maya children of our study have a different relationship between stunting and physical activity levels than children in Sao Paulo. From these data on the Maya children, it is not possible to know whether the lower levels of physical activity in the stunted Maya are driven by biological or social pressures.

Extrapolating findings from a related field allows us to make an educated guess. Examining the physical fitness of under-nourished children allows us to indirectly estimate the metabolic reserves. In landmark study by Reina and Spurr in 1988 under-nourished children and adequately nourished children were all feed a hot lunch and then played organized sporting games. The under-nourished children

were not able to maintain the same level of activity as the adequately nourished children (Spurr and Reina 1988). From those findings, it is expected that under-nutrition leads to decreased metabolic reserves and thus lower physical capabilities. However acutely under-nourished (wasted) and chronically under-nourished (stunted) were combined in this classic study, making it difficult to draw conclusions about either type of under-nutrition. A more relevant study relating physical fitness with stunting specifically was performed in rural indigenous and urban children in Oaxaca, Mexico. This study by Malina and colleagues found that stunted children exhibit the similar fitness levels as non-stunted, adequately nourished children (Malina et al 2011). These findings suggest that in children chronic under-nutrition, in the absence of acute under-nutrition (*i.e.* stunting with appropriate BMI-for-age z-scores) the metabolic reserves of children are comparable to non-stunted children. It must be stated that this interpretation is highly speculative, being based upon only one study, and thus should be considered only as a possible direction for future studies.

If the stunted children have equivalent metabolic reserves to their non-stunted peers, and thus are physically capable of being as physically active, it suggests that the lower AEE in these stunted Maya children is due to cultural or other non-biological factors.

2. Sex and activity energy expenditure

The influence of cultural factors on AEE is notable in the finding that the only children who failed to spend an hour a day in MVPA were girls. That the majority of these girls (4 of 5) were stunted is suggestive of a biosocial or biocultural relationship related to the sex of the child. The Maya traditionally have very strong gender roles and traditional gender roles are still common in wider Mexican culture. While there are presently no data in this population on this issue, it is possible that boys are more encouraged to play sports or do more physically demanding play activities, while girls are encouraged or permitted to be more sedentary and participate in low intensity household activities. It is conceivable that cultural expectations may be more pronounced in girls who are small and therefore more likely to be considered delicate, leading to even less encouragement for stunted girls.

It is likely that this sample of girls will become less active in adolescence (Dumith et al. 2011), increasing their risk for obesity in adulthood. Reduced physical activity during adolescence is a common finding for girls in many cultures (Telama et al. 2005). The mothers of these children had very high levels of obesity (Varela Silva et al. 2012) (Chapter 5, section A.2) indicating that these girls are themselves at high risk for obesity. Indirect evidence also points to the stunted girls having very high risk of OW/OB (Florencio et al. 2003). It is likely that this differential is in part due to cultural factors such as the more physically demanding occupations of men, for example in the construction industry. Many countries have seen an increase in OW/OB levels when physically demanding occupations begin to be replaced by less energetically demanding occupations, such as those in the service industry.

More research has focused on the health effects of female double burden than male because of the intergenerational influences of the former. Children born to women with individual double burden are at risk for negative health effects related to both stunting and OW/OB. Stunted mothers are more likely to have children who are low birth weight (Victora et al. 2008), who die before their 5th birthday (Monden and Smits 2009) and who are stunted (Delisle 2008). Overweight mothers are more likely to miscarry, have gestational diabetes and have children who are overweight (Catalano and Ehrenberg 2006). Thus a population with a large number of women with individual double burden faces a public health concern in for multiple generations facing cycles of ill health.

This is not to discount the impact of a stunted father on children. Stunted men have reduced earning potential on average (Case and Paxson 2007). Lower income and socio-economic status is associated with multiple health problems in children (see Chapter 2, section C.2). Therefore both male and female stunting must be combated in order to most effectively break the cycle of poverty.

3. Activity energy expenditure and modernisation

Yet on the whole, even the stunted girls spent an average of 60 minutes per day in MVPA. This is a highly active sample, by the standards of developed countries, with 85% (n=28) spending at least one hour per day in moderate-to-vigorous physical activity. Recommendations are that children should be moderately to vigorously active for a minimum of 60 minutes a day (DepartHealth 2004) for independent reduction of chronic disease risk (Balas-Nakash et al. 2010; Bell et al.

2007). Such high levels of physical activity stand in contrast to data from developed countries, where the average time spent in MVPA is closer to 30 minutes (Ekelund et al. 2012) with only 30-40% spending at least 60 minutes per day in MVPA (Ekelund et al. 2011). Adult data relating lower levels of MVPA to urbanisation and modernisation have prompted some to extend the relationship to children.

It is worth noting that as this population continues to undergo modernisation, more sedentary activities will become available to them, such as the internet, video games and passive transport, the population is at risk of decreasing levels of physical activity. This is particularly true of adults in developing countries where occupations shift from labour intensive industries toward more service dominated industries. Therefore, while low time spent in MVPA is not a current issue in this sample, it is likely to become one in the future.

Lower energy expenditure may leave the stunted Maya children to be more susceptible to weight and fat gain if they ingest as much energy as the taller children. Stunted children have been found to be less able to regulate energy intake than their non-stunted, weight-for-height and age-matched peers (Grillo et al 2006).

Another possibility for the high rates of obesity in stunted individuals is that stunting is related to metabolic shifts toward altered substrate utilisation. For example, reduced fat oxidation (and therefore preferential fat storage) may exacerbate the risk to overweight and excessive fatness. A study in indigenous Siberians found that shorter adults had reduced fat oxidation compared to their taller peers (Leonard et al 2009). We did not measure directly metabolic activity at the physiological level, and this needs to be done to better evaluate our findings in light of past research.

4. *Strengths and limitations*

The applicability of the results of this study to other human groups is limited by a small sample size, the use of a group calibration curve for the energy expenditure estimation and a BIA predictive equation that was not specific to the Maya. Some strengths of this study are the variety of data collected simultaneously, especially the use of an objective and well-validated instrument, the Actiheart, to estimate energy expenditure. The cost and participant burden of traditional methods of objectively measuring energy expenditure have tended to limit their use in low

income groups of developing countries, with self-reported physical activity being the preferred method.

F. *Conclusion*

Complex interactions between body size, body composition and metabolic activity appear to elevate the risk for current and later life obesity in this sample of Maya children. These children were found to be highly active but girls and stunted children exhibited the lowest level of physical activity. In this sample, shorter children were less active than taller children, and stunted children spent significantly less time in MVPA than non-stunted children. The effect of stature seems to be mediated via the lower FFM of the shorter/stunted children and not primarily due to the stunting. Even so, an important point to make here is that lower FFM is associated with short stature/stunting and public health workers and policy planners may still use short stature as a proxy for higher risk of negative health outcomes.

Chapter 9. *Discussion and conclusions*

This chapter brings together the overall findings from each of the individual results chapter and discusses the main aims of the thesis, identifies gaps and limitations associated with the research. It also highlights policy implications of the findings.

A. *Key findings and implications*

This sample of urban Mexican Maya appears to be rapidly undergoing nutrition transition. In terms of nutritional status, this is a double burdened sample, with very high levels of adiposity coexisting with chronic under-nutrition in both the women and children. This fact places both age groups at a very high risk for negative health outcomes related to nutrition-related non-communicable diseases (NR-NCDs). These children's physical activity levels are relatively high and therefore not consistent with the behavioural transition towards sedentary behaviour which has been shown to accompany nutrition transition in other societies.

1. *Aim 1: Describe the current living conditions of the urban Maya of Merida*

This research has shown that these Maya are of low SES but live in an urban area of a middle-income country that has been working toward improving the health of its population for decades (Frenk et al 2006). This low-income, urban population appears to be on their way toward meeting the United Nations' Millennium Development Goals (UNICEF 2011). Most of the children have access to clean drinking water and there is no readily apparent gender inequality in these children in terms of school attendance or nutritional status. However the overall nutritional status of this sample of double burdened children is not ideal, even though acute under-nutrition in childhood does not appear to be a concern in this sample. The health care in this area appears to be relatively accessible and the vaccine rates in this sample seem to be high because of several mobile vaccination campaigns that visit the schools frequently. The vast majority of births (57 out of 58) took place in a free public hospital located within the broad community and almost all women received pre-natal care. The under-5 mortality decreased from an estimated 40% in the mother's generation to close to 5% in the

children's. Consuming clean drinking water increased from 39% of families when the mothers were children to 70% in the current generation. Also, reliance onto purchased foods (including sugar sweetened beverages) has greatly increased within a generation. Thus this sample selected from the urban Maya appears to be transitioning out of their tradition of systematic marginalisation and as such represents a case study of what may occur as part of very rapid nutritional transition of a marginalised group.

This sample of urban Maya appears to have all but eliminated acute under-nutrition in childhood and adulthood as no participant in this study was found to be wasted or under-weight. However OW/OB and excess adiposity appears to be very widespread in this population. Over 90% of the mothers and roughly 30% of the children were classified as OW/OB by BMI. These 7- to 9-year old children had a mean %BF of 28% and approximately 80% were above the 85th percentile for %BF-for-age. The high levels of adiposity in these young children place them at a higher risk for NR-NCDs than would be expected using solely BMI. The mothers are also at very high risk for such diseases as they have high levels of overall excess adiposity (approximately 90% of women have %BF over 33%) and abdominal obesity (mean WC = 88 cm, the cut-off for abdominal obesity in women (Lear et al. 2010)).

These high rates of OW/OB and excess adiposity support previous findings that document the rapid rise of OW/OB during rapid transition. The high level of adiposity among these pre-pubertal children is noteworthy. Most studies on obesity during the nutrition transition use BMI. The rates of OW/OB in these children when using BMI (30%) are relatively high but the rates of over-fat (OF) increase to 80% when %BF is used instead of BMI. Chronic disease risk factors are related to %BF in children, particularly at high levels of %BF (Going et al 2011). The prevalence of OW/OB based upon BMI in these children as they age may surpass the very high current prevalence in their mothers (>90%). Thus these Maya may be at risk for having one of the highest chronic disease burdens in the world, similar to Samoans. Some of the highest rates of OW/OB in adult women are found in

Samoans, who have been shown to have a proportionately high chronic disease burden (Keighley et al 2007).

This high prevalence of OW/OB and excess adiposity co-exists with chronic under-nutrition (stunting). The mother's are very short, with a mean stature of 147 cm (z-score= -2.152). The children are relatively taller than the mothers, in terms of z-scores or percentiles on growth charts, but still have a high prevalence of stunting (31%) and are all below the 50th percentile for height-for-age. Both short stature and OW/OB are associated with increased mortality and reduced economic potential. The Maya have previously been shown to have higher rates of stunting than of wasting (Crooks 1994, Jenkins 1981) and be shorter than low SES, non-Maya groups in the same geographical area (Bogin et al 1992).

While not directly comparable due to the age differences, the 2010 stunting rates of these 7-9 year old urban Maya (31%) are twice as high as the 2006 average for Mexican children aged <5 years (15.5%) (Lutter et al 2011). Thus these urban Maya have experienced very poor chronic nutrition in comparison to the Mexican average. The stunting rates of Mexico are middling compared to other Latin American middle-income countries. The under-5 stunting rates for other Latin American middle-income countries are: Costa Rica (5.6%), Brazil (6.8%), Argentina (8%), Colombia (16.3%) and Peru (29.8%) (Lutter et al 2011). Such wide ranges of stunting between countries are due to different political attitudes and will in different countries.

The prevalence of such double burdened populations is increasing throughout the world, particularly in middle-income countries (WHO 2009). It appears as though becoming doubled burdened is difficult to avoid without preventative measures in place for a population undergoing the very rapid epidemiological transition and modernisation that is common in low and middle-income countries. Investigating double burdened populations may lead to methods for avoiding double burden in other transitioning groups, especially in low-income settings.

It is well known that the choice of criteria for classifying individuals impacts the appearance of anthropometric data. For example, much research has been

published on the impact of adopting WHO growth standards in populations that were not exclusively breast fed or high SES (Norris et al 2009, Prost et al 2010, Wang et al 2009). Even growth references from different populations can impact the appearance of the data (Janssen et al 2005). Therefore it is not surprising that the choice of reference curve and cut-offs impacts the appearance of these data on urban Maya. What is surprising is the degree to which the data are apparently changed. The prevalence of stunting is 15.5% using the -2 z-score of Frisancho's Comprehensive reference versus 37.9% using the 5th percentile of the WHO reference. When using the -2 z-score cut-off, the difference between the Comprehensive reference and the WHO reference is 11% for these urban Maya children. This is in comparison to a 6% difference in stunting prevalence between the CDC 2000 charts and the WHO 2006 standards in a sample of children from urban South Africa (Norris et al 2009). The large impact of the cut-offs and reference curves used for stunting classification are likely due to the clustering of these urban Maya children around the stunting cut-off points.

The choice of the measure for overweight also impacts the results substantially. Less than 10% of the sample has a high weight-for-age, while over 80% of the sample has %BF over the 85th percentile for their age and sex. When using BMI as the measure of overweight, roughly 30% of the sample is overweight or obese. The difference in prevalence rates between different reference charts for BMI is far less than for stunting. Reference curves for adiposity have only become available in recent years and few researchers have classified children as OF. Therefore it is unclear what impact using different %BF reference charts will have on the classifications of children as OF. When examining a sample such as this, the classification criterion for malnutrition requires careful selection and interpretation. This is particularly important when disseminating information to policy makers, as they will be unlikely to have the time to understand the complexities of the data.

2. *Aim 2: Determine whether BMI predicts adiposity indicators in a sample with a high prevalence of stunting*

BMI z-scores appear to be useful in predicting adiposity indicators in these urban Maya children, particularly in predicting total body adiposity and central adiposity. Total body adiposity and visceral adiposity are more closely related to chronic disease risk than peripheral adiposity. Therefore the close relationship between BMI and total body fat and visceral adiposity is useful for easily classifying children as at risk for negative health outcomes using BMI. While BMI is not a complete measure of nutritional status, it provides a broad over-view with minimal effort. This is particularly beneficial in clinical settings such as in the south of Merida, where doctors split their time between the public hospital in the south and the more lucrative private hospitals and clinics located in other parts of the city. The time restrictions placed on the doctors are likely very high, requiring them to maximise their efficiency, which will be maximised when they are able to use simple indicators like BMI to assess risk.

It must be noted however that if BMI is the only measure of nutritional status used, the complete picture will be missed due to the short stature of these children. For example, this sample of Maya children is short (chronically under-nourished) but have normal to high BMIs and therefore would be classified as adequately or over-nourished if BMI is used in isolation from stature. It is therefore recommended that BMI is used in conjunction with other measures such as stature and WC in order to correctly identify the health risks of these children. Measuring WC in stunted children may be especially useful in identifying risk as stunted children may be at an increased risk of abdominal obesity as the interaction between stunting and RMR significantly predicted higher WC z-scores in children.

Neither stunting status nor relative leg length impacted the power of BMI to predict adiposity indicators in any model. This indicates that BMI can be used in Maya populations of women and children without a correction for leg length or stunting status, which has not been previously found in stunted populations. Such a correction has been found to increase the usefulness of BMI in non-Western

populations with relatively long legs (Australian Aborigines) (Norgan and Jones 1995). That leg length does not impact the relationship between BMI and adiposity indicators in these short Maya may be due to a lack of variance in the statures and body proportions of this sample.

In contrast to its use in children, BMI is not recommended for use in predicting adiposity indicators for these urban Maya women. For the mothers, BMI significantly predicted waist circumference but no other measure of adiposity. This lack of relationship between total body and peripheral adiposity reduces the usefulness of BMI as a health screen tool in these Maya women. BMI has also been found to be a less powerful predictor of risk factors for NR-NCDs in stunted Mexican adults (Lara-Esqueda et al. 2004) These Maya women had very high levels of abdominal obesity, which is closely related to negative health outcomes (Huxley et al 2010, Seidell 2010). It may be more appropriate to replace BMI in clinical settings with WC for Maya women. WC is simpler to measure than BMI, requiring only one measurement and no calculations. Thus it is likely that measuring WC will be an efficient use of time in clinical settings, particularly when time constraints are high. When possible, more measures of nutritional status should be made such as stature, weight and %BF (possibly using BIA), particularly as this population is at very high risk for NR-NCDs related to obesity.

3. Aim 3: Determine whether measures of adult women's linear growth can be predicted by her recalled childhood environment

It appears as though macro- and community-level factors during childhood play a larger role in these Maya women's linear growth than recalled, individual SES. This was evidenced by results showing that the decade in which the mothers were born was the strongest predictor of stature. Furthermore leg length and SHR were best predicted by modernisation (estimated in part by city living). Overall, this research has shown that the relationship between recalled childhood environment and linear growth measures is present but weak. This indicates that recalled childhood environment should not be used as the only measures of chronic conditions that occurred in early life.

This study found that the Maya women's recalled estimates of modernisation in early life and her family size were related to measures of leg growth (SHR and leg length). Stature appeared to be more influenced by childhood material wealth (consumer durable ownership), macro-economic trends (women's birth decade) and sibling death. Indicators of family resources influence both leg length and stature but explain the least amount of variance.

Women from families with fewer resources or a lower ability to invest in a single child are shorter and have shorter leg lengths (relative and absolute). This agrees with the literature, which has found an influence of material wealth (Webb et al 2008) and family size (Gunnell et al 1998) on linear growth. Sibling death was associated with lower stature, perhaps indicating that sibling death is indicative of wider problems experienced by the family that occurred throughout the women's childhoods. It was advantageous for women to be first born. This is likely because they would have experienced reduced competition for resources, such as attention and food, compared to children born of higher parity. First born women had longer legs than those women born with older siblings. However pure sibling competition does not fully explain the relationship between linear growth and family size. The women who had 5-7 siblings had the longest legs of the sample. Thus it appears as though being in a small family may have been a disadvantage, perhaps because small family size may be an indicator of a limiter placed on the family. Since these mothers were born at a time when family planning was not widespread in Mexico, particularly in low SES groups, a small family may indicate that the parents had a health problem or were rarely together. These complex relationships between family demographics and women's linear growth measures highlight how it is necessary to use multiple measures of chronic environments in order to capture the complexity of the interaction between environment and lifestyles across the life courses.

Modernisation was positively related to and explained the largest amount of the variance in both proportional and absolute leg length models. While the factors used to assess modernisation (city living, sugar sweetened beverage and packaged food consumption) are likely indicative of more modern overall living

conditions. Also, sugar sweetened beverage and packaged food are energy-dense, and sugar sweetened beverage has been linked to higher energy intakes in high-income countries (Stookey et al 2007) and increased body weight (Berkey et al 2004). This increase in energy may be sufficient for improved linear growth in these Maya women when they were young. Also, living in urban areas is often associated with improved linear growth, which has been linked to improved access to services and health care (Oyhenart et al. 2008; Smith et al. 2005). It is likely that the consumption of sugar sweetened beverage and packaged foods is also indicative of a larger amount of market integration of the community and income of the family.

Macro-economic trends were the single most important recalled factor predicting stature included in this analysis. Women born in the relatively affluent 1970s (Santander 2008) were significantly and approximately 3 cm taller in adulthood than women born during the 1960s or in the 1980s recession. A similar, though smaller (2 cm), influence of birth decade on adult stature has also been found in other populations in Mexico (Malina et al 2010). Finding an influence of birth decade on linear growth in such a small sample was unexpected as typically the influence of macro-economics is assessed using large data sets (Steckel 2009). A significant influence of birth decade, which is one of the dominant factors predicting women's stature signifies how closely the living conditions of this population were tied to the wider economy in the late twentieth century. It must be noted that even the women born in the tallest decade (1970s) were stunted on average (mean stature = 148 cm). Therefore even when the population was doing relatively well in terms of nutritional status compared to other decades, they were still marginalised and had very high rates of under-nutrition.

4. Aim 4: Determine whether the relationship between estimated free-living energy expenditure and body composition is altered by stunting

These children were highly active, being well over the internationally recommended 60 minutes of moderate-vigorous physical activity each day (DepartHealth 2004), although activity energy expenditure did not impact body composition. Physical activity is known to reduce NR-NCD risk factors independently of body composition (*ibid*). This indicates that physical activity in urban Maya children should be promoted for its own health benefits, not as a weight control tool for pre-pubertal children. The dominant predictor of body composition was resting energy expenditure (REE). The dominance of lean mass on REE is in keeping with previous research which found that the influence of stunting on body composition was related to REE (Hoffman et al 2000, Soares-Wynter and Walker 1996).

Obtaining accurate, objective measures of physical activity and energy expenditure is difficult under the best of conditions (McClain and Tudor-Locke 2009). The logistical challenges are greatly increased when measuring children in a new and harsh environment. This research used a combined heart rate and accelerometer, an Actiheart, for an objective measurement of energy expenditure and physical activity. Using the Actihearts in children in a tropical climate required a high amount of participant contact in order to obtain usable data (Wilson et al 2011). However the standard protocol for the use of Actihearts had to be adapted for use in the children living in a tropical climate. While the Actiheart is simple to wear, it was unfamiliar to these urban Maya who required instructions (verbal and written) and familiarisation with the device. The instructions had to be given to the mothers and caregivers due to the young age of the children. Also, to avoid breakage of the Actihearts, the mothers were asked to limit their children's rough play and to have their children wear shirts, which proved an effective strategy to preserve the devices but may have reduced the children's physical activity. The tropical climate also caused many problems, primarily related to the reduced adhesion of the ECG pads. In the hot and humid weather of the Yucatan, the pads

did not reliably adhere for longer than 4 days, causing lost heart rate data. The loss of adhesion required the field workers to change the pads midway through the week of monitoring. Including extra pads along with the instruction sheet also increased the data that was collected. All of these factors will be important for other researchers to be aware of when using these devices in similar settings to objectively measure activity levels.

B. *Strengths*

One of the main strengths of this research is the high quality, in depth data that was collected. The anthropometric assessors all underwent reliability training which ensures that the anthropometric measurements are standardised. The interview was piloted and altered to be more appropriate to the population in order to increase the quality of the data collected. Also, multiple measures of SES were collected in order to capture several dimensions of the living environment. Such high quality data ensures that the results of the study are reflective of the relationships as they actually occur within the sample.

The objective measurement of physical activity and objective estimation of energy expenditure is incredibly important for assessing the risk of OW/OB in a population. In low-income communities of developing countries, it is uncommon for such objective measurements to be taken, self-report is often used. Self-report is cost-effective but tends to be biased. Objective estimate of energy expenditure allows for the risk of excess adiposity and negative health outcomes to be more accurately assessed.

The interpretation of these analyses as placed in a biosocial framework, which acknowledges that people exist in and are influenced by a wide range of factors that include societal and biological. The levels of influences on an individual can be at the any level, from the global, as was seen with the influence of the global economy on the Maya mother's stature, to the individual, as can be seen with the negative influence of obesity on employment. Each of these levels influences all other levels in a complex web of interactions. Ignoring the complexity of the relationships between society and biology overly simplifies the influences on

health and reduces the applicability of the results to affect change in the relevant populations. Interpreting the results within this complex network of relationships, the interactions between societal and biological factors can be more fully understood, increasing the effectiveness of evidence-based change attempted.

C. *Limitations*

As with all of research, the data used in this thesis have their limitations. Some of the limitations are related to the development of my knowledge throughout the learning process of the PhD, some are related to limitations in the methods available for accurately assessing measures in a field environment, and others are due to the constraints of resources available. Firstly, no measure of maternal education was collected. Maternal education is a very common measure of SES and has been shown to directly influence her own health as well as her children's health (Campbell et al 2006). As such, having a measure of maternal education would have provided a much more complete picture of the SES of the family. The variable was accidentally omitted in the first draft of the interview, constructed by HW, and the omission was not noticed until fieldwork was almost complete. The research group at Cinvestav in Merida have a policy of keeping participant burden to a minimum (in line with ethical principles) and it was not deemed acceptable to re-contact all of the mothers to gain this additional information. Despite the lack of a measure of maternal education, the data set does contain a variety of other types of measures of SES, which have been shown to be associated with a number of the anthropometric measures taken from the sample.

Secondly, this sample is likely to be over-representative of stay-at-home mothers. The sampling technique (inviting mothers to an information session at the school at the beginning of the school day) and the schedule of the local field assistants (not available for field-work on nights or weekends) lead to the majority of participating mothers having a schedule that was flexible during the week. Such a schedule is less likely to occur when the mother is working full time. Families with a mother who works outside the home may have different health outcomes due to a variety of factors related to the differences in the mothers' schedules. However

the increased money brought into the household by a working mother may help to offset any negative impacts of the more limited time she is able to spend with her children.

Thirdly, the lack of usable dietary data greatly limits the analysis that can be done on the predictors of adiposity and body composition. The food frequency questionnaire did not give specify a time frame for the consumption of foods. The lack of time frame given as part of the FFQ results in data that is far more reflective of what the individuals think they are eating, rather than what they are actually consuming. Such data was less relevant to the questions addressed by this research than the actual dietary intake, which is far more important to nutritional status than perceived intake. As adiposity is related to energy intake and dietary quality as well as energy expenditure, quality dietary data would have been very useful in further testing the research questions being addressed in this thesis.

Fourthly, the energy expenditure data lacks individual calibration, increasing error. There are well known inter-individual differences in heart rate (Corder et al. 2005). Since the Actiheart was placed on the children during school hours, the time requirements of performing the step test for individual calibration (15 minutes) (CamNtech 2009) in addition to collecting other measurements for the study such as anthropometric data precluded the application of the step test. As the Actiheart is a relatively new device, only a small number of group calibration curves exist. Therefore, a group calibration curve based upon 13 year old English children was used (Corder et al. 2005). This increases the likely error in the estimations of energy expenditure because it is unknown how similar or different the Maya children in this study would be to the estimates provided by that curve. The amount of error introduced is unknown but it is likely substantial enough to disallow the comparison of the values of energy expenditure to other studies. However, since these Maya children have similar genetics and have experienced similar conditions throughout their lives, comparisons within the sample are likely to be appropriate.

Due to a limited time for and resources to support fieldwork and a small field team (typically only 2-3 researchers), with limited time available for data collection, the number of participants that could be sampled was small, which

reduced statistical power (Cohen 1992). Also, emphasis was placed on interviewing technique to maximise the quality of the data, which reduced the number of interviews each field worker could perform in one day. The emphasis on quality over quantity limited the power of the statistical tests.

D. *Suggestions for future research*

These findings suggest that the focus of future research needs to include measures of macro- and community-level factors including food availability and cost as well as measures of SES, perhaps examining food balance sheets. It is known that macro-level factors play a large role in an individual's health (Reyes et al. 2010; Sheehy and Sharma 2010). As much of the world is undergoing rapid urbanisation and epidemiological transition, it would be very useful to understand the relationship between factors related to urbanisation and health in terms of the effects of specific goods and services. The majority of the research in this area in transitioning economics has not given an in depth consideration to the influences of access to goods and services and the influence on health outcomes related to chronic disease risk.

Also, research into the metabolic rates and energy expenditures of younger children should be done to determine the influences on metabolic rates and whether this stunted population has decreased fat oxidation, measured by indirect calorimetry, which increases their risk of excess adiposity. The dominance of RMR in predicting body composition in combination with the high levels of adiposity in these Maya children This indicates that factors occurring earlier in life than 7-9 years are likely the main factors of concern as RMR is influenced heavily by early life events (Hoffman et al. 2000b). Extending this research into young children would be very useful in understanding the relationship between early life under-nutrition and later obesity.

Another area for potential future research relates to substrate utilisation. Evidence in adults indicates that the preferential storage of fats and burning of carbohydrates for energy, rather than energy expenditure, may occur in stunted individuals, increasing their risk of adiposity (Leonard et al. 2009). Preferential

utilisation of carbohydrates in the diet of stunted individuals may lead to storage of fat, increasing the risk of OW/OB.

It is also clear that future studies assessing the prevalence of specific NR-NCD risk factors, such as hypertension, dyslipidemia and glucose intolerance, must be performed in double burdened populations, such as the urban Maya. The prevalence as well as the relationship between chronic under-nutrition (stunting) and adiposity with NR-NCD risk factors needs to be explored in more detail. While some of these studies have occurred, they tend to focus on individually double burdened adults or household double burden (Delisle 2008; Florencio et al. 2001). The high levels of adiposity and normal BMI z-scores of these short children suggest that they may have risk factors early in life. This thesis has laid the ground work to begin to understand these associations but further work with larger samples is needed to better understand the combined relationships between these factors.

E. *Concluding remarks*

The Maya of southern Merida are a population undergoing rapid nutrition transition. The long history of marginalisation and the rapid transition of the population combined with their genetic homogeneity (Ibarra-Rivera et al. 2008) make the Maya a good population for examining the consequences of the nutrition transition on chronic disease outcomes. From this small sample, the urban Maya of Merida appear to be a double burdened population. The problems traditionally facing low SES populations in developing countries (e.g. lack of schooling, health care, clean drinking water) have broadly been overcome in this urban Maya sample. This is supported by the children having higher height-for-age z-scores (-1.15) than their mothers (mean -1.99). Though conditions seem to be improving in Merida, the high rates of stunting coupled with the high rates of excess adiposity found in this sample indicate that these urban Maya will face chronic disease burdens that are similar to or greater than those currently being experienced by high income countries.

Combating the burden of NR-NCDs requires knowledge of appropriate tools and the most effective timings for interventions. BMI is best applied to urban Maya children, while it might be more appropriate for WC to be used in Maya women to screen for obesity. Interventions to reduce childhood adiposity need to begin very early in life for best results. Research into the low SES groups of middle-income countries, offers insight to what may occur in low-income countries as they advance in the nutrition transition.

Literature cited

www.mathgoodies.com/calculators/random_no_custom.html.

- Adair LS, and Popkin BM. 2005. Are child eating patterns being transformed globally? *Obesity Research* 13(7):1281-1299.
- Aekplakorn W, Hogan MC, Chongsuvivatwong V, Tatsanavivat P, Chariyalertsak S, Boonthum A, Tiptaradol S, and Lim SS. 2007. Trends in obesity and associations with education and urban or rural residence in Thailand. *Obesity (Silver Spring)* 15(12):3113-3121.
- Alderman H, Hoddinott J, and Kinsey B. 2006. Long term consequences of early childhood malnutrition. *Oxford Economic Papers-New Series* 58(3):450-474.
- Amuna P, and Zotor FB. 2008. Epidemiological and nutrition transition in developing countries: impact on human health and development. *Proc Nutr Soc* 67(1):82-90.
- ANAPRAC. 2005. La industria de refrescos y aguas carbonatadas en 2005: anuario estadístico.
- Aquino R, de Oliveira NF, and Barreto ML. 2009. Impact of the family health program on infant mortality in Brazilian municipalities. *American Journal of Public Health* 99(1):87-93.
- Arvidsson D, Slinde F, and Hulthen L. 2009. Free-living energy expenditure in children using multi-sensor activity monitors. *Clinical Nutrition* 28(3):305-312.
- Assah FK, Ekelund U, Brage S, Wright A, Mbanya JC, and Wareham NJ. 2011. Accuracy and validity of a combined heart rate and motion sensor for the measurement of free-living physical activity energy expenditure in adults in Cameroon. *International Journal of Epidemiology* 40(1):112-120.
- Azcorra H. 2007. Migracion familiar y crecimiento infantil en Merida, Yucatan, Mexico. Merida, Yucatan, Mexico: Unidad Merida. 137 p.
- Azcorra H, Dickinson F, and Rothenberg SJ. 2009. Family Migration and Physical Growth in Merida, Yucatan, Mexico. *American Journal of Human Biology* 21(3):398-400.
- Baker J, Hurtado AM, Pearson OM, Hill KR, Jones T, and Frey MA. 2009. Developmental plasticity in fat patterning of Ache children in response to variation in interbirth intervals: a preliminary test of the roles of external environment and maternal reproductive strategies. *Am J Hum Biol* 21(1):77-83.
- Balas-Nakash M, Benitez-Arciniega A, Perichart-Perera O, Valdes-Ramos R, and Vadillo-Ortega F. 2010. The effect of exercise on cardiovascular risk markers in Mexican school-aged children: comparison between two structured group routines. *Salud Publica De Mexico* 52(5):398-405.
- Bandini LG, Vu DM, Must A, and Dietz WH. 1997. Body fatness and bioelectrical impedance in non-obese pre-menarcheal girls: comparison to anthropometry and evaluation of predictive equations. *European Journal of Clinical Nutrition* 51(10):673-677.

- Barbeau P, Johnson MH, Howe CA, Allison J, Davis CL, Gutin B, and Lemmon CR. 2007. Ten months of exercise improves general and visceral adiposity, bone, and fitness in black girls. *Obesity (Silver Spring)* 15(8):2077-2085.
- Barker DJ. 1993. Maternal nutrition and cardiovascular disease. *Nutr Health* 9(2):99-106.
- Barker DJ. 1995a. The fetal and infant origins of disease. *Eur J Clin Invest* 25(7):457-463.
- Barker DJ. 1995b. Fetal origins of coronary heart disease. *Bmj* 311(6998):171-174.
- Barker DJ, Forsen T, Eriksson JG, and Osmond C. 2002. Growth and living conditions in childhood and hypertension in adult life: a longitudinal study. *J Hypertens* 20(10):1951-1956.
- Barker DJ, Osmond C, Golding J, Kuh D, and Wadsworth ME. 1989a. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. *Bmj* 298(6673):564-567.
- Barker DJ, Osmond C, Kajantie E, and Eriksson JG. 2009. Growth and chronic disease: findings in the Helsinki Birth Cohort. *Annals of Human Biology* 36(5):445-458.
- Barker DJ, Winter PD, Osmond C, Margetts B, and Simmonds SJ. 1989b. Weight in infancy and death from ischaemic heart disease. *Lancet* 2(8663):577-580.
- Barker DJP, Kajantie E, Osmond C, Thornburg KL, and Eriksson JG. 2011. How Boys Grow Determines How Long They Live. *American Journal of Human Biology* 23(3):412-416.
- Barquera S, Hernandez-Barrera L, Tolentino ML, Espinosa J, Ng SW, Rivera JA, and Popkin BM. 2008. Energy Intake from Beverages Is Increasing among Mexican Adolescents and Adults. *Journal of Nutrition* 138(12):2454-2461.
- Barquera S, Peterson KE, Must A, Rogers BL, Flores M, Houser R, Monterrubio E, and Rivera-Dommarco JA. 2007. Coexistence of maternal central adiposity and child stunting in Mexico. *Int J Obes (Lond)* 31(4):601-607.
- Barros FC, Victora CG, Barros AJ, Santos IS, Albernaz E, Matijasevich A, Domingues MR, Sclowitz IK, Hallal PC, Silveira MF and others. 2005. The challenge of reducing neonatal mortality in middle-income countries: findings from three Brazilian birth cohorts in 1982, 1993, and 2004. *Lancet* 365(9462):847-854.
- Baur JA, Pearson KJ, Price NL, Jamieson HA, Lerin C, Kalra A, Prabhu VV, Allard JS, Lopez-Lluch G, Lewis K and others. 2006. Resveratrol improves health and survival of mice on a high-calorie diet. *Nature* 444(7117):337-342.
- Behrman JR, and Skoufias E. 2004. Correlates and determinants of child anthropometrics in Latin America: background and overview of the symposium. *Econ Hum Biol* 2(3):335-351.
- Bell B. 2006 6 October. Cola Wars in Mexico. In *These Times*.
- Bell LM, Watts K, Siafarikas A, Thompson A, Ratnam N, Bulsara M, Finn J, O'Driscoll G, Green DJ, Jones TW and others. 2007. Exercise alone reduces insulin resistance in obese children independently of changes in body composition. *J Clin Endocrinol Metab* 92(11):4230-4235.
- Beltaifa L, Traissac P, El Ati J, Lefevre P, Romdhane HB, and Delpeuch F. 2009. Prevalence of obesity and associated socioeconomic factors among Tunisian women from different living environments. *Obes Rev* 10(2):145-153.

- Bergstrom A, Pisani P, Tenet V, Wolk A, and Adami HO. 2001. Overweight as an avoidable cause of cancer in Europe. *Int J Cancer* 91(3):421-430.
- Beydoun MA, Powell LM, and Wang Y. 2008. The association of fast food, fruit and vegetable prices with dietary intakes among US adults: is there modification by family income? *Soc Sci Med* 66(11):2218-2229.
- Biddle SJ, Gorely T, and Stensel DJ. 2004. Health-enhancing physical activity and sedentary behaviour in children and adolescents. *J Sports Sci* 22(8):679-701.
- Bjerregaard JK, and Dahl-Petersen IK. 2011. How well does social variation mirror secular change in prevalence of cardiovascular risk factors in a country in transition? *American Journal of Human Biology*.
- Bogin B. 1999. *Patterns of Human Growth*. Cambridge: Cambridge University Press.
- Bogin B, and Beydoun MA. 2007. The relationship of sitting height ratio to body mass index and fatness in the United States, 1988-1994. *Human Ecology* S15:1-8.
- Bogin B, and Keep R. 1999. Eight thousand years of economic and political history in Latin America revealed by anthropometry. *Ann Hum Biol* 26(4):333-351.
- Bogin B, and Loucky J. 1997. Plasticity, political economy, and physical growth status of Guatemala Maya children living in the United States. *Am J Phys Anthropol* 102(1):17-32.
- Bogin B, and MacVean RB. 1981. Biosocial effects of urban migration on the development of families and children in Guatemala. *Am J Public Health* 71(12):1373-1377.
- Bogin B, and MacVean RB. 1983. The relationship of socioeconomic status and sex to body size, skeletal maturation, and cognitive status of Guatemala City schoolchildren. *Child Development* 54:115-128.
- Bogin B, Smith P, Orden AB, Varela Silva MI, and Loucky J. 2002. Rapid change in height and body proportions of Maya American children. *Am J Hum Biol* 14(6):753-761.
- Bogin B, and Varela-Silva MI. 2008. Fatness biases the use of estimated leg length as an epidemiological marker for adults in the NHANES III sample. *International Journal of Epidemiology* 37:201- 209.
- Bogin B, and Varela-Silva MI. 2010. Leg length, body proportion, and health: a review with a note on beauty. *Int J Environ Res Public Health* 7(3):1047-1075.
- Bollen KA, Glanville JL, and Stecklov G. 2001. Socioeconomic status and class in studies of fertility and health in developing countries. *Annual Review of Sociology* 27:153-185.
- Bonilla C, Gutierrez G, Parra EJ, Kline C, and Shriver MD. 2005. Admixture analysis of a rural population of the state of Guerrero, Mexico. *Am J Phys Anthropol* 128(4):861-869.
- Bosy-Westphal A, Plachta-Danielzik S, Dorhofer RP, and Muller MJ. 2009. Short stature and obesity: positive association in adults but inverse association in children and adolescents. *Br J Nutr* 102(3):453-461.
- Brage S, Brage N, Franks PW, Ekelund U, and Wareham NJ. 2005. Reliability and validity of the combined heart rate and movement sensor Actiheart. *European Journal of Clinical Nutrition* 59(4):561-570.
- Bunt JC, Tataranni PA, and Salbe AD. 2005. Intrauterine exposure to diabetes is a determinant of hemoglobin A(1)c and systolic blood pressure in pima Indian children. *J Clin Endocrinol Metab* 90(6):3225-3229.

- Burnett N. 2008. Education for all: an imperative for reducing poverty. *Ann N Y Acad Sci* 1136:269-275.
- Butte NF, Moon JK, Wong WW, Hopkinson JM, and Smith EO. 1995. Energy requirements from infancy to adulthood. *American Journal of Clinical Nutrition* 62(5 Suppl):1047S-1052S.
- Butte NF, Wong WW, Adolph AL, Puyau MR, Vohra FA, and Zakeri IF. 2010. Validation of cross-sectional time series and multivariate adaptive regression splines models for the prediction of energy expenditure in children and adolescents using doubly labeled water. *Journal of Nutrition* 140(8):1516-1523.
- Caballero B. 2007. The global epidemic of obesity: an overview. *Epidemiol Rev* 29:1-5.
- Cameron N. 2002. Human growth curve, canalization, and catch-up growth. In: Cameron N, editor. *Human growth and development*. Amsterdam: Academic Publishers. p 1-20.
- Cameron N, and Demerath EW. 2002. Critical periods in human growth and their relationship to diseases of aging. *Am J Phys Anthropol Suppl* 35:159-184.
- Cameron N, Johnston FE, Kgampe JS, and Lunz R. 1992. Body-Fat Patterning in Rural South-African Black-Children. *American Journal of Human Biology* 4(3):353-364.
- Cameron N, Jones LL, Griffiths PL, Norris SA, and Pettifor JM. 2009. How well do waist circumference and body mass index reflect body composition in pre-pubertal children? *European Journal of Clinical Nutrition* 63(9):1065-1070.
- Cameron N, Wright MM, Griffiths PL, Norris SA, and Pettifor JM. 2005a. Stunting at 2 years in relation to body composition at 9 years in African urban children. *Obes Res* 13(1):131-136.
- Cameron N, Wright MM, Griffiths PL, Norris SA, and Pettifor JM. 2005b. Stunting at 2 years in relation to body composition at 9 years in African urban children. *Obes Res* 13(1):131-136.
- CamNtech. 2009. The Actiheart User Manual: v. 4.0.34. Cambridge: CamNtech Ltd. 125 p.
- Campbell DI, Elia M, and Lunn PG. 2003. Growth faltering in rural Gambian infants is associated with impaired small intestinal barrier function, leading to endotoxemia and systemic inflammation. *Journal of Nutrition* 133(5):1332-1338.
- Carpenter KM, Hasin DS, Allison DB, and Faith MS. 2000. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: Results from a general population study. *American Journal of Public Health* 90(2):251-257.
- Casagrande SS, Franco M, Gittelsohn J, Zonderman AB, Evans MK, Fanelli Kuczmarski M, and Gary-Webb TL. 2011. Healthy food availability and the association with BMI in Baltimore, Maryland. *Public Health Nutr* 14(6):1001-1007.
- Case A, Fertig A, and Paxson C. 2005. The lasting impact of childhood health and circumstance. *Journal of Health Economics* 24(2):365-389.
- Case A, and Paxson C. 2006. Children's health and social mobility. *Future Child* 16(2):151-173.

- Case A, and Paxson C. 2008. Stature and status: Height, ability, and labor market outcomes. *Journal of Political Economy* 116(3):499-532.
- Case A, and Paxson C. 2010. Causes and consequences of early-life health. *Demography* 47 Suppl:S65-85.
- Catalano P, and Ehrenberg H. 2006. The short- and long-term implications of maternal obesity on the mother and her offspring. *BJOG* 113:1126-1133.
- CDC. 2009. About BMI for Adults. In: Division of Nutrition PAaO, National Center for Chronic Disease Prevention and Health Promotion, editor. *Healthy Weight: Center for Disease Control*.
- Cernerud L. 1993. The association between height and some structural social variables: a study of 10-year-old children in Stockholm during 40 years. *Annals of Human Biology* 20(5):469-476.
- Chattopadhyay A, White MJ, and Debuur C. 2006. Migrant fertility in Ghana: selection versus adaptation and disruption as causal mechanisms. *Popul Stud (Camb)* 60(2):189-203.
- Checkley W, Buckley G, Gilman RH, Assis AM, Guerrant RL, Morris SS, Molbak K, Valentiner-Branth P, Lanata CF, and Black RE. 2008. Multi-country analysis of the effects of diarrhoea on childhood stunting. *Int J Epidemiol* 37(4):816-830.
- Chumlea WCG, S. S. 2002. The assessment of human growth. In: Cameron N, editor. *Human growth and development*. Amsterdam: Academic Publishers. p 249- 362.
- Clement K, Vaisse C, Manning BS, Basdevant A, Guygrand B, Ruiz J, Silver KD, Shuldiner AR, Froguel P, and Strosberg AD. 1995. Genetic-Variation in the Beta(3)-Adrenergic Receptor and an Increased Capacity to Gain Weight in Patients with Morbid-Obesity. *New England Journal of Medicine* 333(6):352-354.
- Clemes SA, Matchett N, and Wane SL. 2008. Reactivity: an issue for short-term pedometer studies? *Br J Sports Med* 42(1):68-70.
- Cohen J. 1992. A Power Primer. *Psychological Bulletin* 112(1):115-159.
- Cohen S, Janicki-Deverts D, Chen E, and Matthews KA. 2010. Childhood socioeconomic status and adult health. *Ann N Y Acad Sci* 1186:37-55.
- Colditz GA. 1999. Economic costs of obesity and inactivity. *Medicine and Science in Sports and Exercise* 31(11):S663-S667.
- Colin-Ramirez E, Castillo-Martinez L, Orea-Tejeda A, Romero ARV, Castaneda AV, and Lafuente EA. 2009. Waist Circumference and Fat Intake Are Associated with High Blood Pressure in Mexican Children Aged 8 to 10 Years. *Journal of the American Dietetic Association* 109(6):996-1003.
- Coly AN, Milet J, Diallo A, Ndiaye T, Benefice E, Simondon F, Wade S, and Simondon KB. 2006. Preschool stunting, adolescent migration, catch-up growth, and adult height in young senegalese men and women of rural origin. *J Nutr* 136(9):2412-2420.
- Corder K, Brage S, Mattocks C, Ness A, Riddoch C, Wareham NJ, and Ekelund U. 2007. Comparison of two methods to assess PAEE during six activities in children. *Med Sci Sports Exerc* 39(12):2180-2188.
- Corder K, Brage S, Wareham NJ, and Ekelund U. 2005. Comparison of PAEE from combined and separate heart rate and movement models in children. *Med Sci Sports Exerc* 37(10):1761-1767.

- Corvalan C, Uauy R, Flores R, Kleinbaum D, and Martorell R. 2008. Reductions in the energy content of meals served in the Chilean National Nursery School Council Program did not consistently decrease obesity among beneficiaries. *Journal of Nutrition* 138(11):2237-2243.
- Crooks DL. 1994. Growth Status of School-Age Mayan Children in Belize, Central-America. *American Journal of Physical Anthropology* 93(2):217-227.
- Cummins S, and Macintyre S. 2002. "Food deserts"--evidence and assumption in health policy making. *Bmj* 325(7361):436-438.
- Darmon N, and Drewnowski A. 2008. Does social class predict diet quality? *American Journal of Clinical Nutrition* 87(5):1107-1117.
- Darmon N, Ferguson EL, and Briend A. 2002. A cost constraint alone has adverse effects on food selection and nutrient density: an analysis of human diets by linear programming. *J Nutr* 132(12):3764-3771.
- de Onis M, and Blossner M. 2003. The World Health Organization Global Database on Child Growth and Malnutrition: methodology and applications. *International Journal of Epidemiology* 32(4):518-526.
- Delisle HF. 2008. Poverty: the double burden of malnutrition in mothers and the intergenerational impact. *Annals of the New York Academy of Sciences* 1136:172-184.
- DepartHealth. 2004. At least five a week. In: Department of Health PA, Health Improvement and Prevention, editor. London.
- Deurenberg P, Yap MD, Wang J, Lin FP, and Schmidt G. 1999. The impact of body build on the relationship between body mass index and percent body fat. *International Journal of Obesity* 23(5):537-542.
- DiBello JR, McGarvey ST, Kraft P, Goldberg R, Campos H, Quested C, Laumoli TS, and Baylin A. 2009. Dietary patterns are associated with metabolic syndrome in adult Samoans. *Journal of Nutrition* 139(10):1933-1943.
- Dickinson F. 1997. Desnutrición y obesidad en poblaciones Yucatecas. In: Aréchiga Viramontes J, and Bertran Vilá M, editors. *Significación Sociocultural de la Variación Morfológica*. México, D. F.: Universidad Nacional Autónoma de México. p 69-88.
- Dietz WH. 1994. Critical periods in childhood for the development of obesity. *Am J Clin Nutr* 59(5):955-959.
- Doak CM, Adair LS, Bentley M, Monteiro C, and Popkin BM. 2005. The dual burden household and the nutrition transition paradox. *Int J Obes (Lond)* 29(1):129-136.
- Doak CM, Adair LS, Monteiro C, and Popkin BM. 2000. Overweight and underweight coexist within households in Brazil, China and Russia. *J Nutr* 130(12):2965-2971.
- Drake AJ, and Walker BR. 2004. The intergenerational effects of fetal programming: non-genomic mechanisms for the inheritance of low birth weight and cardiovascular risk. *J Endocrinol* 180(1):1-16.
- Drewnowski A. 2004. Obesity and the food environment: dietary energy density and diet costs. *Am J Prev Med* 27(3 Suppl):154-162.
- Drewnowski A, Almiron-Roig E, Marmonier C, and Lluch A. 2004. Dietary energy density and body weight: is there a relationship? *Nutr Rev* 62(11):403-413.

- Drewnowski A, and Specter SE. 2004. Poverty and obesity: the role of energy density and energy costs. *American Journal of Clinical Nutrition* 79(1):6-16.
- Dumith SC, Gigante DP, Domingues MR, and Kohl HW, 3rd. 2011. Physical activity change during adolescence: a systematic review and a pooled analysis. *International Journal of Epidemiology* 40(3):685-698.
- Dumortier O, Blondeau B, Duvillie B, Reusens B, Breant B, and Remacle C. 2007. Different mechanisms operating during different critical time-windows reduce rat fetal beta cell mass due to a maternal low-protein or low-energy diet. *Diabetologia* 50(12):2495-2503.
- Dwyer JT, Gardner J, Halvorsen K, Krall EA, Cohen A, and Valadian I. 1989. Memory of food intake in the distant past. *Am J Epidemiol* 130(5):1033-1046.
- Ekelund U, Luan J, Sherar LB, Esliger DW, Griew P, and Cooper A. 2012. Moderate to vigorous physical activity and sedentary time and cardiometabolic risk factors in children and adolescents. *JAMA* 307(7):704-712.
- Ekelund U, Tomkinson G, and Armstrong N. 2011. What proportion of youth are physically active? Measurement issues, levels and recent time trends. *Br J Sports Med* 45(11):859-865.
- Ellis KJ. 2001. Selected body composition methods can be used in field studies. *Journal of Nutrition* 131(5):1589S-1595S.
- Ellis KJ, Abrams SA, and Wong WW. 1999. Monitoring childhood obesity: assessment of the weight/height² index. *American Journal of Epidemiology* 150(9):939- 946.
- Ellison PT. 2002. Puberty. In: Cameron N, editor. *Human Growth and Development*. Amsterdam: Academic Press. p 434.
- Engle PL, and Black MM. 2008. The effect of poverty on child development and educational outcomes. *Ann N Y Acad Sci* 1136:243-256.
- Eriksson JG, Forsen T, Tuomilehto J, Osmond C, and Barker DJ. 2001. Early growth and coronary heart disease in later life: longitudinal study. *Bmj* 322(7292):949-953.
- Fernald LC. 2007. Socio-economic status and body mass index in low-income Mexican adults. *Soc Sci Med* 64(10):2030-2042.
- Fernald LC, Gutierrez JP, Neufeld LM, Olaiz G, Bertozzi SM, Mietus-Snyder M, and Gertler PJ. 2004. High prevalence of obesity among the poor in Mexico. *Jama* 291(21):2544-2545.
- Fezeu L, Minkoulou E, Balkau B, Kengne AP, Awah P, Unwin N, Alberti GK, and Mbanya JC. 2006. Association between socioeconomic status and adiposity in urban Cameroon. *International Journal of Epidemiology* 35(1):105-111.
- Field A. 2005. *Discovering statistics using SPSS*. London: Sage Publications. 779 p.
- Filmer D, and Pritchett LH. 2001. Estimating wealth effects without expenditure data--or tears: an application to educational enrollments in states of India. *Demography* 38(1):115-132.
- Flegal KM, Graubard BI, Williamson DF, and Gail MH. 2005. Excess deaths associated with underweight, overweight, and obesity. *Jama* 293(15):1861-1867.
- Florencio TM, Ferreira HS, de Franca AP, Cavalcante JC, and Sawaya AL. 2001. Obesity and undernutrition in a very-low-income population in the city of Maceio, northeastern Brazil. *Br J Nutr* 86(2):277-284.

- Florencio TT, Ferreira HS, Cavalcante JC, Luciano SM, and Sawaya AL. 2003. Food consumed does not account for the higher prevalence of obesity among stunted adults in a very-low-income population in the Northeast of Brazil (Maceio, Alagoas). *European Journal of Clinical Nutrition* 57(11):1437-1446.
- Florencio TT, Ferreira HS, Cavalcante JC, Stux GR, and Sawaya AL. 2007. Short stature, abdominal obesity, insulin resistance and alterations in lipid profile in very low-income women living in Maceio, north-eastern Brazil. *European Journal of Cardiovascular Prevention & Rehabilitation* 14(2):346-348.
- Floyd B. 2007. Focused life history data and linear enamel hypoplasia to help explain intergenerational variation in relative leg length within Taiwanese families. *Am J Hum Biol* 19(3):358-375.
- Floyd B. 2008. Intergenerational gains in relative knee height as compared to gains in relative leg length within Taiwanese families. *American Journal of Human Biology* 20(4):462-464.
- Floyd B. 2009. Associations between height, body mass, and frequency of decayed, extracted, and filled deciduous teeth among two cohorts of Taiwanese first graders. *Am J Phys Anthropol* 140(1):113-119.
- Fotso JC. 2007. Urban-rural differentials in child malnutrition: trends and socioeconomic correlates in sub-Saharan Africa. *Health Place* 13(1):205-223.
- Fotso JC, and Kuate-Defo B. 2005. Socioeconomic inequalities in early childhood malnutrition and morbidity: modification of the household-level effects by the community SES. *Health Place* 11(3):205-225.
- Fowler-Brown A, and Kahwati LC. 2004. Prevention and treatment of overweight in children and adolescents. *American Family Physician* 69(11):2591-2598.
- Fox KR, and Hillsdon M. 2007. Physical activity and obesity. *Obes Rev* 8 Suppl 1:115-121.
- Frankenfield DC, Rowe WA, Cooney RN, Smith JS, and Becker D. 2001. Limits of body mass index to detect obesity and predict body composition. *Nutrition* 17(1):26-30.
- Freedman DS, Khan LK, Dietz WH, Srinivasan SR, and Berenson GS. 2001. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: The Bogalusa Heart Study. *Pediatrics* 108(3):712-718.
- Frenk J, Sepulveda J, Gomez-Dantes O, and Knaul F. 2003. Evidence-based health policy: three generations of reform in Mexico. *Lancet* 362(9396):1667-1671.
- Frisancho AR. 2003. Reduced rate of fat oxidation: a metabolic pathway to obesity in the developing nations. *Am J Hum Biol* 15(4):522-532.
- Frisancho AR. 2007. Relative leg length as a biological marker to trace the developmental history of individuals and populations: Growth delay and increased body fat. *American Journal of Human Biology* 19(5):703-710.
- 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. 335 p.
- Frisancho AR, Gilding N, and Tanner S. 2001. Growth of leg length is reflected in socio-economic differences. *Acta Med Auxol* 33(1):47- 50.
- Fuhrer R, Head J, and Marmot MG. 1999. Social position, age, and memory performance in the Whitehall II Study. *Ann N Y Acad Sci* 896:359-362.

- Fuke Y, Okabe S, Kajiwara N, Suastika K, Budhiarta AA, Maehata S, and Taniguchi H. 2007. Increase of visceral fat area in Indonesians and Japanese with normal BMI. *Diabetes Res Clin Pract* 77 Suppl 1:S224-227.
- Gabrielsson BG, Johansson JM, Lonn M, Jernas M, Olbers T, Peltonen M, Larsson I, Lonn L, Sjostrom L, Carlsson B and others. 2003. High expression of complement components in omental adipose tissue in obese men. *Obesity Research* 11(6):699-708.
- Gallo EA, Anselmi L, Dumith SC, Scazufca M, Menezes AM, Hallal PC, and Matijasevich A. 2011. [Size at birth and mental health problems at 11 years of age in a Brazilian birth cohort]. *Cad Saude Publica* 27(8):1622-1632.
- Garnier D, Simondon KB, Hoarau T, and Benefice E. 2003. Impact of the health and living conditions of migrant and non-migrant Senegalese adolescent girls on their nutritional status and growth. *Public Health Nutr* 6(6):535-547.
- Garrett J, and Ruel MT. 2005. The coexistence of child undernutrition and maternal overweight: prevalence, hypotheses, and programme and policy implications. *Matern Child Nutr* 1(3):185-196.
- Gates MF. What non-profits can learn from Coca-cola. In: TEDxChange, editor; 2010 10 September 2010; New York City, New York. TED.
- Glass TA, and McAtee MJ. 2006. Behavioral science at the crossroads in public health: extending horizons, envisioning the future. *Social Science & Medicine* 62(7):1650-1671.
- Gluckman PD, and Hanson MA. 2004. Developmental origins of disease paradigm: a mechanistic and evolutionary perspective. *Pediatr Res* 56(3):311-317.
- Gluckman PD, Hanson MA, Cooper C, and Thornburg KL. 2008. Effect of in utero and early-life conditions on adult health and disease. *N Engl J Med* 359(1):61-73.
- Goldenberg RL, Culhane JF, Iams JD, and Romero R. 2008. Epidemiology and causes of preterm birth. *Lancet* 371(9606):75-84.
- Gomez LF, Parra DC, Lobelo F, Samper B, Moreno J, Jacoby E, Lucumi DI, Matsudo S, and Borda C. 2007. Television viewing and its association with overweight in Colombian children: results from the 2005 National Nutrition Survey: a cross sectional study. *Int J Behav Nutr Phys Act* 4:41.
- Gonzalez-Martin A, Gorostiza A, Rangel-Villalobos H, Acunha V, Barrot C, Sanchez C, Ortega M, Gene M, and Calderon R. 2008. Analyzing the genetic structure of the Tepehua in relation to other neighbouring Mesoamerican populations. A study based on allele frequencies of STR markers. *Am J Hum Biol* 20(5):605-613.
- Gordon C, Chumlea C, and Roche A. 1988. Stature, recumbent length and weight. In: Lohman T, Roche A, and Martorell R, editors. *Anthropometric standardization reference manual (abridged edition)*. Champaign, IL: Human Kinetics. p 3-8.
- Goto R, Mascie-Taylor CG, and Lunn PG. 2009. Impact of intestinal permeability, inflammation status and parasitic infections on infant growth faltering in rural Bangladesh. *Br J Nutr* 101(10):1509-1516.
- Griffiths PL, Rousham EK, Norris SA, Pettifor JM, and Cameron N. 2008. Socio-economic status and body composition outcomes in urban South African children. *Arch Dis Child* 93(10):862-867.

- Grillo LP, Siqueira AFA, Silva AC, Martins PA, Verreschi ITN, and Sawaya AL. 2005. Lower resting metabolic rate and higher velocity of weight gain in a prospective study of stunted vs nonstunted girls living in the shantytowns of Sao Paulo, Brazil. *European Journal of Clinical Nutrition* 59(7):835-842.
- Gunnell D, Smith GD, McConnachie A, Greenwood R, Upton M, and Frankel S. 1999. Separating in-utero and postnatal influences on later disease. *Lancet* 354(9189):1526-1527.
- Gunnell DJ, Smith GD, Frankel S, Nanchahal K, Braddon FEM, Pemberton J, and Peters TJ. 1998a. Childhood leg length and adult mortality: follow up of the Carnegie (Boyd Orr) Survey of Diet and Health in Pre-war Britain. *Journal of Epidemiology and Community Health* 52(3):142-152.
- Gunnell DJ, Smith GD, Frankel SJ, Kemp M, and Peters TJ. 1998b. Socio-economic and dietary influences on leg length and trunk length in childhood: a reanalysis of the Carnegie (Boyd Orr) survey of diet and health in prewar Britain (1937-39). *Paediatric and Perinatal Epidemiology* 12:96-113.
- Guo SS, Chumlea WC, Roche AF, and Siervogel RM. 1997. Age- and maturity-related changes in body composition during adolescence into adulthood: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord* 21(12):1167-1175.
- Haas JD, Martinez EJ, Murdoch S, Conlisk E, Rivera JA, and Martorell R. 1995. Nutritional supplementation during the preschool years and physical work capacity in adolescent and young adult Guatemalans. *J Nutr* 125(4 Suppl):1078S-1089S.
- Habicht JP, Martorell R, and Rivera JA. 1995a. Nutritional Impact of Supplementation in the Incap Longitudinal-Study - Analytic Strategies and Inferences. *Journal of Nutrition* 125(4):S1042-S1050.
- Habicht JP, Martorell R, and Rivera JA. 1995b. Nutritional impact of supplementation in the INCAP longitudinal study: analytic strategies and inferences. *J Nutr* 125(4 Suppl):1042S-1050S.
- Hall DM, and Cole TJ. 2006. What use is the BMI? *Arch Dis Child* 91(4):283-286.
- Harris TB, Visser M, Everhart J, Cauley J, Tylavsky F, Fuerst T, Zamboni M, Taaffe D, Resnick HE, Scherzinger A and others. 2000. Waist circumference and sagittal diameter reflect total body fat better than visceral fat in older men and women. The Health, Aging and Body Composition Study. *Ann N Y Acad Sci* 904:462-473.
- Hebert PR, Richedwards JW, Manson JE, Ridker PM, Cook NR, Oconnor GT, Buring JE, and Hennekens CH. 1993. Height and Incidence of Cardiovascular-Disease in Male Physicians. *Circulation* 88(4):1437-1443.
- Heckman JJ. 2008. Role of income and family influence on child outcomes. *Ann N Y Acad Sci* 1136:307-323.
- Helmke I. 2011. A decade towards better health in Chile. *Bulletin of the World Health Organization* 89:710-711.
- Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM, and Parra-Cabrera S. 1999. Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico city. *Int J Obes Relat Metab Disord* 23(8):845-854.
- Heys M, Jiang C, Cheng KK, Zhang W, Lam TH, Leung GM, and Schooling CM. 2011. Does childhood meat eating contribute to sex differences in risk factors

- for ischaemic heart disease in a developing population? *J Epidemiol Community Health* 65(6):522-528.
- Hoffman DJ, Martins PA, Roberts SB, and Sawaya AL. 2007. Body fat distribution in stunted compared with normal-height children from the shantytowns of Sao Paulo, Brazil. *Nutrition* 23(9):640-646.
- Hoffman DJ, Roberts SB, Verreschi I, Martins PA, de Nascimento C, Tucker KL, and Sawaya AL. 2000a. Regulation of energy intake may be impaired in nutritionally stunted children from the shantytowns of Sao Paulo, Brazil. *J Nutr* 130(9):2265-2270.
- Hoffman DJ, Sawaya AL, Coward WA, Wright A, Martins PA, de Nascimento C, Tucker KL, and Roberts SB. 2000b. Energy expenditure of stunted and nonstunted boys and girls living in the shantytowns of Sao Paulo, Brazil. *Am J Clin Nutr* 72(4):1025-1031.
- Hoffman DJ, Sawaya AL, Martins PA, McCrory MA, and Roberts SB. 2006. Comparison of techniques to evaluate adiposity in stunted and nonstunted children. *Pediatrics* 117(4):e725-732.
- Hoffman DJ, Sawaya AL, Verreschi I, Tucker KL, and Roberts SB. 2000c. Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate and fat oxidation in shantytown children from Sao Paulo, Brazil. *Am J Clin Nutr* 72(3):702-707.
- Hosegood V, and Campbell OM. 2003. Body mass index, height, weight, arm circumference, and mortality in rural Bangladeshi women: a 19-y longitudinal study. *American Journal of Clinical Nutrition* 77(2):341-347.
- Huneault L, Mathieu ME, and Tremblay A. 2011. Globalization and modernization: an obesogenic combination. *Obes Rev* 12(5):e64-72.
- Huxley R, Mendis S, Zheleznyakov E, Reddy S, and Chan J. 2010. Body mass index, waist circumference and waist:hip ratio as predictors of cardiovascular risk--a review of the literature. *European Journal of Clinical Nutrition* 64(1):16-22.
- Hyman IE, and Billings FJ. 1998. Individual differences and the creation of false childhood memories. *Memory* 6:1-20.
- Ibarra-Rivera L, Mirabal S, Regueiro MM, and Herrera RJ. 2008. Delineating genetic relationships among the Maya. *Am J Phys Anthropol* 135(3):329-347.
- Jafar TH, Hatcher J, and Bhutta ZA. 2008. Rapidly rising rates of overweight and obesity coupled with persistently high rates of undernutrition among school aged children in an urban Indo-Asian population: authors' response. *Arch Dis Child* 93(11):1000-1001.
- James WP. 2006. The challenge of childhood obesity. *Int J Pediatr Obes* 1(1):7-10.
- James WPT. 2008. The epidemiology of obesity: the size of the problem. *Journal of Internal Medicine* 263(4):336-352.
- Jamison DT, and Mosley WH. 1991. Disease control priorities in developing countries: health policy responses to epidemiological change. *American Journal of Public Health* 81:15-22.
- Jauregui A, Villalpando S, Rangel-Baltazar E, Castro-Hernandez J, Lara-Zamudio Y, and Mendez-Gomez-Humaran I. 2011. The physical activity level of Mexican children decreases upon entry to elementary school. *Salud Publica Mex* 53(3):228-236.

- Jehn M, and Brewis A. 2009. Paradoxical malnutrition in mother-child pairs: untangling the phenomenon of over- and under-nutrition in underdeveloped economies. *Econ Hum Biol* 7(1):28-35.
- Jenkins CL. 1981. Patterns of growth and malnutrition among preschoolers in Belize. *Am J Phys Anthropol* 56(2):169-178.
- Jinabhai CC, Taylor M, and Sullivan KR. 2003. Implications of the prevalence of stunting, overweight and obesity amongst South African primary school children: a possible nutritional transition? *European Journal of Clinical Nutrition* 57(2):358-365.
- Johnston FE, Low SM, de Baessa Y, and MacVean RB. 1985. Growth status of disadvantaged urban Guatemalan children of a resettled community. *Am J Phys Anthropol* 68(2):215-224.
- Jones LL, Griffiths PL, Adair LS, Norris SA, Richter LM, and Cameron N. 2008. A comparison of the socio-economic determinants of growth retardation in South African and Filipino infants. *Public Health Nutr* 11(12):1220-1228.
- Jorgensen ME, Moustgaard H, Bjerregaard P, and Borch-Johnsen K. 2006. Gender differences in the association between westernization and metabolic risk among Greenland Inuit. *Eur J Epidemiol* 21(10):741-748.
- Katzmarzyk PT, and Mason C. 2009. The physical activity transition. *J Phys Act Health* 6(3):269-280.
- Kelishadi R. 2007. Childhood overweight, obesity, and the metabolic syndrome in developing countries. *Epidemiol Rev* 29:62-76.
- Kelles A, and Adair L. 2009. Offspring consume a more obesogenic diet than mothers in response to changing socioeconomic status and urbanization in Cebu, Philippines. *Int J Behav Nutr Phys Act* 6:47.
- Kimani-Murage EW, Kahn K, Pettifor JM, Tollman SM, Dunger DB, Gomez-Olive XF, and Norris SA. 2010. The prevalence of stunting, overweight and obesity, and metabolic disease risk in rural South African children. *BMC Public Health* 10.
- Kinra S, Sarma KV, Hards M, Smith GD, and Ben-Shlomo Y. 2011. Is relative leg length a biomarker of childhood nutrition? Long-term follow-up of the Hyderabad Nutrition Trial. *International Journal of Epidemiology* 40(4):1022-1029.
- Komlos J. 1985. Stature and nutrition in the Habsburg monarchy: the standard of living and economic development in the eighteenth century. *Am Hist Rev* 90(5):1149-1161.
- Komlos J. 1998. Shrinking in a growing economy? The mystery of physical stature during the industrial revolution. *Journal of Economic History* 58(3):779-802.
- Komlos J. 1999. On the biological standard of living in Russia and the Soviet Union. *Slavic Review* 58(1):71-79.
- Komlos J. 2008. Stagnation of heights among second-generation US-born army personnel. *Social Science Quarterly* 89(2):445-455.
- Komlos J, and Coclanis P. 1997. On the puzzling cycle in the biological standard of living: The case of antebellum Georgia. *Explorations in Economic History* 34(4):433-459.
- Komlos J, and Lauderdale BE. 2007. Underperformance in affluence: The remarkable relative decline in US heights in the second half of the 20th century. *Social Science Quarterly* 88(2):283-305.

- Kontogianni MD, Panagiotakos DB, and Skopouli FN. 2005. Does body mass index reflect adequately the body fat content in perimenopausal women? *Maturitas* 51(3):307-313.
- Kuzawa CW. 2005. Fetal origins of developmental plasticity: are fetal cues reliable predictors of future nutritional environments? *Am J Hum Biol* 17(1):5-21.
- Kuzawa CW, and Pike IL. 2005. Introduction. Fetal origins of developmental plasticity. *Am J Hum Biol* 17(1):1-4.
- Langenberg C, Shipley MJ, Batty GD, and Marmot MG. 2005. Adult socioeconomic position and the association between height and coronary heart disease mortality: findings from 33 years of follow-up in the Whitehall Study. *American Journal of Public Health* 95(4):628-632.
- Lara-Esqueda A, Aguilar-Salinas CA, Velazquez-Monroy O, Gomez-Perez FJ, Rosas-Peralta M, Mehta R, and Tapia-Conyer R. 2004. The body mass index is a less-sensitive tool for detecting cases with obesity-associated co-morbidities in short stature subjects. *International Journal of Obesity* 28(11):1443-1450.
- Lasker GW, and Mascie-Taylor CG. 1989. Effects of social class differences and social mobility on growth in height, weight and body mass index in a British cohort. *Annals of Human Biology* 16(1):1- 8.
- Lau DC, Dhillon B, Yan H, Szmitko PE, and Verma S. 2005. Adipokines: molecular links between obesity and atherosclerosis. *American Journal of Physiology-Heart and Circulation Physiology* 288:H2031-H2041.
- Lawson DW, and Mace R. 2008. Sibling configuration and childhood growth in contemporary British families. *International Journal of Epidemiology* 37(6):1408-1421.
- Lear SA, James PT, Ko GT, and Kumanyika S. 2010. Appropriateness of waist circumference and waist-to-hip ratio cutoffs for different ethnic groups. *European Journal of Clinical Nutrition* 64(1):42-61.
- Leatherman TL, and Goodman A. 2005. Coca-colonization of diets in the Yucatan. *Social Science & Medicine* 61(4):833-846.
- Leatherman TL, Goodman AH, and Stillman T. 2010. Changes in stature, weight, and nutritional status with tourism-based economic development in the Yucatan. *Econ Hum Biol* 8(2):153-158.
- Leatherman TL, Stillman JT, and Goodman AH. 2000. The effects of tourism-led development on the nutritional status of Yucatec Mayan children. Paper presented at the Annual Meeting of the American Association of Physical Anthropologists, April 2000. San Antonio, TX. *American Journal of Physical Anthropology Supplement* 30:207.
- Lee CMY, Huxley RR, Wildman RP, and Woodward M. 2008. Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: a meta-analysis. *Journal of Clinical Epidemiology* 61(7):646-653.
- Leitch I. 1951. Growth and health. *Br J Nutr* 5(1):142-151.
- Leonard WR, Sorensen MV, Mosher MJ, Spitsyn V, and Comuzzie AG. 2009. Reduced Fat Oxidation and Obesity Risks Among the Buryat of Southern Siberia. *American Journal of Human Biology* 21(5):664-670.

- Li L, Dangour AD, and Power C. 2007. Early life influences on adult leg and trunk length in the 1958 British birth cohort. *American Journal of Human Biology* 19(6):836-843.
- Lobstein T, and Jackson-Leach R. 2006. Estimated burden of paediatric obesity and co-morbidities in Europe. Part 2. Numbers of children with indicators of obesity-related disease. *International Journal of Pediatric Obesity* 1(1):33-41.
- Lohman TG, Caballero B, Himes JH, Hunsberger S, Reid R, Stewart D, and Skipper B. 1999. Body composition assessment in American Indian children. *American Journal of Clinical Nutrition* 69(4 Suppl):764S-766S.
- Lohman TG, Roche AF, and Martorell R. 1988. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics Books.
- Lopez-Alvarenga JC, Montesinos-Cabrera RA, Velazquez-Alva C, and Gonzalez-Barranco J. 2003. Short stature is related to high body fat composition despite body mass index in a Mexican population. *Archives of Medical Research* 34(2):137-140.
- Lozada M, Sanchez-Castillo CP, Cabrera GA, Mata, II, Pichardo-Ontiveros E, Villa AR, and James WP. 2008. School food in Mexican children. *Public Health Nutr* 11(9):924-933.
- Lumey LH, Stein AD, Kahn HS, and Romijn JA. 2009. Lipid profiles in middle-aged men and women after famine exposure during gestation: the Dutch Hunger Winter Families Study. *Am J Clin Nutr* 89(6):1737-1743.
- Lundberg M, Diderichsen F, and Hallqvist J. 2002. Is the association between short stature and myocardial infarction explained by childhood exposures--a population-based case referent study (SHEEP). *Scand J Public Health* 30(4):249-258.
- Lutter CK, Chaparro CM, and Munoz S. 2011. Progress towards Millennium Development Goal 1 in Latin America and the Caribbean: the importance of the choice of indicator for undernutrition. *Bull World Health Organ* 89(1):22-30.
- Malina RM, Pena Reyes ME, Chavez GB, and Little BB. 2011a. Secular change in height and weight of indigenous school children in Oaxaca, Mexico, between the 1970s and 2007. *Annals of Human Biology*.
- Malina RM, Pena Reyes ME, and Little BB. 2008a. Epidemiologic transition in an isolated indigenous community in the Valley of Oaxaca, Mexico. *Am J Phys Anthropol* 137(1):69-81.
- Malina RM, Pena Reyes ME, Tan SK, and Little BB. 2011b. Physical fitness of normal, stunted and overweight children 6-13 years in Oaxaca, Mexico. *European Journal of Clinical Nutrition* 65(7):826-834.
- Malina RM, Reyes ME, Tan SK, and Little BB. 2008b. Physical activity in youth from a subsistence agriculture community in the Valley of Oaxaca, southern Mexico. *Appl Physiol Nutr Metab* 33(4):819-830.
- Maluccio JA, Hoddinott J, Behrman JR, Martorell R, Quisumbing AR, and Stein AD. 2009. The Impact of Improving Nutrition during Early Childhood on Education among Guatemalan Adults. *Economic Journal* 119(537):734-763.
- Mao X, Bigham AW, Mei R, Gutierrez G, Weiss KM, Brutsaert TD, Leon-Velarde F, Moore LG, Vargas E, McKeigue PM and others. 2007. A genomewide admixture mapping panel for Hispanic/Latino populations. *Am J Hum Genet* 80(6):1171-1178.

- Mardones F, Villarroel L, Karzulovic L, Barja S, Arnaiz P, Taibo M, and Mardones-Restat F. 2008. Association of perinatal factors and obesity in 6-to 8-year-old Chilean children. *International Journal of Epidemiology* 37(4):902-910.
- Martin-Gronert MS, and Ozanne SE. 2006. Maternal nutrition during pregnancy and health of the offspring. *Biochemical Society Transactions* 34:779-782.
- Martin RM, Gunnell D, Pemberton J, Frankel S, and Davey Smith G. 2005. Cohort profile: The Boyd Orr cohort--an historical cohort study based on the 65 year follow-up of the Carnegie Survey of Diet and Health (1937-39). *Int J Epidemiol* 34(4):742-749.
- Martins PA, Hoffman DJ, Fernandes MTB, Nascimento CR, Roberts SB, Sesso R, and Sawaya AL. 2004. Stunted children gain less lean body mass and more fat mass than their non-stunted counterparts: a prospective study. *British Journal of Nutrition* 92(5):819-825.
- Martorell R. 1995. Results and implications of the INCAP follow-up study. *J Nutr* 125(4 Suppl):1127S-1138S.
- Martorell R, Habicht JP, and Rivera JA. 1995. History and Design of the Incap Longitudinal-Study (1969-77) and Its Follow-up (1988-89). *Journal of Nutrition* 125(4):S1027-S1041.
- Martorell R, Khan LK, Hughes ML, and Grummer-Strawn LM. 1998. Obesity in Latin American women and children. *Journal of Nutrition* 128(9):1464-1473.
- Matsuzawa Y. 2008. The role of fat topology in the risk of disease. *Int J Obes (Lond)* 32 Suppl 7:S83-92.
- McCarthy HD, Cole TJ, Fry T, Jebb SA, and Prentice AM. 2006. Body fat reference curves for children. *Int J Obes (Lond)* 30(4):598-602.
- McClain JJ, and Tudor-Locke C. 2009. Objective monitoring of physical activity in children: considerations for instrument selection. *J Sci Med Sport* 12(5):526-533.
- McCrary J, and Royer H. 2011. The Effect of Female Education on Fertility and Infant Health: Evidence from School Entry Policies Using Exact Date of Birth. *American Economic Review* 101(1):158-195.
- McDade TW, Leonard WR, Burhop J, Reyes-Garcia V, Vadez V, Huanca T, and Godoy RA. 2005. Predictors of C-reactive protein in Tsimane' 2 to 15 year-olds in lowland Bolivia. *Am J Phys Anthropol* 128(4):906-913.
- MeasureDHS. 2011. Measure DHS. Calverton, MD: Measure DHS.
- Mendez MA, Monteiro CA, and Popkin BM. 2005. Overweight exceeds underweight among women in most developing countries. *Am J Clin Nutr* 81(3):714-721.
- Menezes AM, Perez-Padilla R, Jardim JR, Muino A, Lopez MV, Valdivia G, Montes de Oca M, Talamo C, Hallal PC, and Victora CG. 2005. Chronic obstructive pulmonary disease in five Latin American cities (the PLATINO study): a prevalence study. *Lancet* 366(9500):1875-1881.
- Monda KL, Adair LS, Zhai F, and Popkin BM. 2008. Longitudinal relationships between occupational and domestic physical activity patterns and body weight in China. *Eur J Clin Nutr* 62(11):1318-1325.
- Monda KL, Gordon-Larsen P, Stevens J, and Popkin BM. 2007. China's transition: The effect of rapid urbanization on adult occupational physical activity. *Social Science & Medicine* 64(4):858-870.

- Monden CWS, and Smits J. 2009. Maternal Height and Child Mortality in 42 Developing Countries. *American Journal of Human Biology* 21(3):305-311.
- Monteiro C, Conde WL, Lu B, and Popkin BM. 2004. Obesity and inequities in health in the developing world. *International Journal of Obesity* 28(9):1181-1186.
- Monteiro CA, Conde WL, and Popkin BM. 2002. Is obesity replacing or adding to undernutrition? Evidence from different social classes in Brazil. *Public Health Nutr* 5(1A):105-112.
- Mukuddem-Petersen J, and Kruger HS. 2004. Association between stunting and overweight among 10-15-y-old children in the North West Province of South Africa: the THUSA BANA Study. *Int J Obes Relat Metab Disord* 28(7):842-851.
- Ness AR, Maynard M, Frankel S, Smith GD, Frobisher C, Leary SD, Emmett PM, and Gunnell D. 2005. Diet in childhood and adult cardiovascular and all cause mortality: the Boyd Orr cohort. *Heart* 91(7):894-898.
- Neufeld LM, Hernandez-Cordero S, Fernald LC, and Ramakrishnan U. 2008. Overweight and obesity doubled over a 6-year period in young women living in poverty in Mexico. *Obesity* 16(3):714-717.
- Ng SW, Norton EC, and Popkin BM. 2009. Why have physical activity levels declined among Chinese adults? Findings from the 1991-2006 China health and nutrition surveys. *Social Science & Medicine* 68(7):1305-1314.
- Norgan NG. 1994a. Population differences in body composition in relation to the body mass index. *European Journal of Clinical Nutrition* 48 Suppl 3:S10-25; discussion S26-17.
- Norgan NG. 1994b. Relative sitting height and the interpretation of the body mass index. *Annals of Human Biology* 21(1):79-82.
- Norgan NG, and Jones PR. 1995. The effect of standardising the body mass index for relative sitting height. *Int J Obes Relat Metab Disord* 19(3):206-208.
- Novotny R, Daida Y, Morimoto Y, Shepherd J, and Maskarinec G. 2011. Puberty, body fat, and breast density in girls of several ethnic groups. *American Journal of Human Biology* 23(3):359-365.
- Ogden CL, Carroll MD, Curtin LR, Lamb MM, and Flegal KM. 2010. Prevalence of high body mass index in US children and adolescents, 2007-2008. *Jama* 303(3):242-249.
- Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, and Flegal KM. 2006. Prevalence of overweight and obesity in the United States, 1999-2004. *Jama* 295(13):1549-1555.
- Olaiz G, Rivera J, Shamah T, Rojas R, Villapando S, Hernandez M, and Sepulveda J. 2006. Encuesta Nacional de Salud y Nutricion 2006. Cuernavaca, Mexico: Instituto Nacional de Salud Publica.
- Onywera VO. 2010. Childhood obesity and physical inactivity threat in Africa: strategies for a healthy future. *Glob Health Promot* 17(2 Suppl):45-46.
- Ostrove JM, and Feldman P. 1999. Education, income, wealth, and health among whites and African Americans. *Ann N Y Acad Sci* 896:335-337.
- Oyhenart EE, Castro LE, Forte LM, Sicre ML, Quintero FA, Luis MA, Torres MF, Luna ME, Cesani MF, and Orden AB. 2008. Socioenvironmental conditions and nutritional status in urban and rural schoolchildren. *Am J Hum Biol* 20(4):399-405.

- Ozaltin E, Hill K, and Subramanian SV. 2010. Association of Maternal Stature With Offspring Mortality, Underweight, and Stunting in Low- to Middle-Income Countries. *Jama-Journal of the American Medical Association* 303(15):1507-1516.
- Paajanen TA, Oksala NK, Kuukasjarvi P, and Karhunen PJ. 2010. Short stature is associated with coronary heart disease: a systematic review of the literature and a meta-analysis. *Eur Heart J* 31(14):1802-1809.
- Paeratakul S, Lovejoy JC, Ryan DH, and Bray GA. 2002. The relation of gender, race and socioeconomic status to obesity and obesity comorbidities in a sample of US adults. *International Journal of Obesity* 26(9):1205-1210.
- Panther-Brick C, Lunn PG, Langford RM, Maharjan M, and Manandhar DS. 2009. Pathways leading to early growth faltering: an investigation into the importance of mucosal damage and immunostimulation in different socio-economic groups in Nepal. *British Journal of Nutrition* 101(4):558-567.
- Parra DC, Lobelo F, Gomez LF, Rutt C, Schmid T, Brownson RC, and Pratt M. 2009. Household motor vehicle use and weight status among Colombian adults: Are we driving our way towards obesity? *Preventive Medicine* 49(2-3):179-183.
- Parsons TJ, Power C, and Manor O. 2001. Fetal and early life growth and body mass index from birth to early adulthood in 1958 British cohort: longitudinal study. *Bmj* 323(7325):1331-1335.
- Poirier P, Giles TD, Bray GA, Hong YL, Stern JS, Pi-Sunyer FX, and Eckel RH. 2006. Obesity and cardiovascular disease - Pathophysiology, evaluation, and effect of weight loss. *Arteriosclerosis Thrombosis and Vascular Biology* 26(5):968-976.
- Pollitt E, Gorman KS, Engle PL, Rivera JA, and Martorell R. 1995. Nutrition in early life and the fulfillment of intellectual potential. *J Nutr* 125(4 Suppl):1111S-1118S.
- Pollitt RA, Rose KM, and Kaufman JS. 2005. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health* 5:7.
- Popkin B, and Gordon-Larsen P. 2004. The nutrition transition: worldwide obesity dynamics and their determinants. *International Journal of Obesity* 28:S2-S9.
- Popkin BM. 1993. Nutritional Patterns and Transitions. *Population and Development Review* 19(1):138-157.
- Popkin BM. 1996. Understanding the nutrition transition. *Urban Health News* (30):3-19.
- Popkin BM. 1999. Urbanization, lifestyle changes and the nutrition transition. *World Development* 27(11):1905-1916.
- Popkin BM. 2002. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr* 5(1A):205-214.
- Popkin BM, Adair LS, and Ng SW. 2012. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev* 70(1):3-21.
- Popkin BM, and Doak CM. 1998. The obesity epidemic is a worldwide phenomenon. *Nutr Rev* 56(4 Pt 1):106-114.
- Popkin BM, Keyou G, Zhai F, Guo X, Ma H, and Zohoori N. 1993. The nutrition transition in China: a cross-sectional analysis. *European Journal of Clinical Nutrition* 47(5):333-346.

- Popkin BM, Lu B, and Zhai F. 2002. Understanding the nutrition transition: measuring rapid dietary changes in transitional countries. *Public Health Nutr* 5(6A):947-953.
- Popkin BM, and Udry JR. 1998. Adolescent obesity increases significantly in second and third generation U.S. immigrants: the National Longitudinal Study of Adolescent Health. *J Nutr* 128(4):701-706.
- Post CL, and Victora CG. 2001. The low prevalence of weight-for-height deficits in Brazilian children is related to body proportions. *Journal of Nutrition* 131(4):1290-1296.
- Prentice AM. 2006. The emerging epidemic of obesity in developing countries. *International Journal of Epidemiology* 35(1):93-99.
- Prentice AM, and Paul AA. 2000. Fat and energy needs of children in developing countries. *Am J Clin Nutr* 72(5 Suppl):1253S-1265S.
- Price KC, and Coe CL. 2000. Maternal constraint on fetal growth patterns in the rhesus monkey (*Macaca mulatta*): the intergenerational link between mothers and daughters. *Human Reproduction* 15(2):452-457.
- Price KC, Hyde JS, and Coe CL. 1999. Matrilineal transmission of birth weight in the rhesus monkey (*Macaca mulatta*) across several generations. *Obstetrics and Gynecology* 94(1):128-134.
- Raitakari OT, Porkka KVK, Taimela S, Telama R, Rasanen L, and Viikari JSA. 1994. Effects of Persistent Physical-Activity and Inactivity on Coronary Risk-Factors in Children and Young-Adults - the Cardiovascular Risk in Young Finns Study. *American Journal of Epidemiology* 140(3):195-205.
- Raj M, Sundaram KR, Paul M, Sudhakar A, and Kumar RK. 2009. Dynamics of growth and weight transitions in a pediatric cohort from India. *Nutrition Journal* 8:-.
- Rankinen T, Kim SY, Perusse L, Despres JP, and Bouchard C. 1999. The prediction of abdominal visceral fat level from body composition and anthropometry: ROC analysis. *Int J Obes Relat Metab Disord* 23(8):801-809.
- Rasanathan K, and Krech R. 2011. Action on social determinants of health is essential to tackle noncommunicable diseases. *Bulletin of the World Health Organization* 89:775-776.
- Rehman AM, Gladstone BP, Verghese VP, Muliylil J, Jaffar S, and Kang G. 2009. Chronic growth faltering amongst a birth cohort of Indian children begins prior to weaning and is highly prevalent at three years of age. *Nutrition Journal* 8:44.
- Reilly JJ, and Kelly J. 2011. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes (Lond)* 35(7):891-898.
- Reyes ME, Chavez GB, Little BB, and Malina RM. 2010. Community well-being and growth status of indigenous school children in rural Oaxaca, southern Mexico. *Econ Hum Biol* 8(2):177-187.
- Reza-Lopez SA, Anderson GH, Szeto IM, Taha AY, and Ma DW. 2009. High vitamin intake by Wistar rats during pregnancy alters tissue fatty acid concentration in the offspring fed an obesogenic diet. *Metabolism* 58(5):722-730.

- Ristow M, Muller-Wieland D, Pfeiffer A, Krone W, and Kahn CR. 1998. Obesity associated with a mutation in a genetic regulator of adipocyte differentiation. *New England Journal of Medicine* 339(14):953-959.
- Rivera JA, Barquera S, Campirano F, Campos I, Safdie M, and Tovar V. 2002. Epidemiological and nutritional transition in Mexico: rapid increase of non-communicable chronic diseases and obesity. *Public Health Nutr* 5(1A):113-122.
- Rivera JA, Barquera S, Gonzalez-Cossio T, Olaiz G, and Sepulveda J. 2004. Nutrition transition in Mexico and in other Latin American countries. *Nutr Rev* 62(7 Pt 2):S149-157.
- Rivera JA, Martorell R, Ruel MT, Habicht JP, and Haas JD. 1995. Nutritional supplementation during the preschool years influences body size and composition of Guatemalan adolescents. *J Nutr* 125(4 Suppl):1068S-1077S.
- Rowlands AV, and Eston RG. 2007. The measurement and interpretation of children's physical activity. *Journal of Sports Science and Medicine* 6(3):270-276.
- Russell M. 1976. Relationship of Family-Size and Spacing to Growth of Preschool Mayan Children in Guatemala. *American Journal of Public Health* 66(12):1165-1172.
- Rutishau L, and Whitehead R. 1972. Energy Intake and Expenditure in 1-3-Year-Old Ugandan Children Living in a Rural Environment. *British Journal of Nutrition* 28(1):145-&.
- Salazar-Martinez E, Allen B, Fernandez-Ortega C, Torres-Mejia G, Galal O, and Lazcano-Ponce E. 2006. Overweight and obesity status among adolescents from Mexico and Egypt. *Arch Med Res* 37(4):535-542.
- Salzano FM. 2002. Molecular variability in Amerindians: widespread but uneven information. *Anais da Academia Brasileira de Ciencias* 74(2):223- 263.
- Santaella JA. 1998. Economic growth in Mexico. *Inter-American Development Bank*.
- Satoh H, Kishi R, and Tsutsui H. 2010. Body mass index can similarly predict the presence of multiple cardiovascular risk factors in middle-aged Japanese subjects as waist circumference. *Intern Med* 49(11):977-982.
- Sauri Bazán MC. 2003. Publicidad televisiva, hábitos alimentarios y salud en adolescentes de la ciudad de Mérida Mérida, México: Centro de Investigación y de Estudios Avanzados del I. P. N.
- Sawaya AL, Martins P, Hoffman D, and Roberts SB. 2003. The link between childhood undernutrition and risk of chronic diseases in adulthood: A case study of Brazil. *Nutrition Reviews* 61(5):168-175.
- Schmolck H, Buffalo EA, and Squire LR. 2000a. Memory distortions develop over time: Recollections of teh O.J. Simpson trial verdict after 15 and 32 months. *Psychological Science* 11(1):39-45.
- Schmolck H, Buffalo EA, and Squire LR. 2000b. Memory distortions develop over time: recollections of the O.J. Simpson trial verdict after 15 and 32 months. *Psychol Sci* 11(1):39-45.
- Schofield WN. 1985. Predicting basal metabolic rate, new standards and review of previous work. *Hum Nutr Clin Nutr* 39 Suppl 1:5-41.
- Schooling CM, Jiang CQ, Heys M, Zhang WS, Adab P, Cheng KK, Lam TH, and Leung GM. 2008a. Are height and leg length universal markers of childhood

- conditions? The Guangzhou Biobank cohort study. *Journal of Epidemiology and Community Health* 62(7):607-614.
- Schooling CM, Jiang CQ, Heys M, Zhang WS, Lao XQ, Adab P, Cowling BJ, Thomas GN, Cheng KK, Lam TH and others. 2008b. Is leg length a biomarker of childhood conditions in older Chinese women? The Guangzhou Biobank Cohort Study. *J Epidemiol Community Health* 62(2):160-166.
- Schroeder DG, Martorell R, and Flores R. 1999. Infant and child growth and fatness and fat distribution in Guatemalan adults. *Am J Epidemiol* 149(2):177-185.
- Seldin MF, Tian C, Shigeta R, Scherbarth HR, Silva G, Belmont JW, Kittles R, Gamron S, Allevi A, Palatnik SA and others. 2007. Argentine population genetic structure: large variance in Amerindian contribution. *Am J Phys Anthropol* 132(3):455-462.
- Serdula MK, Ivery DC, R.J., Freedman DS, Williamson DF, and Byers T. 1993. Do obese children become obese adults? A review of the literature. *Preventive Medicine* 22:167-177.
- Sharrock KC, Kuzawa CW, Leonard WR, Tanner S, Reyes-Garcia VE, Vadez V, Huanca T, and McDade TW. 2008. Developmental changes in the relationship between leptin and adiposity among Tsimane children and adolescents. *Am J Hum Biol* 20(4):392-398.
- Shea JL, King MT, Yi Y, Gulliver W, and Sun G. 2011. Body fat percentage is associated with cardiometabolic dysregulation in BMI-defined normal weight subjects. *Nutr Metab Cardiovasc Dis*.
- Sheehy T, and Sharma S. 2010. The nutrition transition in Barbados: trends in macronutrient supply from 1961 to 2003. *British Journal of Nutrition* 104(8):1222-1229.
- Siegel SR, Malina RM, Reyes ME, Barahona EE, and Cumming SP. 2011. Correlates of physical activity and inactivity in urban Mexican youth. *American Journal of Human Biology* 23(5):686-692.
- Siervo M, Horta BL, Stephan BC, Victora CG, and Wells JC. 2010. First-borns carry a higher metabolic risk in early adulthood: evidence from a prospective cohort study. *PLoS One* 5(11):e13907.
- Silventoinen K, Sans S, Tolonen H, Monterde D, Kuulasmaa K, Kesteloot H, and Tuomilehto J. 2004. Trends in obesity and energy supply in the WHO MONICA Project. *Int J Obes Relat Metab Disord* 28(5):710-718.
- Singh-Manoux A, Adler NE, and Marmot MG. 2003. Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. *Soc Sci Med* 56(6):1321-1333.
- Singh-Manoux A, Marmot MG, and Adler NE. 2005. Does subjective social status predict health and change in health status better than objective status? *Psychosom Med* 67(6):855-861.
- Singha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K, Savoye M, Rieger V, Taksali S, Barbetta G and others. 2004. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *N Engl J Med* 346(11):802-809.
- Smith LC, Ruel MT, and Ndiaye A. 2005. Why is child malnutrition lower in urban than in rural areas? Evidence from 36 developing countries. *World Development* 33(8):1285-1305.

- Smith PK, Bogin B, Varela-Silva MI, and Loucky J. 2003. Economic and anthropological assessments of the health of children in Maya immigrant families in the US. *Econ Hum Biol* 1(2):145-160.
- Soares-Wynter SY, and Walker SP. 1996. Resting metabolic rate and body composition in stunted and nonstunted children. *American Journal of Clinical Nutrition* 64(2):137-141.
- Sodjinou R, Agueh V, Fayomi B, and Delisle H. 2009. Dietary patterns of urban adults in Benin: relationship with overall diet quality and socio-demographic characteristics. *Eur J Clin Nutr* 63(2):222-228.
- Song YM, and Song J. 2008. Adult height and the risk of mortality in South Korean women. *American Journal of Epidemiology* 168(5):497-505.
- Song YM, and Sung J. 2008. Adult height and the risk of mortality in South Korean women. *Am J Epidemiol* 168(5):497-505.
- Soobader M, LeClere FB, Hadden W, and Maury B. 2001. Using aggregate geographic data to proxy individual socioeconomic status: does size matter? *American Journal of Public Health* 91(4):632-636.
- Sparks CS. 2011. Parental investment and socioeconomic status influences on children's height in Honduras: An analysis of national data. *American Journal of Human Biology* 23(1):80-88.
- Spurr GB, and Reina JC. 1988. Patterns of daily energy expenditure in normal and marginally undernourished school-aged Colombian children. *European Journal of Clinical Nutrition* 42(10):819-834.
- Spurr GB, Reina JC, and Barac-Nieto M. 1986. Marginal malnutrition in school-aged Colombian boys: metabolic rate and estimated daily energy expenditure. *American Journal of Clinical Nutrition* 44(1):113-126.
- Steckel RH. 1995. Stature and the standard of living. *Journal of Economic Literature* 33(4):1903-1940.
- Steckel RH. 2009. Heights and human welfare: Recent developments and new directions. *Explorations in Economic History* 46(1):1-23.
- Stein AD, Wang M, Martorell R, Norris SA, Adair LS, Bas I, Sachdev HS, Bhargava SK, Fall CH, Gigante DP and others. 2010. Growth patterns in early childhood and final attained stature: data from five birth cohorts from low- and middle-income countries. *American Journal of Human Biology* 22(3):353-359.
- Stevens G, Dias RH, Thomas KJ, Rivera JA, Carvalho N, Barquera S, Hill K, and Ezzati M. 2008. Characterizing the epidemiological transition in Mexico: national and subnational burden of diseases, injuries, and risk factors. *PLoS Med* 5(6):e125.
- Stolarczyk LM, Heyward VH, Hicks VL, and Baumgartner RN. 1994. Predictive accuracy of bioelectrical impedance in estimating body composition of Native American women. *Am J Clin Nutr* 59(5):964-970.
- Strath SJ, Bassett DR, Jr., Swartz AM, and Thompson DL. 2001. Simultaneous heart rate-motion sensor technique to estimate energy expenditure. *Med Sci Sports Exerc* 33(12):2118-2123.
- Szeto IM, Das PJ, Aziz A, and Anderson GH. 2009. Multivitamin supplementation of Wistar rats during pregnancy accelerates the development of obesity in offspring fed an obesogenic diet. *Int J Obes (Lond)* 33(3):364-372.

- Tanner JM. 1981. A history of the study of human growth. New York: Cambridge University Press. 499 p.
- Tanner JM. 1986. Growth as a mirror of the conditions of society: secular trends and class distinctions. In: Demirjian A, editor. Human Growth: A multidisciplinary review. London: Taylor & Francis. p 3- 34.
- Tanner JM. 1990. Foetus into man: Physical growth from conception to maturity. Cambridge, Massachusetts: Harvard University Press.
- Tanner JM, Hayashi T, Preece MA, and Cameron N. 1982. Increase in length of leg relative to trunk in Japanese children and adults from 1957 to 1977: comparison with British and with Japanese Americans. *Ann Hum Biol* 9(5):411-423.
- Terry MB, Flom J, Tehranifar P, and Susser E. 2009. The role of birth cohorts in studies of adult health: the New York women's birth cohort. *Paediatr Perinat Epidemiol* 23(5):431-445.
- Torun B, Stein AD, Schroeder D, Grajeda R, Conlisk A, Rodriguez M, Mendez H, and Martorell R. 2002. Rural-to-urban migration and cardiovascular disease risk factors in young Guatemalan adults. *International Journal of Epidemiology* 31(1):218-226.
- Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, and McDowell M. 2008. Physical activity in the United States measured by accelerometer. *Med Sci Sports Exerc* 40(1):181-188.
- Turgeon JL, Carr MC, Maki PM, Mendelsohn ME, and Wise PM. 2006. Complex actions of sex steroids in adipose tissue, the cardiovascular system, and brain: insights from basic science and clinical studies. *Endocrine Reviews* 27(6):575-605.
- Uauy R, Albala C, and Kain J. 2001. Obesity trends in Latin America: transiting from under- to overweight. *J Nutr* 131(3):893S-899S.
- UNICEF. 2010. At a glance: Mexico.
http://www.unicef.org/infobycountry/mexico_statisticshtml.
- UNICEF. 2011. Millennium Development Goals. New York City, NY, USA.
- UNICEF, and WHO. 2009. Diarrhoea: Why children are still dying and what can be done.
- UNPF. 2007. Linking population, poverty and development. New York City: United Nations Population Fund.
- Unwin N, McLarty D, Machibya H, Aspray T, Tamin B, Carlin L, Patel S, Walker M, and Alberti KG. 2006. Changes in blood pressure and lipids associated with rural to urban migration in Tanzania. *J Hum Hypertens* 20(9):704-706.
- Van de Poel E, Hosseinpoor AR, Speybroeck N, Van Ourti T, and Vega J. 2008. Socioeconomic inequality in malnutrition in developing countries. *Bull World Health Organ* 86(4):282-291.
- Van de Poel E, O'Donnell O, and Van Doorslaer E. 2007. Are urban children really healthier? Evidence from 47 developing countries. *Social Science & Medicine* 65(10):1986-2003.
- Van de Poel E, O'Donnell O, and Van Doorslaer E. 2009. Urbanization and the spread of diseases of affluence in China. *Econ Hum Biol* 7(2):200-216.
- Varela-Silva MI, Azcorra H, Dickinson F, Bogin B, and Frisancho AR. 2009. Influence of maternal stature, pregnancy age, and infant birth weight on growth during

- childhood in Yucatan, Mexico: a test of the intergenerational effects hypothesis. *Am J Hum Biol* 21(5):657-663.
- Varela-Silva MI, Frisancho AR, Bogin B, Chatkoff D, Smith PK, Dickinson F, and Winham D. 2007. Behavioral, environmental, metabolic and intergenerational components of early life undernutrition leading to later obesity in developing nations and in minority groups in the U.S.A. *Coll Antropol* 31(1):39-46.
- Varela Silva MI, Dickinson F, Wilson H, Azcorra H, Griffiths P, and Bogin B. 2011. The nutritional dual-burden in developing countries. How is it assessed and what are the health implications? *Coll Antropol* in press.
- Varela Silva MI, Dickinson F, Wilson H, Azcorra H, Griffiths P, and Bogin B. 2012. The nutritional dual-burden in developing countries. How is it assessed and what are the health implications? *Collegium Anthropologicum* in press.
- Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, and Sachdev HS. 2008. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* 371(9609):340-357.
- Wadsworth MEJ, Hardy RJ, Paul AA, Marshall SF, and Cole TJ. 2002. Leg and trunk length at 43 years in relation to childhood health, diet and family circumstances; evidence from the 1946 national birth cohort. *International Journal of Epidemiology* 31(2):383-390.
- Walker AR, Adam F, and Walker BF. 2001. World pandemic of obesity: the situation in Southern African populations. *Public Health* 115(6):368-372.
- Walker SP, Chang SM, and Powell CA. 2007. The association between early childhood stunting and weight status in late adolescence. *Int J Obes (Lond)* 31(2):347-352.
- Walker SP, Powell CA, Grantham-McGregor SM, Himes JH, and Chang SM. 1991. Nutritional supplementation, psychosocial stimulation, and growth of stunted children: the Jamaican study. *Am J Clin Nutr* 54(4):642-648.
- Walley J, Wright J, and Hubley J. 2005. *Public Health: an action guide to improving health in developing countries*. Oxford: Oxford University Press. 290 p.
- Wang LY, Chyen D, Lee S, and Lowry R. 2008. The association between body mass index in adolescence and obesity in adulthood. *J Adolesc Health* 42(5):512-518.
- Wang Y, Chen X, Klag MJ, and Caballero B. 2006. Epidemic of childhood obesity: implications for kidney disease. *Adv Chronic Kidney Dis* 13(4):336-351.
- Wang YF, and Zhang Q. 2006. Are American children and adolescents of low socioeconomic status at increased risk of obesity? Changes in the association between overweight and family income between 1971 and 2002. *American Journal of Clinical Nutrition* 84(4):707-716.
- Ward DS, Evenson KR, Vaughn A, Rodgers AB, and Troiano RP. 2005. Accelerometer use in physical activity: best practices and research recommendations. *Med Sci Sports Exerc* 37(11 Suppl):S582-588.
- Webb EA, Kuh D, Pajak A, Kubinova R, Malyutina S, and Bobak M. 2008. Estimation of secular trends in adult height, and childhood socioeconomic circumstances in three Eastern European populations. *Econ Hum Biol* 6(2):228-236.
- Weinsier RL, Schutz Y, and Bracco D. 1992. Reexamination of the relationship of resting metabolic rate to fat-free mass and to the metabolically active

- components of fat-free mass in humans. *American Journal of Clinical Nutrition* 55(4):790-794.
- Wells JC. 2010. Maternal capital and the metabolic ghetto: An evolutionary perspective on the transgenerational basis of health inequalities. *American Journal of Human Biology* 22(1):1-17.
- Wells JC, Griffin L, and Treleaven P. 2010. Independent changes in female body shape with parity and age: A life-history approach to female adiposity. *American Journal of Human Biology* 22(4):456-462.
- Westerterp KR. 2009a. Assessment of physical activity: a critical appraisal. *Eur J Appl Physiol* 105(6):823-828.
- Westerterp KR. 2009b. Physical activity assessment with accelerometers in children. *Indian Pediatr* 46(12):1053-1054.
- Weyer CW, Pratley RE, Lindsay RS, and Tataranni PA. 2000. Relationship between birth weight and body composition, energy metabolism, and sympathetic nervous system activity later in life. *Obesity Research* 8:559- 565.
- Whitehead RG, and Paul AA. 2000. Growth patterns of breastfed infants. *Acta Paediatr* 89(2):136-138.
- Whitmer RA, Gunderson EP, Barrett-Connor E, Quesenberry CP, and Yaffe K. 2005. Obesity in middle age and future risk of dementia: a 27 year longitudinal population based study. *British Medical Journal* 330(7504):1360-1362B.
- WHO. 2006. Obesity and overweight. Geneva: World Health Organization. 1- 3 p.
- WHO. 2009. Obesity. In: Organization WH, editor.
- Wilkins LR, Hankin JH, Yoshizawa CN, Kolonel LN, and Lee J. 1992. Comparison of long-term dietary recall between cancer cases and noncases. *Am J Epidemiol* 136(7):825-835.
- Willett W. 1998. Nutritional epidemiology. New York: Oxford University Press. 514 p.
- Willett WC, Sampson L, Browne ML, Stampfer MJ, Rosner B, Hennekens CH, and Speizer FE. 1988. The use of a self-administered questionnaire to assess diet four years in the past. *Am J Epidemiol* 127(1):188-199.
- Wilson H, Dickinson F, Griffiths P, Bogin B, and Varela-Silva MI. 2011. Logistics of using the Actiheart physical activity monitors in urban Mexico among 7- to 9-year-old children. *American Journal of Human Biology* 23(3):426-428.
- Wolf AM, and Colditz GA. 1998. Current estimates of the economic cost of obesity in the United States. *Obes Rev* 6(2):97-106.
- WorldBank. 2010. <http://data.worldbank.org/country/mexico>. The World Bank.
- Wren RE, Blume H, Mazariegos M, Solomons N, Alvarez JO, and Goran MI. 1997. Body composition, resting metabolic rate, and energy requirements of short- and normal-stature, low-income Guatemalan children. *American Journal of Clinical Nutrition* 66(2):406-412.
- Yach D, Hawkes C, Gould CL, and Hofman KJ. 2004. The global burden of chronic diseases: overcoming impediments to prevention and control. *Jama* 291(21):2616-2622.
- Yach D, Stuckler D, and Brownell KD. 2006. Epidemiologic and economic consequences of the global epidemics of obesity and diabetes. *Nature Medicine* 12(1):62-66.

- Yadav K, and Krishnan A. 2008. Changing patterns of diet, physical activity and obesity among urban, rural and slum populations in north India. *Obes Rev* 9(5):400-408.
- Yamamoto-Kimura L, Posadas-Romero C, Posadas-Sanchez R, Zamora-Gonzalez J, Cardoso-Saldana G, and Mendez Ramirez I. 2006. Prevalence and interrelations of cardiovascular risk factors in urban and rural Mexican adolescents. *J Adolesc Health* 38(5):591-598.
- Yu ZB, Han SP, Zhu GZ, Zhu C, Wang XJ, Cao XG, and Guo XR. 2011. Birth weight and subsequent risk of obesity: a systematic review and meta-analysis. *Obes Rev* 12(7):525-542.
- Zhai FY, Wang HJ, Du SF, He YN, Wang ZH, Ge KY, and Popkin BM. 2009. Prospective study on nutrition transition in China. *Nutrition Reviews* 67(5):S56-S61.
- Ziol-Guest KM, Duncan GJ, and Kalil A. 2009. Early childhood poverty and adult body mass index. *Am J Public Health* 99(3):527-532.
- Zottarelli LK, Sunil TS, and Rajaram S. 2007. Influence of parental and socioeconomic factors on stunting in children under 5 years in Egypt. *East Mediterr Health J* 13(6):1330-1342.

Chapter 10. Appendices

A. Participant information sheets

1. Loughborough ethics committee



Childhood growth status in urban Mayans

Mother's Participant Information Sheet

***Translation into Spanish will occur prior to fieldwork**

Hannah Wilson
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School of Sport, Exercise
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Loughborough University
Loughborough
Leicestershire
LE11 3TU
United Kingdom

Dr Federico Dickinson Bannack
Email: dickinso@mda.cinvestav.mx
Tel. Number: +(999) 942 94 00
CINVESTAV
Dept. Ecologia Humana
Unidad Mérida
Carretera Antigua a Progreso
Cordemex, 97310
Mérida

What is the purpose of the study?

This study will help figure out if a mother's childhood diet and living conditions is related to her child's health.

Who can take part?

Any Maya woman with a child between the ages of 7 to 9 years old.

Once I take part, can I change my mind?

Of course! You can leave the study at any time, even after you have left the session. You may also refuse to answer any question or have any measurement taken on you or your child. You will not be asked why you changed your mind. Your child may also leave the study, regardless of if you continue to participate. If you want to leave the study, please just tell Hannah Wilson, Frederico Dickinson Bannack or any other member of the research team.

What will I be asked to do?

You will be asked to fill out questionnaires about your childhood and you and your child's life. If you are unclear on anything we ask, please ask us to explain. We will also want to measure your height, weight, height while seated, length of your lower leg, the size of your waist, the size of your upper arm and your grip strength. We will also want to measure the amount of fat that lies just under the skin on the back of the upper arm. This will be done by gently squeezing the skin and measuring how thick the squeezed area is. These measures will be done three times each by a woman researcher in private. This session will last about 2 hours.

What will my child be asked to do?

We will ask to measure many of the same things on your child as we did you, with you present. We will want to measure your child's height, weight, height while seated, lower leg length and size of the hips and waist. Also we will want to measure your child's amount of fat that lies just under the skin by gently squeezing the skin and measuring how thick the squeezed area is. We will measure your child's upper arms, stomach, upper leg, lower leg and back. All of these measurements will be taken three times each with you present. If you or your child wishes for a man to be present, please just let us know.

We also ask your child to wear a device which measures how much activity is done over a 5 day period. This device is a wire that connects to your child's chest in two places. The wire is connected to your child's chest by sticky pads. Your child will rub his chest with paper to clean the skin for the pads. Then the researcher will stick the pads on the skin and attach the wire. While your child is wearing this device, your child will be able to do everything your child normally does. The activity monitors will be collected from you by a researcher on Monday morning.

What type of clothing should I wear?

Light clothes or whatever you feel comfortable in.

Are there any risks in participating?

Some people may feel uncomfortable about being measured. If you or your child feels uncomfortable please tell us and we will do our best to make you more comfortable.

Will my taking part in this study be kept confidential?

We will ask your date of birth. No other information that can personally identify you will be asked. Your information will be kept on a protected computer that can only be accessed by a password.

What do I get for participating?

We will provide you with an information sheet that explains what each measure means and compares your child to the United States averages for height and weight.

I have some more questions who should I contact?

Hannah Wilson Email: H.Wilson@lboro.ac.uk
Phone number: TBD

Maria Inês Varela-Silva Email: M.I.O.Varela-Silva@lboro.ac.uk
Phone number: TBD

Frederico Dickinson Bannack Email: dickinso@mda.cinvestav.mx
Telephone: +52999 942-94-06

If you are unhappy with the way we conducted your session please visit the link below:
[http://www.lboro.ac.uk/admin/committees/ethical/Whistleblowing\(2\).htm](http://www.lboro.ac.uk/admin/committees/ethical/Whistleblowing(2).htm).
This is the link for the group that makes sure we conduct ourselves properly.



**Influence of Mayan mother's childhood diet on her child's weight
Child's Participant Information Sheet**

Hannah Wilson
Email: H.Wilson@lboro.ac.uk
Tel. Number: TBD
Department of Human Sciences
Sciences
Loughborough University
University
Loughborough, Leicestershire
LE11 3TU
UK

Maria Inês Varela-Silva
Email: M.I.O.Varela-Silva@lboro.ac.uk
Tel. Number: TBD
Department of Human
Loughborough
Loughborough, Leicestershire
LE11 3TU
U.K.

What is the purpose of the study?

This study will help figure out if a mother's childhood diet and living conditions is related to her child's weight.

Who can take part?

Any Mayan between the ages of 7 to 9 years old and their mother.

Once I take part, can I change my mind?

Of course! You or your mother can leave the study at any time, even after you have given us your session. You will not be asked why you changed your mind. If you or your mother wants to leave the study, please just tell Hannah Wilson.

What will I be asked to do?

With your mother present we will want to measure your height, weight, height while seated, lower leg length and size of the hips and waist. Also we will want to measure the amount of fat that lies just under the skin by gently squeezing the skin and measuring how thick the squeezed area is. If you or your mother wishes for a man to be present, please just let us know.

Also we may ask you to wear an activity monitor for 5 days. The monitor goes around the waist and should be put on in the mornings and taken off at night and during baths

or showers. The activity monitors will be collected from you by a researcher on Monday morning.

What type of clothing should I wear?

Light clothes or whatever you feel comfortable in.

Are there any risks in participating?

Some people may feel uncomfortable about being measured. If you feel uncomfortable please tell a researcher or your mother and we will do our best to make you more comfortable.

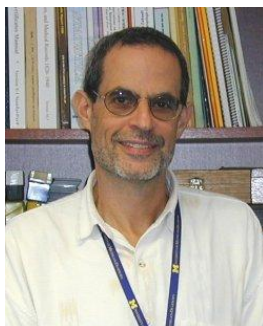
What do I get for participating?

We will provide you with an information sheet that explains what each measure means and compares you to the United States averages.

I have some more questions who should I contact?



Hannah Wilson
Silva
H.Wilson@lboro.ac.uk
Phone number: TBD



Dr. Barry Bogin
B.A.Bogin@lboro.ac.uk



Dr. Maria Inês Varela-Silva
M.I.O.Varela-Silva@lboro.ac.uk

If you are unhappy with the way we conducted your session please visit the link below:
[http://www.lboro.ac.uk/admin/committees/ethical/Whistleblowing\(2\).htm](http://www.lboro.ac.uk/admin/committees/ethical/Whistleblowing(2).htm).

This is the link for the group that makes sure we conduct ourselves properly.

2. Mexico

FORMA DE CONSENTIMIENTO

Estado de nutrición y de salud en una población maya en Yucatán, investigación

llevada al cabo por:

Dra. Maria Inês Varela Silva (Universidad de Loughborough, Reino Unido)

Dr. Federico Dickinson Bannack (CINVESTAV – Mérida, MEXICO)

Dr. Barry Bogin (Universidad de Loughborough, Reino Unido)

Estimada Señora:

Como Usted sabe, actualmente en México hay un aumento considerable de individuos de diversas edades con sobrepeso u obesidad, lo que significa un gran riesgo de desarrollar serias enfermedades del corazón y diabetes (azúcar en la sangre), entre otras. Por esta razón es conveniente realizar estudios sobre las causas del aumento de peso, especialmente en niños para eliminar o reducir los riesgos que corran de llegar a ser obesos.

El objetivo de esta investigación es estudiar varios aspectos de salud y nutrición de un grupo de madres y de sus hijos, para lo cual evaluaremos:

- El estado de crecimiento infantil: peso, talla, pliegues subcutáneos y circunferencias. Estas medidas son parecidas a las que se toman en los centros de salud. **Ninguna de estas pruebas es dolorosa ni molesta,**
- Ritmo cardíaco: se obtendrá un electrocardiograma para cada niño, para lo cual será necesario sujetar un electrodo en su piel. **No se aplicará corriente eléctrica a los niños, quienes no sentirán dolor alguno.**
- Modo de vida: se aplicará, a las madres, un cuestionario sobre el modo de vida de su familia y sus hábitos de nutrición.
- Actividad física: se pedirá a cada niño que durante cinco (5) días use un pequeño aparato, del tamaño de una grabadora pequeña, llamado acelerómetro. Este aparato, que medirá el movimiento del niño, estará sujeto a un cinturón que irá debajo de su ropa. **Esta prueba no es dolorosa.**
- Salud de las madres: se tomarán algunas mediciones, como peso y talla, y se les aplicará un cuestionario sobre sus embarazos y el cuidado de sus hijos pequeños.

Su participación en este estudio nos permitiría conocer el estado de nutrición de Usted y de su hija o hijo, lo que nos permitiría proporcionar a Usted información y orientación útiles para mejorar su salud y la de su familia y para que Usted pueda tomar mejores decisiones relacionadas con su salud y nutrición.

Además, su contribución contribuirá a que los resultados de esta investigación beneficien a otras personas de su comunidad y de otros lugares.

Para corresponder su colaboración, el proyecto hará a la escuela o al centro de salud de su comunidad un donativo de \$260 por cada mujer participante, para mejorar las condiciones educativas o de atención a la salud.

La información obtenida será utilizada con fines estadísticos, y se manejará de manera estrictamente confidencial; en ningún informe, publicación o presentación de los resultados

de esta investigación se usarán nombre ni otras características personales de los participantes. Cada participante recibirá un número de identificación que no estará ligado a su nombre. Para protección de la información que Usted nos proporcione, se seguirán las leyes mexicanas. El Comité de Bioética del Cinvestav, el equivalente de la Universidad de Loughborough u otras autoridades que supervisarán esta investigación podrán revisar los registros del estudio, para asegurar la protección de los participantes y la calidad de la información.

En caso de que en el transcurso de la investigación llegáramos a saber algo que pudiera afectar su disposición para que su hija o hijo y/o Usted sigan participando en ella, Usted sería informada de inmediato.

En caso de que Usted tuviese preguntas, dudas o alguna preocupación relacionadas con su participación en este estudio, Usted puede hacer contacto con cualquiera de las siguientes personas:

Dr. Federico Dickinson (dickinso@mda.cinvestav.mx), Departamento de Ecología Humana, Cinvestav–Mérida, Antigua carretera a Progreso km 6, 97310 Mérida, Yucatán (Tel. 942-94-06; Fax 981-46-70).

Su participación y la de su hija o hijo en esta investigación es voluntaria. Incluso después de haber firmado esta forma de consentimiento Usted puede dejar de participar en el estudio o rehusarse a contestar cualquier pregunta que le resulte incómoda, sin consecuencia alguna para Usted y sin perder los beneficios a los que tenga derecho.

Una copia de este documento se mantendrá en los registros del proyecto y, si Usted así lo desea, podrá obtener una copia para Usted.

He leído y comprendido la información anterior

_____ me ha ofrecido contestar cualquier pregunta que yo tenga en relación a este estudio, por lo que otorgo mi consentimiento para participar en él y para que lo haga mi hija(o)

_____.

Nombre (en letra de molde)

Firma de consentimiento

Tutor de la o el niño

Nombre (en letra de molde)

Firma de consentimiento

B. Interview

1. English

**Nutritional status and health in a dual burdened group of Yucatecan Maya
Departamento de Ecología Humana de Cinvestav-Mérida and the Department of
Human Sciences, University of Loughborough, England
Questionnaire for mothers and urban Maya children**

| | |
|--|---|
| Mother's birth date Day ____ Month ____ Year ____ | Child's birth date Day ____ Month ____ Year ____ |
|--|---|

1. How many brothers and sisters do you have? _____
2. How many older brothers and sisters do you have? _____
3. How many of your brothers and sisters lived to at least 5 years old? ¿ _____
4. Did you have any brothers or sisters die before they turned 5 years old? Yes ____ No ____
If so, how many? _____

CHILDHOOD OF THE MOTHERS

5. Your mother's job when you were a girl: _____
6. Your father's job when you were a girl: _____

7. FREQUENCY OF FOODS OF THE MOTHER DURING HER CHILDHOOD

| | |
|---|--|
| Bottled drinks (Coca Cola, Pepsi Cola or bottled juicea) | |
| Packaged foods (cookies, crisps, charritos, chicharrones de harina) | |
| Meat: Poultry, pork or beef | |

Frequency: 0: Never; 1: Daily; 2: Regularly; 3: Occassionally; 4: Only festivals (Christmas, New Year, Hanal Pixan, Reyes, or similar).

8. Did you ever go to bed hungry? Yes _____ No _____
(If yes, explain) _____
9. What was your favourite food when you were a girl? ¿ (Write what is mentioned)
 _____ Frequency: _____
 _____ Frequency: _____
 _____ Frequency: _____
10. What other types of food did you regularly eat when you were a girl?
 _____ Frequency: _____
 _____ Frequency: _____

_____ Frequency: _____

LIFE OF THE MOTHER DURING HER CHILDHOOD

11. What type of drinking water did you family drink when you were a girl?

House well _____

Public well _____

Purified _____

Piped water _____

Other _____

12. What did our household do to clean your drinking wáter when you were a girl?

Strained through a cloth _____

Chlorine and _____

Filtered _____

Boiled _____

Nothing _____

Other _____

13. When you were a girl ,did your household use:

Flush toilet _____

Latrine _____

Garden _____

Other _____

14. What materials was your house built out of?

Roof _____

Walls _____

Floor _____

15. What was the main source of light in your house when you were a girl?

Electricity___ Kerosene___ Gas___ Petrol___ Candles___ Other_____

16. When you were a girl, did you family have a milpa? ¿ Yes___ NO___ (if NO go to question 19)

17. How many mecates was your milpa? _____

18. Was your family's milpa more productive tan your neighbours? Yes___ NO___

19. When you were a girl did your family have a vegetable garden? Yes___ NO___ (if NO, go to question 21)

20. What did your family grow in your vegetable garden? ¿

21. When you were a girl, did your family have animals to eat or use as transportation? Yes___ NO___ (if NO, go to question 23)

22. What and how many animals did you have as a girl? ¿

| Animals | Number |
|---------|--------|
| | |
| | |
| | |
| | |

- 23.** When you were a girl did your household have a these following ítems? If you had them, what age were you when your family got it?

| | | |
|-------------------|-----------|---|
| Radio | SI__ NO__ | How old were you when you got it? _____ |
| Telephone | SI__ NO__ | How old were you when you got it? _____ |
| Refrigerador | SI__ NO__ | How old were you when you got it? _____ |
| Car | SI__ NO__ | How old were you when you got it? _____ |
| Television | SI__ NO__ | How old were you when you got it? _____ |
| Bicycle/ tricycle | SI__ NO__ | How old were you when you got it? _____ |
| Western bed | SI__ NO__ | How old were you when you got it? _____ |
| Hammock | SI__ NO__ | How old were you when you got it? _____ |

- 24.** How old were you when you were expected to work as hard and be productive in the house as your mother?

SOCIAL STATUS OF THEMOTHER DURING HER CHILDHOOD

- 25.** This is a ladder representing where people stand in society. At the top are the people who are best off- those that have the most money, most education and the best jobs. At the bottom are the people who are the worst off- who have the least money, least education and worst or no jobs. The higher up in the ladder you are, the closer you are to people at the top, the lower you are on the ladder, the closer you are to the bottom. Where would you put your family on the ladder when you were a child? Please put a large X on the rung where you think you stand.

SOCIAL STATUS OF THEMOTHER DURING HER CHILDHOOD

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26. When you were a girl, did you live in a:
City__ Town__ Commissaryship__ Ranch or hacienda __ Other __

If CITY go to question 31

27. How long did it take you to travel to the nearest town or city?
<30min__ 30 min__ <1 hour__ 1- 2 hours__ >2 hours__

28. How often did you travel to the nearest town or city?
Daily__ Weekly__ Monthly__ Yearly__ __ Never__

29. How did you travel to the nearest town or city? ¿
Car/ van (propio) __ Bus__ Bicycle/ tricycle__
Walked__ Animals (horse, donkey) __ Other__

30. What was the name of your childhood home? _____

31. Were the roads or sidewalks paved in your childhood home? Yes__ NO__

32. Can you please tell us if when you were a child, did you family experience any of the following:

- Serious illness of parents, siblings or grandparents? Yes__ NO__
- Death of parents, aunts/ uncles, siblings or grandparents? Yes__ NO__
- Divorce of parents or caregivers? Yes__ NO__
- Job loss of parents or caregivers Yes__ NO__
- Migration of a family member that caused problems in the family Yes__ NO__
- Necessary sale of land, house, jewelry, vehicle, tv or other valuable item Yes__ NO__
- Pawning or house, jewelry, television, etc Yes__ NO__

33. When you were a girl was there any other situation not mentioned above, that caused problems for your family?

34. Were you born in Merida? Yes__ NO__ (If yes, go to question 38)

Las siguientes preguntas serán de cuando se mudó a Mérida.

35. At what age did you completely leave your childhood home? _____

36. How long have you lived in Merida? ____months ____years

37. Why did you move to Merida?

PARTNERSHIP OF THE MOTHER

38. How long have you been married or lived with your partner? _____ Years

39. Do you currently live with your husband or partner? Yes__ NO__

40. Does your husband or partner contribute money to the household? Yes__ NO__

Appendices

| Cereal and Tubers | | Animal origin | | Fat | | Legumes | | | Fruits | | Vegetables | | | |
|-----------------------|------|----------------------------|------|------------|--------|-----------|--------------|------|------------|------|-------------|----------|--|--|
| Food | Freq | Food | Freq | Food | Freq | Food | Freq | | Food | Freq | Food | Freq | | |
| Rice | | Pork | | Mayonnaise | | Beans | | | | | Lettuce | | | |
| Avena | | Pork sausage | | Margarine | | Lentils | | | Watermelon | | Carrot | | | |
| Boxed cereal | | Poultry sausage | | Oil | | Ibes | | | Papaya | | Pumpkin | | | |
| Corn tortilla | | Pork ham | | Lard | | | | | Melon | | Chayote | | | |
| Wheat Tortilla | | Poultry ham | | Sugar | | Dry beans | | | Orange | | Onion | | | |
| Corn dough | | Longaniza | | Honey | | Dairy | | | Apple | | Cauliflower | | | |
| Purchased white bread | | Beef | | Sugar | | Food | Type | Freq | Banana | | Jicama | | | |
| Purchased brown bread | | Fish | | | | Leche | Skimmed | | Other: | | Beetroot | | | |
| French bread | | Seafood | | | | | Semi-Skimmed | | | | Cabbage | | | |
| Potato | | Chicken | | | | | | | | | | Cucumber | | |
| Pasta | | Egg | | | | | | | | | | Tomato | | |
| | | | | | | | | | | | Radish | | | |
| Sweetcorn | | 41. Current diet of Mother | | | | Leche | Full fat | | | | Others: | | | |
| Sweet bread | | | | | | | | | | | | | | |
| Popcorn | | | | | | | | | | | | | | |
| | | | | | | | | | | | Queso | Manche | | |
| | | | | | | | | | | | | D | | |
| | | | | | Panela | | | | | | | | | |
| | | | | | Oaxaca | | | | | | | | | |
| | | | | | | De Bola | | | | | | | | |

42. What is your favourite food right now? (Write all mentioned)

_____ Frequency: _____
 _____ Frequency: _____
 _____ Frequency: _____

43. What other types of food do you eat regularly? (Write all mentioned)

_____ Frequency: _____
 _____ Frequency: _____
 _____ Frequency: _____

CURRENT LIFE OF THE MOTHER

44. What is your current job?: _____

45. What is your spouse's current job?: _____

46. What is the main source of drinking water for your family now?

Piped water _____ Private well _____ Public well _____
 Purchased, purified water _____ Other _____

45. What do you do to clean your drinking water?

Filter with cloth _____ Chlorine and microdin _____ Filter _____ Boil _____
 Nothing _____ Other _____

46. Now does your family use:

Flush toilet _____ Latrine _____ The garden _____ Other _____

43. Right now does your family have the following items?

Radio Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Telephone Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Refrigerador Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Car Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Television Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Tricycle/ bicycle Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Western bed Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

Hammock Yes _____ NO _____

Have you had it for your child's entire life? Yes _____ NO _____,

(if no) How old was your child when you got it? _____

44. What language do you typically use in your house? Spanish _____ Maya _____

45. What other languages do you use in your house? _____

CURRENT SOCIAL STATUS

46. This is a ladder representing where people stand in society. At the top are the people who are best off- those that have the most money, most education and the best jobs. At the bottom are the people who are the worst off- who have the least money, least education and worst or no jobs. The higher up in the ladder you are, the closer you are to people at the top, the lower you are on the ladder, the closer you are to the bottom. Where would you put your family on the ladder when you were a child? Please put a large X on the rung where you think you stand.

CURRENT SOCIAL STATUS

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47. Can you please tell us if any of these things have happened in your family during your child's life:

- Serious illness of parents, siblings or grandparents? Yes___ NO___
- Death of parents, aunts/ uncles, siblings or grandparents? Yes___ NO___
- Divorce of parents or caregivers? Yes___ NO___
- Job loss of parents or caregivers Yes___ NO___
- Migration of a family member that caused problems in the family Yes___ NO___
- Necessary sale of land, house, jewelry, vehicle, tv or other valuable item Yes___ NO___
- Pawning or house, jewelry, television, etc Yes___ NO___

48. Have any other problems that we didn't mention happen to your family since your child has been born?

PREGNANCY OF THE MOTHER

These questions are in relation to your pregnancy with _____ (*ego*).

49. Was this your first pregnancy? ¿Yes___ NO___
50. How many children do you have? _____
51. How many of your children are older than (*ego*)? _____
52. Have any of your children died before they turned 5 years old? ¿ _____
53. When you were pregnant with (*ego*) did you:
 Want to be pregnant___ Want to wait ___ Did not want more children___
54. Did you smoke while pregnant? Yes___ NO___
55. Did anyone in your house smoke while you were pregnant? Yes___ NO___
56. Did you receive medical care while you were pregnant? Yes___ NO___ (If NO, go to question 57)

| | |
|--------------------|--------------|
| First 3 months | Yes___ NO___ |
| Between 4-6 months | Yes___ NO___ |
| After 6 months | Yes___ NO___ |

57. Where did you give birth to your child? _____
 Municipality _____ City _____ State _____

58. Was your child born at:
 Your house___ You're parent's house___ Hospital ___ Rural clinic ___ Health facility ___ Other___

59. What was your child's birth weight? _____ kg

60. If you did not give birth at a hospital or clinic, what was the reason?
 Was not necessary___ Not used to___ Very expensive ___ Bad services ___
 Didn't have time to go ___ Family did not allow ___ It was the best house in the area ___
 Did not know I could___ Other (please specify) _____

61. Who attended the birth?
 Health professional: Doctor___ Nurse___ Midwife___ Other___
 Other person: Friends/ relatives___ Others _____

62. How many weeks were you when you gave birth? _____ weeks _____ months

HEALTH INFORMATION OF _____

63. Do you smoke now? Yes__ NO__
 64. Does anyone smoke in your house? Yes__ NO__
 65. Did you breastfeed? Yes__ NO__
 66. Did you breastfeed within the first 24 hours after birth? Yes__ NO__
 67. How long did you breastfeed? _____(months) _____(years)
 68. When did you stop breastfeeding? _____(months) _____(years)
 69. When did you introduce foods other than breastmilk, including formula or anything else?
_____(months)
 70. Was your child healthy in his/hers first year of life? Yes__ NO__
 71. Was your child healthy in his/hers 1st to 5th years of life? Yes__ NO__
 72. Was your child healthy after his/her 5th year of life? Yes__ NO__
 73. How often has your child had diarrhoea? (frequency)_____ or
Never__ Rarely__ Once a year__ Monthly__ More than once a week__
 74. How many times has your child needed oral rehydration? Yes__ NO__ (If no, go to question 76)
 75. How many time? _____
 76. Has your child been sick in the past 2 weeks? Yes__ NO__
 77. With what? _____
-

Appendices

| Cereal and Tubers | | Animal origin | | Fat | | Legumes | | | Fruits | | Vegetables | | | | | |
|-----------------------|------|--|------|--------------|------|--------------|-------------|-------------|------------|-------|-------------|--------|--|--|--|--|
| Food | Freq | Food | Freq | Food | Freq | Food | Freq | | Food | Freq | Food | Freq | | | | |
| Rice | | Pork | | Mayonnaise | | Beans | | | | | Lettuce | | | | | |
| Avena | | Pork sausage | | Margarine | | Lentils | | | Watermelon | | Carrot | | | | | |
| Boxed cereal | | Poultry sausage | | Oil | | Ibes | | | Papaya | | Pumpkin | | | | | |
| Corn tortilla | | Pork ham | | Lard | | | | | Melon | | Chayote | | | | | |
| Wheat Tortilla | | Poultry ham | | Sugar | | Dry beans | | | Orange | | Onion | | | | | |
| Corn dough | | Longaniza | | Honey | | Dairy | | | Apple | | Cauliflower | | | | | |
| Purchased white bread | | Beef | | Sugar | | Food | Type | Freq | Banana | | Jicama | | | | | |
| Purchased brown bread | | Fish | | | | Leche | Skimmed | | Other: | | Beetroot | | | | | |
| French bread | | Seafood | | | | | | | _____ | _____ | Cabbage | | | | | |
| Potato | | Chicken | | | | | | | _____ | _____ | Cucumber | | | | | |
| Pasta | | Egg | | | | | | | — | — | Tomato | | | | | |
| Sweetcorn | | <div style="border: 1px solid black; padding: 5px; display: inline-block;">78. Current diet of child</div> | | | | Leche | Full fat | | | | Others: | | | | | |
| Sweet bread | | | | | | | | | | | | | | | | |
| Popcorn | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | Queso | Manche | | | | |
| | | | | | | | | | | | | D | | | | |
| | | Panela | | | | | | | | | | | | | | |
| | | Oaxaca | | | | | | | | | | | | | | |
| | | De Bola | | | | | | | | | | | | | | |

Anthropometry

Mother's ID _____ Child's ID _____
 Mother's birth date: __ day __ month ____ year
 Child's birth date: __ day __ month ____ year
 Child's sex: Boy/ Girl

Child's anthropometry

Date _____ Location _____
 Assessor _____ Recorder _____
 BioScan ID number _____
 BioScan Z (impedance) _____

Weight (kg): 1) _____
 Height (cm): 1) _____
 Waist Circumference (cm): 1) _____
 Mid-arm circumference (cm): 1) _____
 Pliegues/ Skinfolds
 Triceps (mm): 1) _____ 2) _____ 3) _____
 Subscapular (mm): 1) _____ 2) _____ 3) _____
 Suprailiac (mm): 1) _____ 2) _____ 3) _____
 Knee Height (cm): 1) _____
 Sitting Height (cm): 1) _____

Mother's anthropometry

Date _____ Location _____
 Assessor _____ Recorder _____
 BioScan ID number _____
 BioScan Z score (impedance) _____

Weight (kg): 1) _____
 Height (cm): 1) _____
 Waist Circumference (cm): 1) _____
 Mid-arm circumference (cm): 1) _____
 Skinfolds
 Triceps (mm): 1) _____ 2) _____ 3) _____
 Knee Height (cm): 1) _____
 Sitting Height (cm): 1) _____

Physical Development Scale (Girls only)

The next questions are about changes that may be happening to your body. These changes normally happen to young people at different ages. Please choose only one answer for each question unless the question says otherwise.

- ☐ self-report
- ☐ interview style

During certain times, young people start to grow a lot and quickly. This is called a growth spurt. Would you say that your growth in height:

- ☐ has not yet begun to spurt ("spurt" means more growth than usual)
- ☐ has barely started
- ☐ is definitely underway
- ☐ seems completed

How about the growth of your body hair? ("Body hair" means hair other than on your head, for example pubic hair.) Would you say that your body hair growth:

- ☐ not yet started

(There is no pubic hair)

- ☐ has barely started

(There is a little long, lightly colored hair; this hair may be straight or curly.)

- ☐ is definitely underway

(The hair is darker than before. It is coarser and more curled. It has spread out and thinly covers a larger area.)

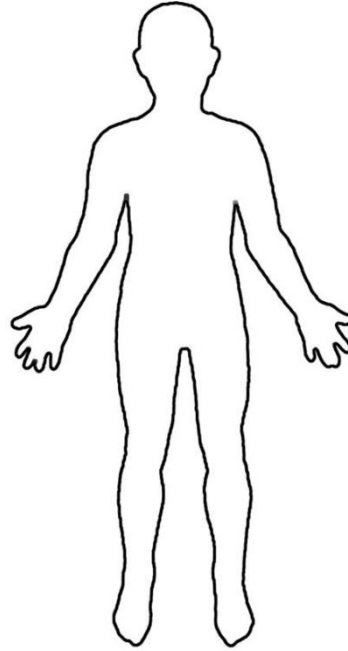
- ☐ seems complete

(The hair is now as dark, curly, and coarse as that of an adult female and forms a triangular pattern in the pubic area.)

Has body hair started to grow on any of the following areas? (Check all that apply)

- ☐ Underarms
- ☐ Pubic area
- ☐ Lower legs

Place an X on the figure below where your body hair is.



Have you noticed any skin changes, especially spots? Skin changes:

- ☐ have not yet started
- ☐ have barely started
- ☐ are definitely underway
- ☐ seem complete

Have you noticed your skin is becoming more oily?

- ☐ No
- ☐ yes

Have you noticed any changes in how your body smells, especially from under your arms (B.O.)?

- ☐ have not noticed any smell
- ☐ have started to smell a little
- ☐ has definitely starting to smell
- ☐ smell everyday

Have your breasts begun to grow?

- ☐ have not started (The breast area is flat.)
- ☐ have barely started (The nipple area has become larger and darker and there is a small mound under the nipple area called the breast bud.)
- ☐ definitely underway (The nipple area and breast are growing and are larger than before)
- ☐ seems complete (The breasts are fully developed and only the nipple sticks out. Breasts look like an adult's but they still may get bigger.)

Do you think your development is any earlier or later than most other girls your age?

- ☐ Much earlier

- ☐ Somewhat earlier
- ☐ About the same
- ☐ Somewhat later
- ☐ Much later

Have you begun your periods?

- ☐ If No→ **Please tell the interviewer you are finished. Fold and place this part of the questionnaire in the envelope.**
- ☐ If Yes, continue below



How old were you when you started your periods?

I was _____ years and _____ months old when I got my first period.

I was in year _____ at school.

- ☐ I don't remember exactly

Do you or have you ever taken hormonal birth control?

- ☐ No
- ☐ Yes, if so, when?

How many days are there from the first day of one of your menstrual periods to the first day of the next?

- ☐ 26-32 days
- ☐ less than 26 days
- ☐ More than 32 days
- ☐ Other _____
- ☐ I don't know

How long does the bleeding normally last?

- ☐ 1-2 days
- ☐ 3-5 days
- ☐ 5-7 days
- ☐ >7 days
- ☐ It changes
- ☐ I don't know

When was the first day of your last menstrual period? (You can refer to your own calendar or the one provided to help you remember)

Date: __/__/__ (DD/MM)

OR How many: days ago _____ weeks ago _____

- ☐ I don't know

Please tell the interviewer you are finished. Fold and place this part of the questionnaire in the envelope.

2. Spanish

Estado nutricional y de salud en un grupo de yucatecos de origen maya con doble carga nutricional

Departamento de Ecología Humana de Cinvestav-Mérida y Departamento de Ciencias Humanas, Universidad de Loughborough, Inglaterra
Cuestionario para parejas madre - hijo mayas urbanos

| | |
|---|--|
| Fecha de nacimiento de la madre Día ____ Mes ____ Año ____ | Fecha de nacimiento del niño Día ____ Mes ____ Año ____ |
|---|--|

41. ¿Cuántos hermanos(as) y hermanitos(as) tiene? _____
42. ¿Cuántos hermanos(as) mayores que usted tiene? _____
43. ¿Cuántos de sus hermanos(as) y hermanitos(as) vivieron al menos hasta los 5 años? _____
44. ¿Tuvo algún hermano(a) o hermanito(a) que murió antes de los 5 años? Sí ____ No ____
¿Cuántos? _____

NIÑEZ DE LA MADRE

45. Ocupación de su madre cuando usted era niña: _____
46. Ocupación de su padre cuando usted era niña: _____

47. FRECUENCIA DE ALIMENTOS DE LA MADRE DURANTE SU INFANCIA

| | |
|--|--|
| Refrescos embotellados (Coca Cola, Pepsi Cola o jugos de bolsa o botella) | |
| Comida empaquetada (Galletas, Sabritas, charritos, chicharrones de harina) | |
| Carne de pollo, puerco o res | |

Frecuencia: 0: Nunca; 1: Diario; 2: Regularmente; 3: Ocasionalmente; 4: Sólo en fechas festivas (Navidad, Hanal Pixan, Reyes, Año Nuevo o similares).

48. ¿Se sentía satisfecha o llena después de cada comida? Si _____ No _____
(Si responde si, explica) _____

49. ¿Cuál era su comida favorita cuando era niña? (*Escribe las que mencione*)

____ Frecuencia: _____
____ Frecuencia: _____
____ Frecuencia: _____

50. ¿Qué otro tipo de comidas consumía regularmente cuando usted era niña?

____ Frecuencia: _____
____ Frecuencia: _____
____ Frecuencia: _____

VIVIENDA DE LA MADRE DURANTE SU INFANCIA

51. ¿Qué tipo de agua para beber consumía su familia cuando usted era niña?

Agua de tubería _____ De pozo en casa _____
De pozo público _____ Pipa de agua _____
Purificada _____ Otro _____

52. ¿Qué se hacía en su casa para limpiar el agua cuando usted era niña?

Colarlo con telas _____ Cloro y microdin _____ Filtro _____
Hervir _____ Nada _____ Otro _____

53. ¿Cuando Usted era niña, en su casa había

Baño (sanitario) _____ Fosa /letrina _____ Se hacía el patio _____ Otro _____

54. ¿De qué materiales estaba construida su casa?

Techo _____
Paredes _____
Piso _____

55. ¿Cuál era la principal fuente de luz que tenían en su casa cuando usted era niña?

Electricidad _____ Queroseno _____ Gas _____ Petróleo _____ Velas _____ Otro _____

56. ¿Cuándo Usted era niña su familia tenía milpa? SI__ NO__ (si contestó NO pasar a la pregunta 19)

57. ¿Cuántos mecates tenía la milpa? _____

58. ¿La milpa que tenía su familia cuando Usted era nina era más productiva que la de sus vecinos? SI__ NO__

59. ¿Cuándo Usted era niña su familia tenía solar en su casa? SI__ NO__ (si contestó NO pasar a la pregunta 21)

60. ¿Qué se producía en el solar de su casa cuando Usted era niña?

61. ¿Cuándo Usted era niña en su casa había animales para consumir o transporte? SI__ NO__ (si contestó NO pasar a la pregunta 23)

62. ¿Qué animales tenía y cuántos de ellos?

| Animales | Número |
|----------|--------|
| | |
| | |
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| | |

- 63.** ¿Cuándo Usted era niña en su casa había alguno de los siguientes bienes? Y si los tenía, ¿Recuerda cuando los obtuvieron?

| | | |
|--------------------|-----------|----------------------------------|
| Radio | SI__ NO__ | Edad de usted o en qué año _____ |
| Teléfono | SI__ NO__ | Edad de usted o en qué año _____ |
| Refrigerador | SI__ NO__ | Edad de usted o en qué año _____ |
| Carro | SI__ NO__ | Edad de usted o en qué año _____ |
| Televisor | SI__ NO__ | Edad de usted o en qué año _____ |
| Triciclo/bicicleta | SI__ NO__ | Edad de usted o en qué año _____ |
| Base de cama | SI__ NO__ | Edad de usted o en qué año _____ |
| Hamaca | SI__ NO__ | Edad de usted o en qué año _____ |

- 64.** ¿A qué edad se esperaba que usted trabajara e hiciera todas las cosas domésticas que hacia su madre? _____

STATUS SOCIAL DE LA MADRE DURANTE SU INFANCIA

- 65.** Esta es una escala que representa la ubicación del status social de las personas. En la parte superior están ubicadas las personas que tienen mayor nivel económico, mayor educación y los empleos mejor pagados. En la parte inferior están las personas de menor nivel económico, con menor educación o sin educación y/o no tienen empleo. Mientras más ascienda Usted en la escala más cerca esta de las personas de clase alta, mientras más descienda estará más cerca de la clase baja. ¿En qué escala colocaría a tu familia cuando Usted era niña?. Por favor escribe una X en el peldaño donde creas que están ubicados.

STATUS SOCIAL DE LA MADRE DURANTE SU INFANCIA

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66. ¿Cuándo Usted era niña vivía en ciudad, pueblo, comisaría o rancho?

Ciudad__ Pueblo__ Comisaría__ Rancho o hacienda __ Otros __

Sí vivió en la ciudad pasar a pregunta 31

67. ¿Cuánto tiempo le tomaba llegar al pueblo o ciudad más cercana?

<30min__ 30 min__ <1 hora__ 1- 2 horas__ >2 horas__

68. ¿Qué tan seguido viajaba al pueblo o ciudad más cercana?

Diario__ Semanal__ Mensual__ Anual__ Nunca__

69. ¿Qué transporte utilizaba para la llegar al pueblo o ciudad más cercana?

Carro/van (propio) __ Autobús__ Bicicleta/triciclo__
Caminando__ Animales (caballo, burro) __ Otro__

70. ¿Cuál es el nombre del lugar en donde vivía? _____

71. ¿Estaban pavimentadas las calles o caminos en donde vivía? SI__ NO__

72. Necesito saber si durante su niñez y juventud hubo en su familia casos como:

- Enfermedad grave de alguno de los padres, hermanos, abuelos SI__ NO__
- Muerte de sus padres o hermanos(as), abuelos, cuidadores SI__ NO__
- Divorcio de los padres o cuidadores SI__ NO__
- Pérdida de trabajo de los padres o cuidadores SI__ NO__
- Emigración de algún miembro de la familia y que esto haya causado problemas en la familia SI__ NO__
- Venta de terrenos, casa, joyas, autos, tv u otros por necesidad SI__ NO__
- Empeño de casa, joyas, tv SI__ NO__

73. Cuando Usted era niña hubo cualquier otra situación además de las antes mencionadas en su familia que haya causado problemas para Usted y su familia

74. ¿Nació en Mérida? SI__ NO__ (Si contestó SI pasar a la pregunta 32)

Las siguientes preguntas serán de cuando se mudó a Mérida.

75. ¿A qué edad se mudó definitivamente del lugar en donde vivió durante su niñez? _____

76. ¿Hace cuánto tiempo que vive en Mérida? ____meses ____años

77. ¿Por qué decidió mudarse a Mérida?

PAREJA DE LA MADRE

78. ¿Cuánto tiempo tiene de casada o de vivir con su actual pareja? _____ años

79. ¿Vive con su esposo o pareja actualmente? SI__ NO__

80. ¿Su pareja aporta dinero para la manutención de la casa? SI__ NO__

Appendices

| CEREALES Y TUBÉRCULOS | | ALIMENTOS DE ORIGEN ANIMAL | | GRASAS | | LEGUMINOSAS | | | FRUTAS | | VERDURAS | |
|-----------------------|------|-------------------------------------|------|-----------------|---------|--------------|------------|--|--|------|-----------|------|
| Alimento | Frec | Alimento | Frec | Alimento | Frec | Alimento | Frec | | Alimento | Frec | Alimento | Frec |
| Arroz | | Cerdo | | Mayonesa | | Frijol | | | Sandía | | Lechuga | |
| Avena | | Salchicha de cerdo | | Margarina | | Lentejas | | | Papaya | | Zanahoria | |
| Cereal Caja | | Salchicha de pavo/pollo | | Aceite | | Ibes | | | Melón | | Calabaza | |
| Tortilla de maíz | | Jamón de cerdo | | Manteca | | | | | Naranja | | Chayote | |
| Tortilla de Harina | | Jamón de pavo/pollo | | AZÚCARES | LÁCTEOS | Frutos secos | | | Coliflor | | Cebolla | |
| Masa de maíz | | Longaniza | | Miel | | | | | Manzana | | Plátano | |
| Pan blanco de caja | | Res | | Azúcar | | Alimento | Tipo | | Otras: _____ _____ _____ _____ | | Jicama | |
| Pan integral de caja | | Pescado | | Semi descremada | | Leche | Descremada | | _____ _____ _____ _____ | | Remolacha | |
| Pan francés | | Mariscos | | | | | | | | | Repollo | |
| Papa | | Pollo | | | | | | | | | Pepino | |
| Pastas | | Huevo | | | | | | | | | Tomate | |
| | | | | | | | | | | | Rábano | |
| Elote | | | | | | Queso | | | | | Otras: | |
| Pan dulce | | | | | | | | | | | | |
| Palomitas | | | | | | | | | | | | |
| | | | | | | | | | | | | |
| | | 41. Alimentación actual de la madre | | | | | D | | | | | |
| | | | | | | | Panela | | | | | |
| | | | | | | | Oaxaca | | | | | |
| | | | | | | | De Bola | | | | | |

42. ¿Actualmente cuál es su comida favorita? (*Escribe las que mencione*)

_____ Frecuencia: _____
 _____ Frecuencia: _____
 _____ Frecuencia: _____

43. ¿Qué otro tipo de comidas consume regularmente? (*Escribe las que mencione*)

_____ Frecuencia: _____
 _____ Frecuencia: _____
 _____ Frecuencia: _____

VIVIENDA ACTUAL DE LA MADRE

44. Que este su ocupación actualmente?: _____

45. Que este cupación de su esoposo actualmente?: _____

46. ¿Cuál es la principal fuente de agua para beber que consume su familia actualmente?

Agua de tubería ___ Agua subterránea (pozo privado) ___ De pozo (público) ___
 Pipa de agua ___ Garrafón ___ Otro _____

45. ¿Qué hace para limpiar el agua actualmente?

Filtrar con telas ___ Cloro y microdin ___ Filtro ___ Hervir ___
 Nada ___ Otro _____

47 Actualmente en su casa hay

Baño (sanitario) ___ Fosa /letrina ___ Se hace el patio ___ Otro _____

78. ¿Actualmente en su casa había alguno de los siguientes bienes?

Radio SI___ NO___

Durante toda su vida contó con este aparato SI___ NO___,
 (si no) ¿Hace cuantos años? _____

Teléfono SI___ NO___

Durante toda su vida contó con dicho aparato eléctrico SI___ NO___, (si no)
 ¿Hace cuantos años? _____

Refrigerador SI___ NO___

Durante toda su vida contó con dicho aparato eléctrico SI___ NO___, (si no)
 ¿Hace cuantos años? _____

Carro SI___ NO___

Durante toda su vida contó con dicho medio de transporte SI___ NO___,
 (si no) ¿Hace cuantos años? _____

Televisor SI___ NO___

Durante toda su vida contó con dicho aparato eléctrico SI___ NO___, (si no)
 ¿Hace cuantos años? _____

Triciclo/bicicleta SI___ NO___

Durante toda su vida contó con dicho medio de transporte SI___ NO___,
 (si no) ¿Hace cuantos años? _____

Base de cama SI___ NO___

Durante toda su vida contó con este bien SI___ NO___,
 (si no) ¿Hace cuantos años? _____

Hamaca SI___ NO___

Durante toda su vida contó con este bien SI___ NO___,
 (si no) ¿Hace cuantos años? _____

79. Que idioma habla habitualmente in su casa? Espanol ___ Maya _____

80. Que otros idiomas habla in su casa? _____

STATUS SOCIAL ACTUAL

81. Esta es una escala que representa la ubicación del status social de las personas. En la parte superior están ubicadas las personas que tienen mayor nivel económico, mayor educación y los empleos mejor pagados. En la parte inferior están las personas de menor nivel económico, con menor educación o sin educación y/o no tienen empleo. Mientras más ascienda Usted en la escala más cerca esta de las personas de clase alta, mientras más descienda estará más cerca de la clase baja. En que escala colocaría a su familia ahora. Por favor escriba una X en el peldaño donde crea que están ubicados.

STATUS SOCIAL ACTUAL

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82. Necesito saber si desde que nació _____ (*ego*) ha habido en su familia casos como:

- | | |
|---|-----------|
| - Enfermedad grave de alguno de los padres, hermanos, abuelos | SI__ NO__ |
| - Muerte de sus padres o hermanos(as), abuelos | SI__ NO__ |
| - Divorcio de los padres | SI__ NO__ |
| - Pérdida de trabajo de los padres | SI__ NO__ |
| - Emigración de algún miembro de la familia y que esto haya causado problemas en la familia | SI__ NO__ |
| - Venta de terrenos, casa, joyas, autos, tv u otros por necesidad | SI__ NO__ |
| - Empeño de casa, joyas, tv | SI__ NO__ |

83. Desde que _____ nació ha habido alguna otra situación además de las antes mencionadas en su familia que haya causado problemas para Usted y su familia

EMBARAZO DE LA MADRE

En relación a su embarazo de _____ (*ego*).

84. ¿Fue su primer embarazo? SI__ NO__

85. ¿Cuántos hijos tiene? _____

86. ¿Cuántos hermanos mayores tiene el niño? _____

87. ¿Alguno de sus hijos falleció antes de cumplir 5 años? _____

88. Cuando se embarazó del niño(a)

Quería embarazarse__ Quería esperar __ No quería tener más hijos__

89. ¿Fumò durante su embarazo? SI__ NO__

90. ¿Alguien que fumò en su casa durante su embarazo? SI__ NO__

91. ¿Recibió cuidados médicos durante su embarazo? SI__ NO__ (*si la respuesta fue negativa pasar a la pregunta 56*)

Primeros tres meses SI__ NO__

Entre el cuarto y sexto mes SI__ NO__

Después del sexto mes SI__ NO__

92. Lugar de nacimiento del niño(a) _____

Comisaria/Municipio Ciudad Estado

93. El niño(a) nació en:

Casa propia__ De sus padres__ Hospital __ Clínica rural __ Casa de salud __ u otra__

94. ¿Cuánto pesó su hijo(a) al nacer? _____ kg

95. Si no dio a luz en un hospital o clínica ¿cuál fue la razón?

No era necesario__ No se acostumbraba__ Muy caro __ El servicio es deficiente __

No hubo tiempo para ir __ La familia no lo permitió __ Es mejor el cuidado en casa__

Carece de conocimiento__ Otro (por favor especifica) __

96. ¿Quién atendió el parto?

Profesionales de la salud: Doctor__ Enfermera__ Partera__ Otro__

Otras personas: Amigos/parientes__ Otras __

97. Al cuánto tiempo de embarazo nació su hijo(a) _____semanas _____meses

INFORMACIÓN DE LA SALUD DE _____

98. ¿Fuma ahora? SI__ NO__
99. ¿Alguien que fuma en su casa ahora? SI__ NO__
100. ¿Alguna vez le dio *chuchú* o pecho? SI__ NO__
101. ¿Le dio *chuchú* antes de que tuviera 24 horas de nacido? SI__ NO__
102. ¿Por cuánto tiempo le dio pecho o *chuchú*? _____(meses) _____(años)
103. ¿Qué edad tenía su hijo(a) cuando dejó de darle pecho o *chuchú*? _____(meses) _____(años)
104. ¿A qué edad le dio alimentos diferentes a la leche materna incluyendo leche de fórmula u otro? _____(meses)
105. ¿Fue un(a) niño(a) saludable durante su primer año de vida? SI__ NO__
106. ¿Fue un(a) niño(a) saludable al menos hasta los 5 años? SI__ NO__
107. ¿Ha sido un niño saludable desde los 5 años hasta ahora? SI__ NO__
108. ¿Cuántas veces desde que nació el (la) niño(a) ha tenido diarrea? (Fr)_____ o Nunca__ Rara vez__ Una vez al año__ Mensualmente__ Más de una vez al mes__
109. ¿Alguna vez su hijo(a) ha necesitado rehidratación oral? SI__ NO__ (Si contesta no, pasar a la pregunta 70)
110. ¿Cuántas veces? _____
111. ¿Ha estado enfermo(a) su hijo(a) en las dos últimas semanas? SI__ NO__
112. ¿De qué? _____
-

Appendices

| CEREALES Y TUBÉRCULOS | | ALIMENTOS DE ORIGEN ANIMAL | | GRASAS | | LEGUMINOSAS | | | FRUTAS | | VERDURAS | |
|-----------------------|------|------------------------------------|------|-----------------|---------|--------------|---------------------------|------|----------|--------|-----------|------|
| Alimento | Frec | Alimento | Frec | Alimento | Frec | Alimento | Frec | | Alimento | Frec | Alimento | Frec |
| Arroz | | Cerdo | | Mayonesa | | Frijol | | | | | Lechuga | |
| Avena | | Salchicha de cerdo | | Margarina | | Lentejas | | | Sandía | | Zanahoria | |
| Cereal Caja | | Salchicha de pavo/pollo | | Aceite | | Ibes | | | Papaya | | Calabaza | |
| Tortilla de maíz | | Jamón de cerdo | | Manteca | | | | | Melón | | Chayote | |
| Tortilla de Harina | | Jamón de pavo/pollo | | AZÚCARES | LÁCTEOS | Frutos secos | | | Naranja | | Cebolla | |
| Masa de maíz | | Longaniza | | Miel | | | | | Manzana | | Coliflor | |
| Pan blanco de caja | | Res | | Azúcar | | Alimento | Tipo | Frec | Plátano | | Jicama | |
| Pan integral de caja | | Pescado | | Semi descremada | | Leche | Descremada | | Otras: | | Remolacha | |
| Pan francés | | Mariscos | | | | | | | | | Repollo | |
| Papa | | Pollo | | | | | | | | | Pepino | |
| Pastas | | Huevo | | | | | | | | | Tomate | |
| | | | | | | | | | | | Rábano | |
| Elote | | 78. Alimentación actual de la niño | | | | Queso | Entera | | | Otras: | | |
| Pan dulce | | | | | | | Manche | | | | | |
| Palomitas | | | | | | | | | | | | |
| | | | | | | | D | | | | | |
| | | | | | | | Panela | | | | | |
| | | | | | | | 68. ALIMENTACIÓN DEL NIÑO | | | | | |

Fecha: _____
 Día/ mes/ año
 Edad: _____ (años)
 ID: _____
 Encuestó: _____
 (iniciales)

1. ESCALA DE DESARROLLO FÍSICO (SOLO NIÑAS)

Las siguientes preguntas son acerca de los cambios corporales que puedes estar experimentando. Estos cambios normalmente ocurren en gente joven a diferentes edades. Por favor elige sólo una respuesta a menos que la pregunta señale lo contrario.

- 1. En determinado período, los jóvenes empiezan a crecer rápidamente. A esto se le ha llamado “estirón de crecimiento”. Por lo tanto puedes decir que tu crecimiento en relación a la talla:**

- ☐ Todavía no ha alcanzado el estirón
- ☐ A penas esta iniciando
- ☐ Definitivamente esta en proceso
- ☐ Se ha completado
- ☐

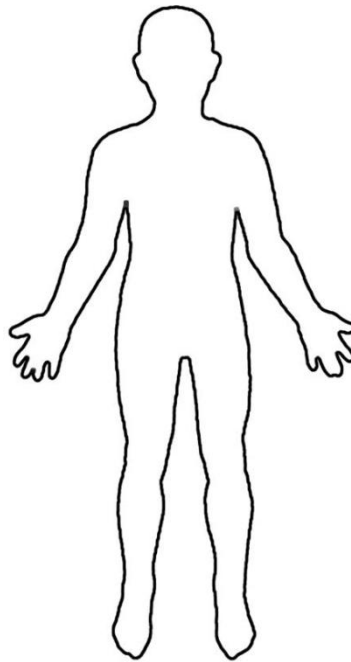
1.1 Acerca del crecimiento del vello corporal (“Vello corporal” significa tener pelo en otras areas del cuerpo además de la cabeza, por ejemplo, vello púbico). Puedes decir que el crecimiento de tu vello corporal:

- ☐ No ha iniciado
(No hay vello púbico)
- ☐ Apenas esta iniciando
(Hay un pequeño crecimiento y decoloración del vello)
- ☐ Definitivamente esta en proceso
(El vello es más oscuro que antes. Es más rizado y se esta extendiendo el crecimiento).
- ☐ Se ha completado
(El vello es oscuro, rizado y grueso como el de una mujer adulta y tiene una forma triangular en el area púbica).

1.1.1 El vello corporal ha estado creciendo en alguna de las siguientes áreas:

- ☐ Axilas
- ☐ Area púbica
- ☐ En la parte baja de las piernas

Marca con una X el área de la figura donde haya habido crecimiento de vello corporal.



2. ¿Has notado algún cambio en la piel especialmente la presencia de acné? Por lo tanto los cambios en la piel:

- ☐ No han iniciado
- ☐ A penas están iniciando
- ☐ Definitivamente están en proceso
- ☐ Se han completado

1.2 ¿Has notado si tu piel se ha vuelto más aceitosa?

- ☐ No
- ☐ Si

1.3 ¿Has notado algún cambio en el olor de tu cuerpo, especialmente en la zona de las axilas?

- ☐ No e notado ningún cambio en el olor
- ☐ Hay cambio mínimos en el olor
- ☐ Definitivamente e notado cambios en el olor
- ☐ Si hay cambios en olor diariamente

1.4 ¿Has notado algún cambio en el tamaño de tus senos?

- ☐ No han habido cambios

(El area de los senos es plana)

- ☐ A penas han iniciado los cambios

(El área del pezón ha empezado a crecer y cambiar de color y hay crecimiento en el area del pezón llamado “brote del seno”)

- ☐ Definitivamente esta en proceso el crecimiento

(El area del pezón y el seno están creciendo y son más grandes que antes)

- ☐ Se ha completado el crecimiento

(Los senos estan completamente desarrollados y solo el pezón sobresale. Los senos lucen como los de una mujer adulta pero aún así pueden crecer más).

1.5 ¿Piensas que tu desarrollo es más temprano o tardío que el de la mayoría de las niñas de tu edad?

- ☐ Muy temprano
- ☐ Un tanto temprano
- ☐ Igual que el de las demás
- ☐ Un tanto tardío
- ☐ Muy tardío

1.6 ¿Has iniciado tus períodos menstruales?

☐ → si No **Por favor dile a la persona que la entrevista ha finalizado. Dobla y guarda esta parte del cuestionario en un sobre. Please tell the interviewer you are finished.**

☐ Si la respuesta es SI, continúe abajo



1.6.1 ¿Qué edad tenías cuando tuviste tu primera menstruación?

Tenía _____ años y _____ meses de edad cuando tuve mi primera menstruación.

Estaba en (grado escolar)_____.

☐ No recuerdo exactamente

1.6.2 ¿Alguna vez has tomado píldoras anticonceptivas, inyección de hormonas o algún implante?

- ☐ No
- ☐ Si

Si la respuesta es SI, ¿cuándo?

1.6.3 ¿Cada cuanto tiempo inicia tu período menstrual?

- ☐ 26-32 días
- ☐ Menos de 26 días
- ☐ Más de 32 días
- ☐ Otro _____
- ☐ No lo sabe

1.6.4 ¿Cuánto tiempo dura tu sangrado?

- ☐ 1-2 días
- ☐ 3-5 días
- ☐ 5-7 días
- ☐ >7 días
- ☐ Varía mucho
- ☐ No lo sabe

1.6.5 ¿Cuándo fue el primer día de tu último período menstrual?

Fecha: __/__/__ (DD/MM)

O Hace cuantos: días _____ semanas _____

☐ No lo sabe

Por favor dile a la persona que la entrevista ha finalizado. Dobla y guarda esta el cuestionario en un sobre.

Ficha Antropométrica

ID de Madre _____ ID de Niño/a _____
 Fecha de nacimiento de la Madre: __ día __mes ____año
 Fecha de nacimiento del Niño/a: __día __mes ____año
 Sexo del niño/a: Hombre/ Mujer

Antropometría del Niño

Fecha/ Date _____ Lugar/ Location _____
 Medidor/ Assessor _____ Apuntador/Recorder _____
 BioScan ID numero/ number _____
 BioScan Z (impedancia) _____

Peso/ Weight (kg): 1) _____
 Talla/ Height (cm): 1) _____
 Circunferencia de Cintura/ Waist Circumference (cm): 1) _____
 Circunferencia Media del brazo/Mid-arm circumference (cm): 1) _____
 Pliegues/ Skinfolds
 Tricipital/Triceps (mm): 1) _____ 2) _____ 3) _____
 Subescapular/Subscapular (mm): 1) _____ 2) _____ 3) _____
 Suprailiaco/Suprailiac (mm): 1) _____ 2) _____ 3) _____
 Altura de Rodilla/Knee Height (cm): 1) _____
 Talla Sentada/ Sitting Height (cm): 1) _____

Antropometría de la Madre

Fecha/ Date _____ Lugar/ Location _____
 Medidor/ Assessor _____ Apuntador/Recorder _____
 BioScan ID numero/ number _____
 BioScan Z score (impedancia) _____

Peso/ Weight (kg): 1) _____
 Talla/ Height (cm): 1) _____
 Circunferencia de Cintura/ Waist Circumference (cm): 1) _____
 Circunferencia Media de brazo/ Mid-arm circumference (cm): 1) _____
 Pliegues/ Skinfolds
 Tricipital/Triceps (mm): 1) _____ 2) _____ 3) _____
 Altura de Rodilla/Knee Height (cm): 1) _____
 Talla Sentada/ Sitting Height (cm): 1) _____

C. Summary of participants measured by each assessor

Intra-observer measurements were not recorded. Therefore this appendix analyses the differences within the entire dataset by assessor.

1. Anthropometry

The only measure which was significantly different between assessors was triceps skinfolds, which was significantly different in both children and mothers (Table C1). *Post hoc* tests for the children found that AV and JT were significantly different for triceps skinfold z-scores. *Post hoc* tests for the mothers' triceps skinfolds found that HW and JT were significantly different. However the significant differences lay between the assessor who performed the fewest assessments and those who performed the most. As triceps skinfolds are included in the equation for arm fat area and arm fat index, these results have to be very cautiously interpreted.

Table C.1: Summary of anthropometric measurements of Maya children and mothers measured by different assessors using ANOVA

| | | HW | AV | JT | p-value |
|-----------------------------|-------------------------------|---------------|---------------|---------------|---------|
| Children¹ | N (%) | 39 (69.4) | 10 (17.9) | 7 (12.5) | |
| | Height z-score | -1.14 (0.89) | -1.28 (0.92) | -1.04 (0.82) | 0.851 |
| | Weight z-score | -0.53 (0.81) | -0.10 (1.24) | -0.65 (0.53) | 0.328 |
| | Waist circumference z-score | 0.25 (0.69) | 0.72 (1.13) | 0.03 (0.41) | 0.142 |
| | Sitting height z-score | -0.75 (0.92) | -0.73 (0.98) | -0.69 (0.91) | 0.989 |
| | Triceps skinfold z-score | 0.55 (0.94) | 1.09 (0.71) | -0.18 (0.31) | 0.015 |
| | Sub-scapular skinfold z-score | 0.59 (0.81) | 1.24 (1.06) | 0.43 (0.54) | 0.067 |
| | Supra-iliac skinfold z-score | 0.70 (1.04) | 1.12 (1.32) | 0.47 (0.48) | 0.405 |
| | Mid-arm circumference z-score | -0.00 (1.13) | 0.40 (1.63) | -0.49 (0.89) | 0.335 |
| | | | | | |
| Mothers² | N (%) | 41 (71.9) | 10 (17.5) | 6 (10.5) | |
| | Height | 147.51 (4.54) | 145.24 (8.52) | 146.03 (7.30) | 0.312 |
| | Weight | 65.37 (11.19) | 62.78 (8.53) | 59.02 (7.30) | 0.347 |
| | Waist circumference | 88.26 (8.55) | 89.73 (8.61) | 85.80 (10.15) | 0.696 |
| | Sitting height | 79.22 (2.71) | 78.35 (2.59) | 78.57 (1.89) | 0.591 |
| | Triceps skinfold | 31.24 (8.68) | 28.41 (5.36) | 22.57 (6.13) | 0.046 |
| | Mid- arm circumference | 31.08 (3.96) | 31.14 (2.65) | 28.52 (1.88) | 0.267 |

¹The children measured by MLA (n=1) and HA (n=1) were excluded from this analysis.

²The one mother measured by MLA was excluded from this analysis.

2. SES indices

The interviewers obtained significantly different outcomes for the family's current economic stability index (Table C.2). This index was not included in any of the main analyses of this thesis.

Table C.2: Summary of Maya women's recalled childhood and current socio-economic status by different interviewer using independent *t*-tests^{1,2}

| | | AV | JT | p-value |
|-------------------------------------|----------------------------|-------------|-------------|----------------|
| | N (%) | 24 (43.6) | 31 (56.4) | |
| Recalled SES indices | Consumer durable ownership | 1.83 (1.37) | 2.19 (1.25) | 0.314 |
| | Economic stability | 1.83 (1.09) | 1.42 (1.15) | 0.181 |
| | Family stability | 2.88 (1.23) | 2.73 (1.17) | 0.667 |
| | Modernisation | 1.25 (0.84) | 1.24 (0.65) | 0.947 |
| Current SES indices | Consumer durable ownership | 3.96 (1.07) | 4.32 (1.05) | 0.212 |
| | Economic stability | 5.17 (1.56) | 4.81 (1.33) | 0.013 |
| | Family stability | 3.39 (0.94) | 3.77 (0.99) | 0.340 |

¹The mothers interviewed by MLA (n=1) and SL (n=2) were excluded from this analysis

²All indices are additive indices

D. *Recall SES and linear growth extra analyses*

SES indices were compared to stature, ELL and SHR using Spearman's correlations (Table D.1, 2). The indices created using PCA were more consistently significant than the additive indices. For stature and SHR the only significant indices were PCA indices. Therefore in ELL, in which both the additive and PCA indices for Modernisation were significant in the bivariate analysis, the PCA index was chosen for inclusion in the multiple regression models for consistency in comparison of analyses across outcomes.

If the individual SES variable was binary, an independent t-test or Mann Whitney U test was performed to compare groups for the anthropometric outcomes (height, SHR, square root of SHR, and ELL). If the SES variable had three categories, an analysis of variance (ANOVA) or Kruskal Wallace test was performed on the anthropometric outcomes. Variables that were borderline significant ($p < 0.1$) were included in the final regression models in order to include relevant predictors that may not be significant in simple analyses. The variables that were significant for SHR were compared to the square root of SHR for inclusion in the final regression model.

Each demographic variable was assessed separately for inclusion in the final regression model as each variable has been shown to have independent and differential impacts on health and growth, negating the usefulness of an index. The individual demography variables were plotted against each linear growth measure (stature, ELL and SHR). This helped to determine which variables had a linear relationship and thus could be used in a linear regression or correlation and which did not. Where a natural break in the data occurred, the data was grouped accordingly. The new variables with two or three groups were then tested for significance in different in the anthropometric outcomes using t-tests or analysis of variance (ANOVA) respectively.

For birth order, a scatter plot was produced against each linear growth measure (stature, ELL and SHR) (Figure D.1). For both stature and ELL, the first and second born women appeared different to the third and later born women. Using an ANOVA, significant differences were detected in the ELL between the second and later born women. No significant differences were found between the statures and

birth order of women. Thus birth order was split into two groups for the linear regression for ELL: first and second born versus and third and later born.

Scatter plots of number of siblings versus linear growth measures were created (Figure D.2). No linear relationship was apparent, nor were there any clear breaks in the data. Therefore the data was split into tertiles. The tertiles of sibling number were found to be borderline significant in their associations with ELL and SHR using an ANOVA.

For sibling death (siblings who died before 5 years), the women were grouped into those who had a sibling die in infancy and those who had not. This variable was then compared with the linear growth measurements using an independent *t*-test. As six women had more than one sibling die in infancy, a scatter plot was produced against each linear growth measure to determine if multiple sibling deaths was related to linear growth. Those with multiple sibling deaths appeared to be different to the women with zero or one sibling deaths however an ANOVA failed to find significant differences between the three groups, possibly because of the small sample size of the final group. Since sibling death was significant overall, though no significant differences were found in *post hoc* tests, it was included in the final model building process.

Table D.1 Bivariate tests and significance of maternal childhood economic and family variables versus linear growth measures

| Index | Variable | Height | | Estimated leg length | | Sitting height ratio (square root) | | Sitting height ratio | |
|--------------------|---------------------|-----------------|--------------|----------------------|--------------|------------------------------------|--------------|----------------------|--------------|
| | | Test | p | Test | p | Test | p | Test | p |
| Ownership | Animals | t-test | 0.477 | t-test | 0.6 | t-test | 0.945 | Mann Whitney | 0.85 |
| | Radio | t-test | 0.331 | t-test | 0.229 | t-test | 0.391 | Mann Whitney | 0.415 |
| | Telephone | t-test | 0.623 | t-test | 0.562 | t-test | 0.668 | Mann Whitney | 0.692 |
| | Refrigerator | <i>t-test</i> | <i>0.073</i> | t-test | 0.047 | t-test | 0.21 | Mann Whitney | 0.181 |
| | Car | t-test | 0.172 | t-test | 0.112 | t-test | 0.279 | Mann Whitney | 0.23 |
| | Television | t-test | 0.748 | t-test | 0.297 | t-test | 0.163 | Mann Whitney | 0.442 |
| | PCA index | Spearman | 0.031 | Spearman | 0.019 | Spearman | 0.177 | Spearman | 0.177 |
| | additive index | Spearman | 0.228 | <i>Spearman</i> | <i>0.067</i> | Spearman | 0.259 | Spearman | 0.259 |
| Economic stability | Job loss | t-test | 0.986 | t-test | 0.94 | t-test | 0.851 | Mann Whitney | 0.762 |
| | Sale of property | t-test | 0.309 | t-test | 0.129 | t-test | 0.131 | Mann Whitney | 0.268 |
| | Pawning of property | t-test | 0.991 | t-test | 0.386 | <i>t-test</i> | <i>0.078</i> | Mann Whitney | 0.189 |
| | PCA index | regression | 0.82 | Pearson | 0.235 | Spearman | 0.139 | Spearman | 0.139 |
| | additive index | regression | 0.697 | Spearman | 0.464 | Spearman | 0.359 | Spearman | 0.359 |
| Economic wealth | PCA index | Spearman | 0.16 | Spearman | 0.064 | Spearman | 0.155 | Spearman | 0.115 |
| | additive index | Spearman | 0.205 | Pearson | 0.469 | Pearson | 0.41 | Spearman | 0.41 |
| Family stability | Serious illness | t-test | 0.998 | t-test | 0.988 | t-test | 0.959 | Mann Whitney | 0.652 |
| | Death | t-test | 0.856 | t-test | 0.567 | t-test | 0.176 | Mann Whitney | 0.108 |
| | Divorce | t-test | 0.244 | t-test | 0.294 | t-test | 0.692 | Mann Whitney | 0.599 |
| | Migration | t-test | 0.687 | t-test | 0.15 | t-test | 0.043 | Mann Whitney | 0.024 |
| | Other problems | t-test | 0.278 | t-test | 0.238 | t-test | 0.497 | Mann Whitney | 0.737 |
| | PCA index | Spearman | 0.832 | Spearman | 0.723 | Spearman | 0.331 | Spearman | 0.331 |
| | additive index | Spearman | 0.972 | Pearson | 0.842 | Pearson | 0.632 | Spearman | 0.632 |

Appendices

Table D.2 Bivariate tests and significance of maternal childhood living condition variables versus linear growth measures

| Index | Variable | Height | | Estimated leg length | | Sitting height ratio (square root) | | Sitting height ratio | |
|---------------|--------------------------------|---------------|--------------|----------------------|--------------|------------------------------------|--------------|----------------------|--------------|
| | | Test | p | Test | p | Test | p | Test | p |
| Housing | Flush toilet | t-test | 0.177 | t-test | 0.293 | t-test | 0.843 | Mann Whitney | 0.933 |
| | Clean water | T-test | 0.536 | T-test | 0.485 | T-test | 0.71 | Mann Whitney | 0.69 |
| | Electric Light | t-test | 0.166 | t-test | 0.313 | t-test | 0.998 | Mann Whitney | 0.722 |
| | Housing materials | regression | 0.647 | regression | 0.674 | regression | 0.883 | Spearman | 0.833 |
| | PCA index | Spearman | 0.16 | Spearman | 0.256 | Spearman | 0.792 | Spearman | 0.792 |
| | additive index | Spearman | 0.472 | Spearman | 0.331 | Spearman | 0.802 | Spearman | 0.802 |
| Modernisation | City dwelt | t-test | 0.422 | t-test | 0.213 | t-test | 0.27 | Mann Whitney | 0.523 |
| | Sugar | | | | | | | | |
| | sweetened beverage consumption | ANOVA | 0.556 | ANOVA | 0.167 | ANOVA | 0.133 | Krusky Wallace | 0.105 |
| | Packaged food consumption | ANOVA | 0.86 | ANOVA | 0.352 | ANOVA | 0.107 | Krusky Wallace | 0.306 |
| Demography | additive index | Spearman | 0.240 | Spearman | 0.101 | Spearman | 0.126 | Spearman | 0.126 |
| | Birth decade | ANOVA | 0.01 | ANOVA | 0.05 | ANOVA | 0.859 | Kruskal Wallace | 0.826 |
| | Birth year | regression | 0.629 | regression | 0.554 | regression | 0.591 | Spearman | 0.782 |
| | Birth decade: 1960-1970 | t-test | 0.11 | t-test | 0.595 | t-test | 0.81 | t-test | 0.818 |
| | Birth decade: 1981-1987 | t-test | 0.012 | t-test | 0.127 | t-test | 0.726 | t-test | 0.731 |
| | Number of siblings | ANOVA | 0.859 | ANOVA | 0.055 | ANOVA | 0.068 | Kruskal Wallace | 0.117 |
| | | regression | 0.45 | regression | 0.149 | regression | 0.113 | Spearman | 0.086 |
| | Birth order | t-test | 0.117 | t-test | 0.048 | t-test | 0.129 | Mann Whitney | 0.107 |
| | | regression | 0.823 | regression | 0.275 | regression | 0.028 | Spearman | 0.035 |
| | Siblings died | t-test | 0.087 | t-test | 0.387 | t-test | 0.536 | Mann Whitney | 0.394 |
| | PCA index | Spearman | 0.431 | Spearman | 0.102 | Spearman | 0.072 | <i>Spearman</i> | 0.072 |

Appendices

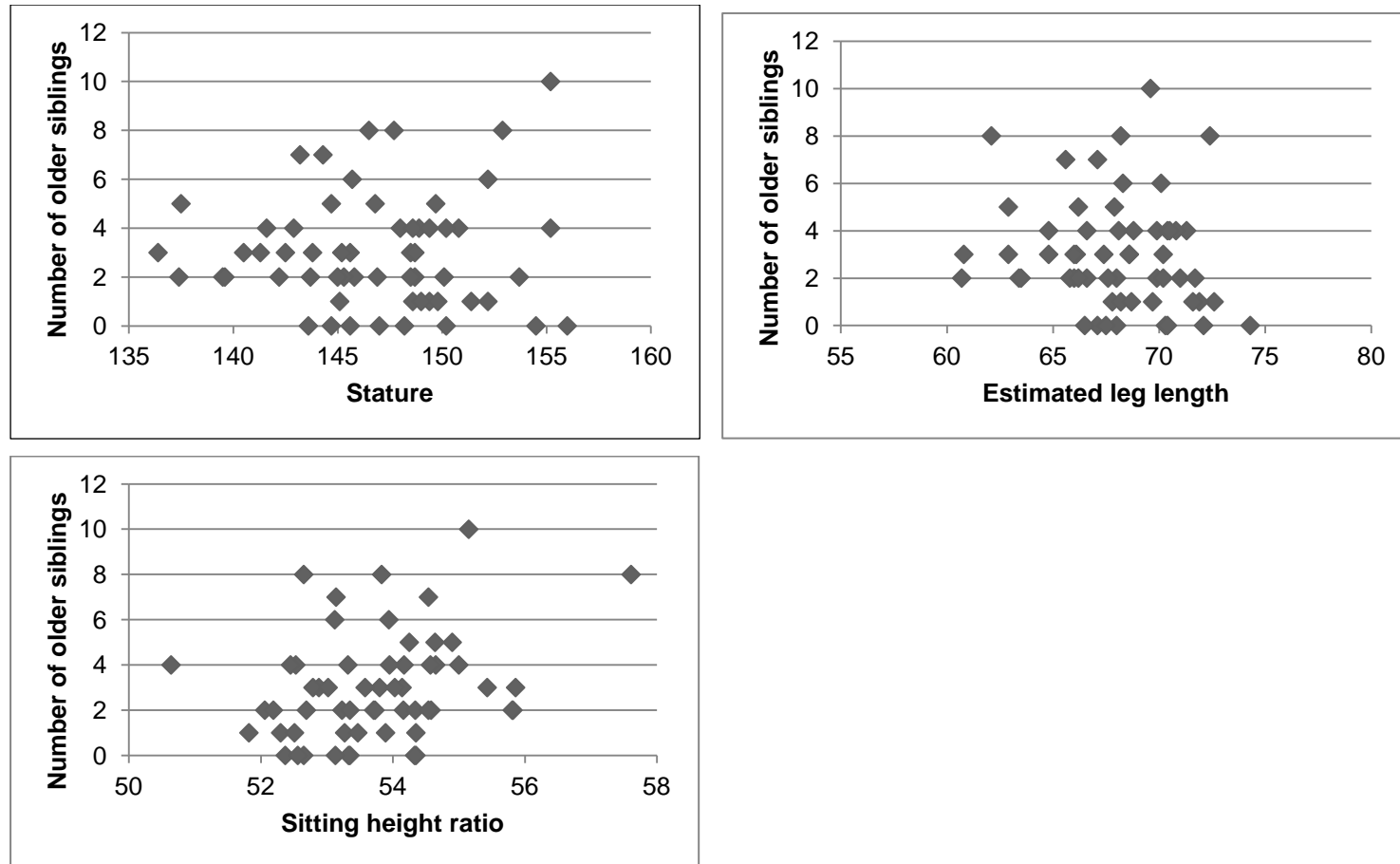


Figure D.1 Scatter-plots of number of older siblings versus measures of linear growth.

Appendices

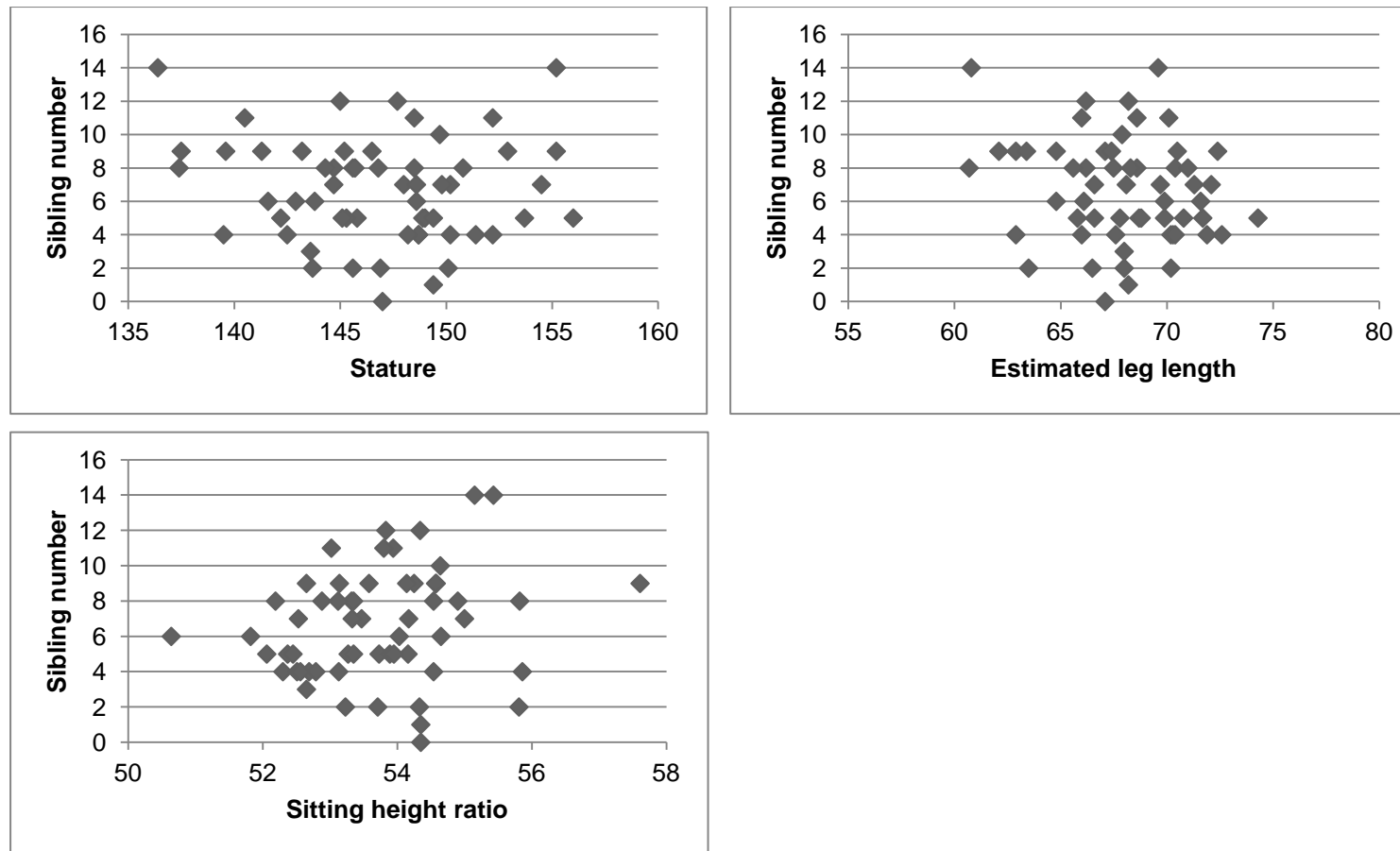


Figure D.2 Scatter-plots of sibling number versus measures of linear growth

E. Published and in press articles

1. Published articles

AMERICAN JOURNAL OF HUMAN BIOLOGY 23:426–428 (2011)

Short Report

Logistics of Using the Actiheart Physical Activity Monitors in Urban Mexico Among 7- to 9-Year-Old Children

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ABSTRACT Logistics of using new measurement devices are important to understand when developing protocols. This paper discusses the logistics of using Actiheart physical activity monitors on children in an urban, tropical environment in a developing country. Actiheart monitoring of 36 children aged 7–9 years old was undertaken for 7 days in the city of Mérida, Yucatán, Mexico. The Actiheart proved fragile for children and difficult to mend in the field. The excessive sweating due to the tropical climate caused poor adherence of the electrode pads, requiring a pad change mid-way through and extra pads to be provided. Also extra time was needed to be allotted for increased instructions to participants and their mothers and for individual calibration. When collecting objectively measured physical activity data under harsh conditions, the protocol must accommodate local conditions and device limitations and allow increased time with participants to obtain good quality data. *Am. J. Hum. Biol.* 23:426–428, 2011. © 2011 Wiley-Liss, Inc.

Researchers need to be informed about the feasibility and logistics of using measurement devices to perform the most efficient research possible. This short report discusses the logistics of using the Actiheart to estimate levels of physical activity and energy expenditure, under free living conditions, among children in urban Mexico. The aim is to inform other researchers planning to use the device in similar field settings of some of the challenges our research team has found, particularly among children in an urban tropical environment in a developing country.

Physical activity patterns are changing worldwide and the developing world is lacking the physical activity data needed to fully understand their activity patterns and effectively combat the rise in overweight and obesity (Satia, 2010). Objectively measured physical activity is more accurate than self-reported data (Westertorp, 2009). The Actiheart provides a very high quality method of estimating physical activity in humans. It is a combined movement sensor and heart rate monitor (Brage et al., 2004), and is composed of two sensors connected by a short wire, which is clamped to the ECG electrode pads placed on the chest of the participants. It combines a uniaxial accelerometer with a heart rate monitor to provide a more accurate estimation of physical activity levels and energy expenditure than either method individually (Barreira et al., 2009; Corder et al., 2005). The Actiheart has been validated in free living conditions in adults in developing country (Assah et al., 2010) and Western European children (Corder et al., 2007; de Bock et al., 2010). These validation studies show that the Actiheart is an appropriate tool for the assessment of physical activity in free-living populations.

took place over 1 week in the tropical, urban environment of Mérida, Yucatán, Mexico, during the dry and rainy seasons (April to July) of 2010. Ethical clearance was obtained from Loughborough University and the Bioethical Committee of Cinvestav in Mexico. Informed consent was obtained from each mother and assent obtained from each child.

Over the course of data collection, complete 5-day physical activity data were successfully collected for 20 of the 36 children. The average total energy expenditure was 1975 kcal/day (288 SD) with no difference between weekdays and weekends or sexes. For the 16 cases with incomplete data: in 10 cases, Actiheart fell off for substantial periods; and in six cases, heart rate data were not consistently recorded. Most of the Actiheart fell off before the protocol was amended to include greater information and instructions to the mothers. Accelerometry data was collected consistently when the Actiheart was worn. The unreliable heart rate data was due to poor adherence of the electrode pads to the skin. Also, six of 11 Actiheart broke and were not able to be fixed during fieldwork. The wires were disconnected in two devices during shirtless rough play, and the lids detached in four devices. This is higher than the failure rate of eight of 24 while monitoring 13-year olds for 5 days in Brazil reported by Reichert et al., (2009). The lid detachments did not vary with age, sex, physical activity, or SES of the child. Two Actiheart were shipped to the U.K. for repair and were confiscated

METHODS AND RESULTS

The researchers used 11 Actiheart to monitor 36 children (18 male) aged 7 to 9 years of low SES; for descriptives, see Table 1. The monitoring of each child

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TABLE 1. Descriptives statistics for urban Maya children ages 7 to 9 years

| | Girls | Boys | Total |
|---|---------------------------|---------------------------|----------------|
| | Mean (SD) | Mean (SD) | Mean (SD) |
| N | 18 | 18 | 36 |
| Height (cm) | 122.12 (7.86) | 120.28 (5.79) | 121.16 (6.75) |
| Height-for-age z-score ^b | -1.20 (0.89) | -1.31 (0.64) | -1.15 (0.86) |
| Weight (kg) | 29.38 (8.72) | 25.15 (4.38) | 27.04 (6.91) |
| Weight-for-age z-score ^b | -0.05 (1.00) ^a | -0.71 (0.65) ^a | -0.45 (0.88) |
| Body mass index (kg/ht ²) | 19.38 (4.18) | 17.25 (2.14) | 18.20 (3.34) |
| Body mass index for-age z-score(kg/ht ²) ^b | 0.88 (1.13) | 0.50 (0.81) | 0.56 (0.95) |
| Total energy expenditure (kcal) ^c | 1987.1 (395.2) | 1986.0 (282.2) | 1986.5 (332.6) |

^aSignificant differences between the sexes using an independent *t*-test, *P* < 0.05.

^bSex- and age-specific z-scores calculated using Frisancho's Comprehensive reference (2008).

^cTotal energy expenditure calculations made using the Actiheart equations for children using group reference data.

by Mexican customs who demanded three times the cost of an Actiheart for their release.

DISCUSSION

The Actiheart is relatively fragile for long term, free living monitoring of children. Two of the children disconnected the wires of their Actihearts while playing. Only the manufacturer was able to reconnect the wire and the monitor, due to the nature of the connection, i.e., clamped not soldered. The two could have been reconnected by soldering in the field, which would have resulted in permanent damage to the Actiheart. Therefore, it was necessary to ship the Actihearts back to the UK. Import laws caused customs delays in the U.K. and Mexico, so the broken devices were unavailable for the rest of fieldwork. Rough playing of the children, especially without shirts, can lead to wire disconnection.

Not all of the problems were associated with the children. Four lids detached exposing the electronics due to the high humidity, which reduced the adhesiveness of the glue. Reattachment had to occur in the U.K. The tropical climate also caused poor adherence of the electrode pads to the skin. The Actihearts were developed in the U.K., where extremely high temperatures are unusual. Our fieldwork was conducted in Yucatán, Mexico, where the average temperature was 33.0°C and with 60% humidity in the dry season, in a poor population without access to air conditioning. Thus, the levels of sweating were very high with slow moisture evaporation. The electrode pads did not stay reliably adhered to the chest for more than 3 days. The lack of long-term adherence was also encountered when using Actihearts in adults in sub-Saharan African (Assah et al., 2010). Therefore, the protocol was amended for researchers to provide the mothers with extra pads and give revamped instructions on how to attach them. Also, the researchers changed the pads midway through the week long monitoring. Approximately 25% of the pads required changing outside of the researchers' visits. Therefore, a minimum of six pads were required for each participant for the week long monitoring. Even when the pads were attached to the chest, the Actiheart did not always maintain close enough contact with the skin for constant heart rate monitoring. When this occurred for short periods (<5 min), the software was able to perform interpolation to estimate the heart rate of the lost period (CamNtech, 2009). This issue was minimized by the frequent changing of electrode pads and increased instructions to the mothers. This protocol change decreased the amount of lost data substantially. Overall, ECG electrode

pads do not stay consistently adhered in free living conditions in hot climates. Researchers working in such environments should budget for extra electrode pads and have a protocol that includes multiple participant visits.

The children were too young to follow the instructions given at the beginning of the monitoring period. Gaining access to mothers to provide instructions on the day the children started monitoring was challenging but often possible. Actihearts were generally placed on the children at school, so in these cases, mothers either came to the school or the fieldworkers went to the child's house to find the mother or caregiver. The mothers were provided with a package containing an instruction sheet and extra electrode pads. The instruction sheet included what to do if the Actiheart or a pad came off, how to attach the Actiheart to the pad and the researchers' phone numbers. At this time, the children were asked not to play rough team sports, to prevent damage to the Actiheart. This request to avoid these behaviors could have changed activity patterns, leading to a less accurate estimation of habitual physical activity in the children. After these instructions were added to the protocol, no more wires were disconnected. Giving instructions to each mother increased the time requirements on both mothers and fieldworkers but also greatly increased the quality of the data.

Physical activity measurement in children is challenging, particularly when the measurement device is relatively fragile and worn continuously in free-living conditions. In our sample, these problems were compounded by the climate. The lack of adherence of the electrode pads in tropical climates will occur no matter the age of the participant but is more likely to be quickly fixed by an adult than a child. Therefore, the data quality is likely to be higher in adults. A factor that must be considered that is inherent to objective heart rate monitoring is individual calibration. Interindividual variations in heart rates are large and must be controlled for effective physical activity estimations (Westertorp 2009). Reference curves can be used but increase the error of estimation (*ibid*). For this study, individual calibration was done using the walking exercise test and 10–15 min was required per test in this study. The Actiheart software allows multiple participants to perform the calibration test simultaneously, thereby, reducing the overall time requirements of individual calibration. Two children were able to do the calibration simultaneously quite easily. Individual calibration is important for accurate results and is easily done, and the time involved must be considered in logistical plans.

CONCLUSION

The use of the Actiheart for long-term monitoring in free-living children is logistically challenging and time consuming. The difficulties in measuring physical activity in children were compounded when using them in poor communities without air conditioning in hot climates. On the basis of our experience, we recommend the following: (1) the Actiheart needs to be more durable or more easily repaired outside of the UK and (2) new ECG electrode pads are needed that can reliably adhere under conditions of consistent sweating before Actiheart can be widely and easily used in free-living populations.

These logistical challenges do not eliminate the possibility of using the Actiheart in such environments but should be considered during the planning of field studies. The Actiheart provides high quality data and can be used under harsh conditions with the proper protocols, though breakages should be expected.

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Literature Cited

- Assah FK, Ekelund U, Brage S, Wright A, MBanya JC, Wareham NJ. 2010. Accuracy and validity of a combined heart rate and motion sensor for the measurement of free-living physical activity energy expenditure in adults in Cameroon. *Int J Epidemiol*. Published ahead of print: DOI: 10.1093/ije/dyq098.
- Barreira TV, Kang M, Caputo JL, Farley RS, Renfrow MS. 2009. Validation of the Actiheart monitor for the measurement of physical activity. *Int J Exer Sci* 2:60–71.
- Brage S, N Brage, PW Franks, U Ekelund, NJ Wareham. 2005. Reliability and validity of the combined heart rate and movement sensor Actiheart. *Eur J Clin Nutr* 59:561–570.
- CamNtech. 2009. The Actiheart User Manual. Issue version: 4.0.34. Cambridge: CamNtech Ltd.
- Corder K, Brage S, Mattocks C, Ness A, Riddoch C, Wareham NJ, Ekelund U. 2007. Comparison of two methods to assess PAEE during six activities in children. *Med Sci Sport Exer* 39:2180–2188.
- Corder K, Brage S, Wareham NJ, Ekelund U. 2005. Comparison of PAEE from combined and separate heart rate and movement models in children. *Med Sci Sport Exer* 37:1761–1767.
- De Bock F, J Menze, S Becker, D Litaker, J Fischer, I Seidel. 2010. Combining accelerometry and heart rate for assessing preschoolers' physical activity. *Med Sci Sport Exer* 42:2237–2243.
- Fossati Reichert F, Batista Menezes AM, Wells JC, Ekelund U, Machado Rodrigues F, Curi Hallal P. 2009. A methodological model for collecting high-quality data on physical activity in developing settings—the experience of the 1993 Pelotas (Brazil) Birth Cohort Study. *J Phys Activ Health* 6:360–366.
- 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. 335 p.
- Satia JA. 2010. Dietary acculturation and the nutrition transition: an overview. *Appl Physiol Nutr Metab* 35:219–223.
- Westerterp KR. 2009. Assessment of physical activity: a critical appraisal. *Eur J Appl Physiol* 105:823–828.

Original Research Article

How Useful Is BMI in Predicting Adiposity Indicators in a Sample of Maya Children and Women with High Levels of Stunting?

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Objectives: Body mass index (BMI) is used frequently to estimate adiposity levels in children and adults. However, the applicability of BMI to populations with high levels of stunting has been questioned. Stunted people can have disproportionately short legs, which may increase BMI without increasing body fat because of the relatively larger trunk compared with the legs.

Methods: A sample of 57 urban Maya schoolchildren, aged 7–9 years (31 boys), and 53 adult women underwent anthropometric assessments and bioelectrical impedance analysis. Multiple linear regression was performed to determine whether the ability of BMI to predict adiposity indicators is altered by stunting and sitting height ratio (SHR). The adiposity indicators were waist circumference, sum of skinfolds, upper arm muscle area, upper arm fat area, and arm fat index.

Results: BMI was the strongest predictor of all adiposity indicators and in most cases, explained more of the variance in adiposity of Maya children than Maya women. Abdominal adiposity was better predicted by BMI than peripheral adiposity in Maya women and Maya children. Stunting was significant in predicting adiposity in some models but never substantially changed the variance explained. SHR was never a significant predictor.

Conclusions: The relationship between BMI and adiposity indicators is not changed by stunting status or body proportions in this short population of urban Maya children and women. BMI can be used as an indicator of adiposity for these children but not the women. It is recommended that BMI is used in conjunction with other estimates of body composition. *Am. J. Hum. Biol.* 23:780–789, 2011. © 2011 Wiley Periodicals, Inc.

Linear growth is determined by a combination of genetics (Aulchenko et al., 2009; Bogin, 1999) and the quality of the environment experienced by the individual (Bogin, 1999). Stunting (very low height-for-age), due to chronically poor conditions, is very common in much of the developing world (Garrett and Ruel, 2005; Van de Poel et al., 2008) including urban centers in Latin America (Van de Poel et al., 2007). Stunting is widespread among indigenous groups (Barquera et al., 2007) and the poor (Malina et al., 2008; Van de Poel et al., 2008). As nutrition transition occurs (Popkin, 1996), stunting rates are declining in Latin America (Malina et al., 2009; Rivera et al., 2004). However, it will be several decades before the stunting rates will be considered acceptable, at a prevalence of less than 5% of the population (Rivera et al., 2004). In this article, adult short stature and childhood stunting will both be referred to as stunting for consistency of presentation. Adult short stature is caused by the same factors as childhood stunting and is in fact the end result of childhood stunting (Bogin, 1999).

Childhood stunting often leads to very short stature in adulthood (Stein et al., 2010). Adults and adolescents with short stature have been shown to have increased mortality rates (Song and Sung, 2008), cardiovascular risk factors (Flores et al., 2007; Kruger et al., 2004) and lower economic productivity (Case and Paxson, 2008). Also short adults are at an increased risk of obesity (Hoffman et al., 2000b, c; Leonard et al., 2009; Lopez-Alvarenga et al., 2003; Martins et al., 2004). The dual burden of simultaneous short stature and obesity is increasingly common in

developing countries individuals (Doak et al., 2005, 2000). With limited resources available, these countries need an efficient and cost effective method of assessing over and undernutrition to be able to monitor and diagnose dual burdened populations.

Body mass index (BMI = weight in kg/stature in meters²) is often used to assess nutritional status and screen for obesity. Although BMI is not intended to distinguish individuals who have excess adipose tissue (Ellis, 2001), in practice it has become the most widely used indicator of overweight, both in clinical settings and in research (Burkhauser and Cawley, 2008; Hall and Cole, 2006; Popkin and Doak, 1998). BMI is very easy to measure, with minimal training, requiring only stature and weight measurements (Burkhauser and Cawley, 2008; Ellis, 2001) and it has been shown to correlate fairly well with total body adiposity (Cameron et al., 2009; Ellis, 2001). It is useful for longitudinal measurements of adults as adult stature is constant and therefore changes in BMI may be considered to be mostly due to body fat (Ellis, 2001). BMI is recommended mainly for large, popu-

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lation-based studies (Burkhauser and Cawley, 2008; Ellis et al., 1999).

The main limitation of BMI is the inability of the tool to differentiate between fat and fat-free mass. Individuals, both children and adults, with normal or overweight BMIs may have a percent body fat (%BF) that is over the cut off for overfat (Ellis, 2001; Frankenfield et al., 2001). People with high fat-free mass, such as some athletes and those engaged in physically demanding occupations, may have a high BMI but a normal or low %BF (Burkhauser and Cawley, 2008). Therefore, when used as a screening tool for obesity, BMI is not precise because it has a low sensitivity (ability to classify overfat individuals) (Ellis, 2001). The problems with using BMI as a screening tool may be especially true in non-Western populations that may have ratios of fat mass to fat free mass (Norgan, 1994a) or fat patterning (Fuke et al., 2007; Gabrielsson et al., 2003; Lau et al., 2005; Norgan, 1994a) different from that of the majority of people living in industrialized nations of Europe and North America.

Nutritionally stunted populations pose a theoretical problem to the use of BMI. First of all, as stature is squared in the denominator of the BMI equation, a reduction in stature will lead to an exaggerated increase in BMI, the effect of which will be greater in children than adults (Cameron et al., 2005). Secondly, body proportions may be altered in stunted individuals due to the body's growth patterns in childhood. The legs are the fastest growing segment of the body before puberty (Leitch, 1951). If the chronically poor conditions that caused the stunting occurs during childhood, the legs may be disproportionately short (Gunnell et al., 1998). Short legs and a long torso will result in a higher body weight due to the size differences between the limbs and torso (Bogin and Beydoun, 2007; Norgan, 1994b). The higher weight will result in a higher BMI, without any change to body composition. This may render BMI inappropriate for use in stunted individuals, especially in those with disproportionately long or short legs.

Norgan (1994a) found that skinfold thickness was greater than expected from the BMI of Australian Aborigines. Australian Aborigines have relatively long legs for their height, which results in a low sitting height ratio ($SHR = [\text{sitting height/stature}] \times 100$) and, it seems, a low BMI. In a sample of 349 adult Aboriginal men and women Norgan found that, "...4% of the individual men and 14% of the individual women had [BMI] values less than 16 kg/m^2 , a value regarded as indicating severe chronic energy deficiency" (p.229). Skinfold measurement, however, indicated no such deficiency. In a related analysis of more than 18,000 nonwestern adults, Norgan (1994b) found that linear regression of BMI on SHR resulted in regression coefficients ($b \pm \text{standard error}$) of 0.78 ± 0.16 ($t = 4.8$) in men and 1.19 ± 0.22 ($t = 5.3$) in women. These regression coefficients compare with a predicted change of 0.9 kg/m^2 per 0.01 difference in SHR using a modeling approach. The predictive model proves inaccurate for both men, in which the model overestimates the change, and women, in which the model underestimates the change. Norgan concludes that the wide variation in these relationships between the sexes and between populations precludes a simple adjustment for SHR on BMI. Norgan also points out that an accurate interpretation of BMI requires additional anthropometric measurements to just height and weight. Norgan's find-

ings have been confirmed other studies. In a sample of 120 Chinese and Dutch adults matched for age, sex, and BMI, Deurenberg et al. (1999) found that BMI varied according to SHR. Relatively shorter legs were associated with greater BMI. Two analyses of the United States Third National Health and Nutrition Survey of 1988–1994 (NHANES III) find that adults with relatively shorter legs as estimated by the SHR have greater fatness (Asao et al., 2006; Bogin and Beydoun, 2007). Another study indicates that the cause of the relationship may be greater gluteal-femoral fatness artificially increasing sitting height and as a consequence artificially decreasing the estimate of leg length relative to total stature (Bogin and Varela-Silva, 2008). Lara-Esqueda et al. (2004) showed that BMI did not work as well in detecting cardiovascular disease risk factors in stunted versus nonstunted Mexican adults.

All of these studies indicate that therefore, a high BMI in very short populations may not be useful in assessing nutritional status. However, it is not clear if these problems with BMI outweigh its usefulness as an indicator of risk for later negative health outcomes in all populations, including stunted populations.

The Maya are indigenous to Central America, including the Yucatan Peninsula. They are a poor, marginalized group of society, which also places them at risk for negative health outcomes. The Mayans tend to be very short, with high levels of stunting (Bogin et al., 1992; Crooks, 1994; Jenkins, 1981). Using data collected in 2007 from the same neighborhoods of Merida, Mexico sampled in this study, Varela-Silva et al. (2009) found that 22% of 4–6-year-old Mayan children were stunted. Stunting was even more common in their mothers, with 69% being under 150 cm tall (Varela-Silva et al., 2009). Also, the Mexican Maya are adopting behaviors (Leatherman and Goodman, 2005; Leatherman et al., 2000) which have been linked to increased obesity and other chronic disease (Leatherman et al., 2010). These characteristics make the Maya a suitable population in which to examine the usefulness of BMI as a predictor of obesity.

Accordingly, this study aims to determine whether BMI is an appropriate estimate of adiposity in stunted and non-stunted urban Maya women and their 7- to 9-year-old children. This study also aims to determine if the association between BMI and adiposity indicators is influenced by body proportions. The age range of 7–9 years in children is useful to examine in relation to the effects of stature on adiposity measures because it is a period of stable growth before puberty (Cameron, 2002). Though it must be noted, it is too old to reverse stunting (Walker et al., 2007) and its effects (Hoffman et al., 2000a; Martins et al., 2004).

MATERIALS AND METHODS

Sample

A cross-sectional survey was undertaken of 58 urban Maya mothers and their children (31 boys), aged 7 to 9 years old, living in Merida, Yucatan, Mexico between March and July of 2010.

Recruitment

Schools located in *colonias* (neighborhoods) in the southern part of Merida, Mexico, that had a high propor-

tion of Maya students were approached. School directors that agreed to participate provided the school lists with the children's full names. From these lists, Maya children were identified as those with two Maya surnames, one from both the father and the mother. The mothers were then invited to information sessions at their children's schools where the study was explained and information sheets were provided. The mothers and one of their children aged 7 to 9 years were recruited. The survey involved administering a semistructured interview to the mother, anthropometric and body composition measurements of the mother and child, and monitoring the physical activity levels of the child.

Pubertal status of the girls was also assessed by maternal interview. Pubertal status of the boys was not assessed due to the later development of boys. Only two of the 26 girls were determined to be pubertal, all analyses were performed with and without these two individuals. The results did not differ between the two separate analyses and therefore the findings from the full sample are shown to maximize the sample size available for analysis.

Written informed consent was obtained from the mothers and verbal assent from the children. Ethical clearance was obtained from the Loughborough University Ethics Committee in the U.K. and the Bioethics Committee of Human Studies of Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional (Cinvestav) in Mexico.

Measurements

Mothers and children underwent anthropometric measurement (Lohman et al., 1988), which included stature, sitting height, weight, waist circumference (WC), mid-arm circumference, and skinfolds (triceps for both mother and children and subscapular, for children only). The sum of two skinfolds (triceps and subscapular) (2SF) was calculated for children (Frisancho, 2008). Body mass index (BMI) was calculated by dividing weight in kilograms by stature in meters squared. Sitting height ratio (SHR) was calculated by dividing sitting height by stature and multiplying by 100. Arm fat index (AFI) was calculated by dividing arm fat area (UFA) by mid-upper arm area (UMA) for an estimation of the fat percentage of the arm (Frisancho, 2008).

Body composition was measured for women and children using bioelectric impedance analysis (BIA) with a BioScan 916, Maltron International. Percent body fat (%BF) was calculated using the impedance and reactance values with equations for North American Indian children [Eq. (1)] (Lohman et al., 1999) and women [Eq. (2)] (Stolarczyk et al., 1994) from the southwest of the United States. North American Indian equations were used because no equations specific to the Maya have been published. Maternal fat free mass as calculated by Eq. (2) was converted to %BF. The equation for women [Eq. (2)] had a reported r^2 of 0.803 and a standard error of estimate (SEE) of 2.38 kg for fat mass (Stolarczyk et al., 1994). The equation for children [Eq. (1)] has a SEE of 3.4% body fat and no r^2 was reported (Lohman et al., 1999).

Equation 1: Child's percentage body fat = $-0.49 \text{ age} + 0.51 \text{ sex} + 0.44 \text{ weight} + 1.55 \text{ triceps skinfold} + 0.15 \text{ sub-$

scapular skinfold $+ 0.54 (\text{stature}^2/\text{resistance}) + 0.13 \text{ reactance} - 0.04 \text{ triceps skinfold} \times \text{stature}^2/\text{resistance} - 10.91$

Equation 2: Women's fat free mass (kg) = $0.001254 (\text{stature}^2) - 0.04904 \text{ resistance} + 0.155 \text{ weight} + 0.1417 \text{ reactance} - 0.0833 \text{ age} + 20.05$

Definitions: Sex coded 1 for girls, 0 for boys. Weight is in kg. Skinfold thicknesses are in mm.

Resistance and reactance are in ohms. Stature is in m for Equation 1 and centimeters for Equation 2.

Frisancho's Comprehensive sex- and age-specific reference charts were used to calculate z-scores and classify children as stunted (2008). This reference was chosen as it was created using NHANES III data from the USA, which includes Mexican-Americans. Women were classified as stunted if their stature was below 150 cm, the nearest whole centimeter to the 5th percentile for adult women from Frisancho's Comprehensive reference (Frisancho, 2008). This cut off has been used previously in Mexican women (Lara-Esqueda et al., 2004; Lopez-Alvarenga et al., 2003; Varela-Silva et al., 2009). Children were classified as being stunted if their height-for-age was below the 5th percentile. For children, age- and sex-specific z-scores were calculated for WC, BMI, AFI, and 2SF. No reference values were available for children's %BF as the references begin at age 12.

Statistical analysis

Normality of all variables was checked. Descriptive statistics were performed and Pearson's correlations, independent *t*-tests, and Pearson's chi square were used for inferential analysis. A simple linear regression was also performed with BMI as the dependent variable and SHR as the predictor variable for women and children separately in order to be able to compare these findings with previous studies.

Multiple linear regressions were performed using the enter method with adiposity indicators as the dependent variables, which were determined based on theory related to the hypothesized effects of the predictor variables in their association with the outcome measure. The independent variables were BMI, stunting, and SHR. To determine whether BMI interacted with stunting or SHR to influence the dependent variable, an interaction between each was included. For model building purposes and to allow for potential moderating effects of other variables, if an interaction had a *P* value below 0.10 in the simple model, it was included in the final model. No more than four predictor variables were used in a single regression model, giving these models the power to detect large, but not small to medium effect sizes due to the size of the sample (Cohen, 1992).

The dependent variables for the children's models were %BF and the z-scores of WC, 2SF, UMA, UFA, and AFI. All continuous variables in the children's regressions were z-scores, except for %BF (Frisancho, 2008). Therefore, in the children's %BF model, age and sex were also entered into the model because %BF was not already standardized for age and sex. The dependent variables for the women's models were WC, %BF, UMA, UFA, and AFI.

Stature is also used to calculate both BMI and SHR, which could potentially lead to collinearity. However, the purpose of squaring stature in the denominator of the

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TABLE 1. Children's descriptive statistics, mean (SD)^{a,b}

| | Boys | Girls | Stunted | Nonstunted | All |
|--------------------------------------|---------------------------|----------------------------|----------------------------|----------------------------|---------------|
| N (%) | 31 (54.4) | 26 (45.6) | 18 (31.6) | 39 (68.4) | 57 (100) |
| Age | 8.23 (0.84) | 8.59 (0.72) | 8.33 (0.85) | 8.47 (0.78) | 8.43 (0.8) |
| Stature | 121.84 (5.95) | 122.54 (7.94) | 115.57 (5.72) [†] | 125.2 (4.99) ^{††} | 122.13 (6.82) |
| Stature z-score | -1.12 (0.86) | -1.20 (0.89) | -2.14 (0.47) [†] | -0.7 (0.58) ^{††} | -1.15 (0.87) |
| Weight | 25.76 (4.83) | 28.38 (7.92) | 22.51 (2.47) [†] | 29.00 (5.94) ^{††} | 26.87 (6.47) |
| Weight z-score | -0.64 (0.81) | -0.21 (0.93) | -1.09 (0.7) [†] | -0.14 (0.81) ^{††} | 0.44 (0.89) |
| BMI ^c | 17.24 (2.31) | 18.62 (3.70) | 16.74 (3.22) | 18.39 (2.91) | 17.83 (3.07) |
| BMI z-score | 0.48 (0.88) | 0.67 (1.03) | 0.14 (0.94) [†] | 0.76 (0.9) [†] | 0.56 (0.95) |
| Sitting height | 65.48 (2.69) | 65.75 (7.92) | 62.86 (3.23) [†] | 66.87 (2.73) ^{††} | 65.6 (3.43) |
| Sitting height z-score | -0.97 (0.80) | -0.51 (0.97) | -1.42 (0.74) [†] | -0.45 (0.81) ^{††} | 0.76 (0.9) |
| SHR ^d | 53.77 (1.28) | 53.68 (1.38) | 54.40 (1.41) [†] | 53.42 (1.15) ^{††} | 53.73 (1.31) |
| SHR z-score | 1.02 (0.74) | 1.26 (0.89) | 1.57 (0.87) [†] | 0.9 (0.72) ^{††} | 1.13 (0.82) |
| WC (cm) ^e | 58.51 (5.86) | 61.4 (9.6) | 55.98 (6.83) [†] | 61.60 (7.73) [†] | 59.77 (7.8) |
| WC z-score | 0.14 (0.70) [*] | 0.58 (0.84) [*] | -0.02 (0.66) [†] | 0.51 (0.8) [†] | 0.34 (0.79) |
| Triceps skinfolds (mm) | 12.58 (6.44) | 15.30 (6.14) | 12.03 (5.98) | 14.64 (6.49) | 13.75 (6.36) |
| Triceps skinfold z-score | 0.45 (0.88) | 0.75 (0.93) | 0.27 (0.86) | 0.73 (0.9) | 0.58 (0.91) |
| Subscapular skinfold (mm) | 9.14 (5.43) [*] | 12.65 (7.23) [*] | 8.71 (5.15) | 11.62 (6.89) | 10.62 (6.46) |
| Subscapular skinfold z-score | 0.61 (0.79) | 0.84 (0.92) | 0.35 (0.8) [†] | 0.89 (0.83) [†] | 0.72 (0.85) |
| Sum of 2 skinfolds (mm) ^f | 21.71 (11.58) | 27.42 (12.63) | 20.74 (10.82) | 26.00 (12.72) | 24.37 (12.31) |
| Sum of 2 skinfolds z-score | 0.33 (0.99) | 0.82 (0.84) | 0.25 (0.88) | 0.7 (0.95) | 0.56 (0.95) |
| Mid-arm circumference (cm) | 19.05 (2.34) [*] | 20.94 (4.16) [*] | 18.27 (2.89) [†] | 20.67 (3.38) [†] | 19.88 (3.38) |
| Mid-arm circumference z-score | -0.56 (0.94) | 0.74 (1.17) | -0.39 (1.01) | 0.23 (1.28) | 0.03 (1.23) |
| TUA (cm ²) ^g | 29.3 (7.27) [*] | 36.22 (15.28) [*] | 27.17 (9.59) [†] | 34.89 (12.36) [†] | 32.33 (11.96) |
| TUA z-score | -0.07 (0.92) | 0.46 (1.09) | -0.33 (0.84) [†] | 0.4 (1.03) [†] | 0.17 (1.02) |
| UMA (cm ²) ^h | 18.31 (3.59) | 21.47 (9.16) | 16.76 (4.68) [†] | 21.04 (7.34) [†] | 19.71 (6.79) |
| UMA z-score | -1.18 (1.01) | -0.58 (1.59) | -1.53 (1.00) [†] | -0.62 (1.37) [†] | -0.91 (1.33) |
| AFA (cm ²) ⁱ | 10.98 (6.36) | 14.74 (8.11) | 10.21 (6.52) | 13.84 (7.56) | 12.62 (7.35) |
| AFA z-score | 0.54 (1.23) | 0.68 (1.18) | 0.12 (1.07) [†] | 0.82 (1.2) [†] | 0.6 (1.2) |
| AFI (%) ^j | 35.55 (12.51) | 39.67 (10.25) | 35.66 (11.12) | 38.25 (11.91) | 37.34 (11.54) |
| AFI z-score | 0.91 (1.22) | 1.26 (1.37) | 0.79 (1.2) | 1.2 (1.33) | 1.07 (1.29) |
| %BF ^k | 26.54 (0.65) [*] | 30.24 (7.22) [*] | 25.09 (6.72) [†] | 29.68 (6.76) [†] | 28.23 (7.03) |

^aAll sex- and age-specific z-scores calculated using Frisancho's comprehensive reference (2008).^bStunted defined as height-for-age below the 5th percentile of sex-specific Frisancho's comprehensive reference curves.^cBMI, body mass index = (weight in kilograms/stature in metres²).^dSHR, sitting height ratio = (sitting height/stature) × 100.^eWC, waist circumference.^fThe skinfolds summed were triceps and subscapular.^gTUA, total upper arm area (Frisancho, 2008).^hUMA, upper arm muscle area (Frisancho, 2008).ⁱAFA, arm fat area (Frisancho, 2008).^jAFI, arm fat index, percent of the upper arm that is fat (Frisancho, 2008).^k%BF, percent body fat calculated from a bioelectric impedance equation specific to American Indian children (Lohman et al., 1999).^{*}Significant differences between stunted and nonstunted children found using an independent *t*-test, *P* < 0.01.[†]Significant differences between stunted and nonstunted children found using an independent *t*-test, *P* < 0.05.^{††}Significant differences between the sexes found using an independent *t*-test, *P* < 0.05.

BMI calculation is to remove the influence of stature and to obtain an overall measure of body size. Multicollinearity of the variables were checked and none excluded the tolerated values (VIF < 1.0) (Bowerman and O'Connell, 1990). Thus, it was deemed statistically as well as theoretically appropriate to use both BMI and SHR as predictors in the same regression model.

All analyses were done using SPSS v. 17.0. Significance was set a priori at *P* < 0.05.

RESULTS

One child and one woman were excluded due to biologically improbable sitting heights. Also, four mothers were excluded due to missing BIA data. The final sample sizes were 57 children (31 boys) and 53 women.

For the children, no significant difference in the prevalence of stunting between the sexes was found (*P* > 0.05). Overall, the children were quite small. Significant differences between the sexes were found for WC z-score, arm circumference, and %BF, with girls being larger and having more body fat than boys. The stunted children were consistently smaller overall than their nonstunted peers (Table 1).

The women were very short, with 75% being stunted, and had high levels of adiposity. However, no significant differences existed between the stunted and nonstunted mothers with respect to the adiposity indicators such as %BF, BMI, WC, AFI, etc. . . (Table 2).

The children's BMI and SHR were not significantly related (*R*² = 0.001) (Fig. 1a). However, the women's BMI and SHR were significantly related (*R*² = 0.102) (Fig. 1b).

For children, BMI was the largest contributor to each model, explaining between 31% (UMA) and 84% (WC) of the variance when significant (Tables 3 and 4). BMI significantly and positively predicted all adiposity indicators and was the only significant predictor for %BF, AFI, and 2SF. The variance explained by the models varied little with the inclusion of covariates other than BMI, including stunting and SHR. %BF had the largest change in variance explained with 0.5% from the BMI only model to the final model. Stunting was statistically significant in some models but it never explained more than 2% of the variance in the adiposity outcomes. The interaction between BMI and stunting significantly and negatively predicted WC. The plot of the interaction shows that nonstunted children have a stronger relationship between BMI and

TABLE 2. Mother's descriptive statistics, mean (SD)^a

| | Stunted | Nonstunted | All |
|-------------------------------------|---------------|---------------|---------------|
| N (%) | 40 (75.5) | 13 (24.5) | 53 (100) |
| Age | 33.74 (6.74) | 36.61 (4.16) | 34.44 (6.3) |
| Stature (cm) | 145.15 (3.73) | 152.66 (2.10) | 147.00 (4.7) |
| Weight (kg) | 61.81 (9.74) | 69.58 (9.28) | 63.72 (10.12) |
| BMI ^b | 29.32 (4.31) | 29.85 (3.84) | 29.45 (4.17) |
| Sitting height (cm) | 77.97 (2.30) | 81.32 (2.04) | 78.79 (2.65) |
| SHR ^c | 53.73 (1.10) | 53.26 (0.87) | 53.61 (1.06) |
| WC (cm) ^d | 87.98 (8.43) | 88.94 (9.2) | 88.22 (8.55) |
| Triceps skinfold (mm) | 28.42 (8.71) | 33.04 (7.05) | 29.55 (8.51) |
| Mid-arm circumference | 30.28 (3.64) | 31.62 (2.44) | 30.62 (3.42) |
| TUA (cm ²) ^e | 73.98 (15.57) | 80.26 (12.34) | 75.52 (16.56) |
| UMA (cm ²) ^f | 36.82 (8.50) | 36.19 (4.04) | 36.67 (7.62) |
| AFA (cm ²) ^g | 37.16 (13.68) | 44.07 (11.32) | 38.86 (13.38) |
| AFI ^h | 49.35 (10.39) | 54.24 (6.67) | 50.55 (9.79) |
| %BF ⁱ | 42.93 (4.19) | 40.49 (4.27) | 42.33 (4.30) |

^aAdult female stunting defined as stature below 150 cm.^bBMI, body mass index = (weight in kilograms/stature in metres²).^cSHR, sitting height ratio = (sitting height/stature) × 100.^dWC, waist circumference.^eTUA, total upper arm area (Frisancho, 2008).^fUMA, upper arm muscle area (Frisancho, 2008).^gAFA, arm fat area (Frisancho, 2008).^hAFI, arm fat index, percent of the upper arm that is fat (Frisancho, 2008).ⁱ%BF, percent body fat calculated from a bioelectric impedance equation specific to American Indian women (Stolarczyk et al., 1994).*Significant differences between the stunted and nonstunted mothers found using an independent *t*-test, *P* < 0.01.**Significant differences between the stunted and nonstunted mothers found using an independent *t*-test, *P* < 0.05.

WC (Fig. 2). SHR was not significantly associated with any adiposity indicator.

For women, BMI significantly predicted every adiposity indicator and was the largest contributor to each model, explaining between 13% and 78% of the variance (Tables 5 and 6). However, all of the women's models explained less of the variance in adiposity than the children's models. Covariates (BMI, stunting, and SHR) explained more of the variance in women than children. %BF had the largest change in variance explained with a 10% increase from the BMI only model to the final model. Being stunted significantly predicted a higher %BF. SHR did not impact any model though it did significantly interact with BMI in the simple %BF models, but was attenuated in the final models with the inclusion of stunting.

DISCUSSION

In children, BMI significantly predicted measures of abdominal (WC) and total body adiposity (%BF, 2SF) but not peripheral adiposity (AFA, AFI) and explained a high proportion of the variance for all significant variables. Stunting status did not modify the power of BMI to predict adiposity indicators. SHR neither significantly moderated nor mediated the effect of BMI on adiposity outcomes. This suggests that BMI is an appropriate tool to estimate total and central adiposity in this sample of 7–9-year-old children, regardless of SHR.

In women, BMI significantly predicted abdominal adiposity (WC) but not peripheral (AFA, AFI) or total body adiposity (%BF). Stunting independently predicted a higher %BF, but did not change the association between BMI and adiposity indicators in any regression model. SHR was neither significant nor altered the association between BMI and any adiposity indicator. BMI appears to be appropriate for use in these adult urban Maya women only to predict abdominal adiposity.

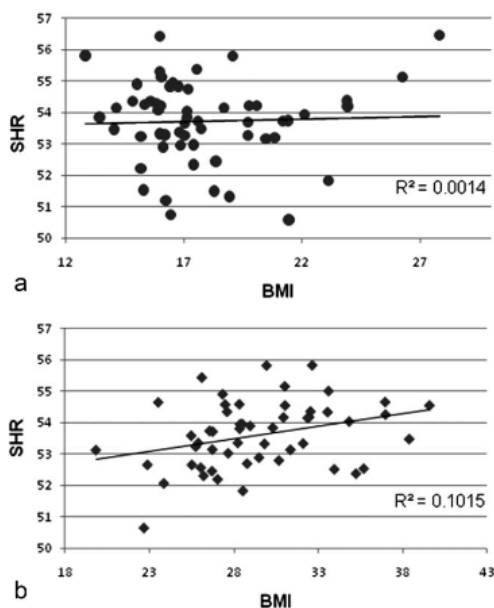


Fig. 1. Simple regression of children's (a) and women's (b) SHR on BMI. Children's raw data was used (not z-scores).

Maternal %BF was not well predicted by BMI as it only explained 30% of the variance. This sample of women falls in a range of BMIs (25–29.99) that has been previously shown to be the worst at classifying %BF (Ellis, 2001). Indeed, the %BF of these women was very high (mean = 42%) as measured by BIA. As such, it is not appropriate to use BMI alone to predict %BF in this sample of adult urban Maya women.

The relationship between BMI and %BF in the children is considerably stronger than in the mothers. BMI explained just over 80% of the variance in children's %BF. Similar studies have been done in other samples of children to determine the relationship between BMI and %BF. Ellis et al. (1999) compared BMI to dual X-ray absorptiometry (DXA) in children aged 3 to 18 years in the USA. They found the two estimates of body fat to be significantly correlated, with the relationship stronger in the girls ($r^2 = 0.70$) than boys ($r^2 = 0.34$).

Hoffman et al. (2006) performed a case control study of stunted and nonstunted children in the shantytowns of São Paulo, Brazil (Hoffman et al., 2006). They found that BMI significantly predicted %BF, as measured by DXA but had a much lower r^2 (0.125) than this study (0.807, Table 3, model 1). This large difference may be due, to a combination of measurement error in this study but also to the low levels of adiposity found by Hoffman. This study of Mayans used BIA and predictive equations validated on a group of North American Indians (Lohman et al., 1999; Stolarczyk et al., 1994). BIA and prediction equations have larger measurement error than the gold standard method of DXA (Going et al., 2006), which may have arti-

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TABLE 3. Estimates of child's body composition using multiple linear regression^a

| | | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---------------------------------|------------------------|----------------|--------|----------------|--------|----------------|--------|----------------|--------|
| | | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P |
| %BF ^b | Constant | 11.991 (4.412) | 0.009 | 12.201 (4.784) | 0.014 | 12.111 (4.708) | 0.013 | 12.745 (4.442) | 0.006 |
| | Age | 1.393 (0.528) | 0.001 | 1.363 (0.544) | 0.015 | 1.338 (0.554) | 0.019 | 1.272 (0.527) | 0.215 |
| | Sex ^c | 2.089 (0.839) | 0.016 | 2.300 (0.888) | 0.012 | 2.004 (0.858) | 0.023 | 2.338 (0.863) | 0.606 |
| | BMI ^d | 6.291 (0.437) | <0.001 | 6.769 (1.419) | <0.001 | 6.238 (0.454) | <0.001 | 5.988 (0.466) | <0.001 |
| | Stunted ^{e,f} | | | -1.214 (0.979) | 0.221 | | | -1.687 (1.032) | 0.108 |
| | BMI × Stunted | | | -0.494 (0.999) | 0.623 | | | | |
| | SHR ^g | | | | | 0.351 (0.538) | 0.517 | 0.751 (0.548) | 0.176 |
| | BMI × SHR | | | | | 0.185 (0.505) | 0.716 | | |
| R ² adj | | 0.807 | | 0.806 | | 0.802 | | 0.812 | |
| WC ^h | Constant | -0.090 (0.049) | 0.074 | -0.237 (0.099) | 0.020 | -0.105 (0.079) | 0.188 | -0.257 (0.114) | 0.029 |
| | BMI | 0.764 (0.045) | <0.001 | 1.036 (0.137) | <0.001 | 0.777 (0.046) | <0.001 | 1.039 (0.139) | <0.001 |
| | Stunted | | | -0.098 (0.095) | 0.309 | | | -0.113 (0.105) | 0.288 |
| | BMI × Stunted | | | -0.211 (0.097) | 0.035 | | | -0.215 (0.099) | 0.034 |
| | SHR | | | | | 0.010 (0.055) | 0.860 | 0.020 (0.056) | 0.722 |
| | BMI × SHR | | | | | -0.059 (0.050) | 0.241 | | |
| | R ² adj | 0.841 | | 0.846 | | 0.836 | | 0.844 | |
| | | | | | | | | | |
| Sum of 2 Skinfolts ⁱ | Constant | 0.083 (0.080) | 0.306 | -0.056 (0.167) | 0.738 | -0.038 (0.128) | 0.765 | -0.024 (0.127) | 0.852 |
| | BMI | 0.839 (0.093) | <0.001 | 1.029 (0.232) | <0.001 | 0.842 (0.075) | <0.001 | 0.836 (0.079) | <0.001 |
| | Stunted | | | 0.049 (0.160) | 0.761 | | | 0.008 (0.170) | 0.962 |
| | BMI × Stunted | | | -0.134 (0.164) | 0.417 | | | | |
| | SHR | | | | | 0.107 (0.089) | 0.233 | 0.093 (0.093) | 0.321 |
| | BMI × SHR | | | | | -0.039 (0.081) | 0.634 | | |
| | R ² adj | 0.702 | | 0.696 | | 0.699 | | 0.698 | |
| | | | | | | | | | |

^aAll variables except stunting and percent body fat are age- and sex-specific z-scores calculated from Frisancho's Comprehensive reference (2008).

^b%BF, percent body fat calculated from an bioelectric impedance equation specific to American Indian children (Lohman et al. 1999).

^cBoys were set as the reference.

^dBMI, body mass index.

^eStunting defined as height-for-age below the 5th percentile of Frisancho's Comprehensive reference (2008).

^fNonstunted was set as the reference.

^gSHR, sitting height ratio.

^hWC, waist circumference.

ⁱThe summed skinfolts were triceps and subscapular.

TABLE 4. Estimates of child's arm composition in multiple linear regression^a

| | | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|------------------|------------------------|----------------|--------|----------------|--------|----------------|--------|----------------|--------|
| | | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P |
| UMA ^b | Constant | -1.356 (0.170) | <0.001 | -1.328 (0.351) | <0.001 | -1.246 (0.276) | <0.001 | -1.182 (0.268) | <0.001 |
| | BMI ^c | 0.794 (0.155) | <0.001 | 0.984 (0.488) | 0.049 | 0.794 (0.162) | <0.001 | 0.723 (0.166) | <0.001 |
| | Stunted ^{d,e} | | | -0.499 (0.337) | 0.144 | | | -0.468 (0.361) | 0.200 |
| | BMI × Stunted | | | -0.195 (0.346) | 0.575 | | | | |
| | SHR ^f | | | | | -0.098 (0.191) | 0.611 | 0.013 (0.198) | 0.948 |
| | BMI × SHR | | | | | 0.025 (0.175) | 0.899 | | |
| | R ² adj | 0.311 | | 0.314 | | 0.288 | | 0.310 | |
| AFA ^g | Constant | 0.002 (0.102) | 0.982 | -0.071 (0.213) | 0.740 | -0.177 (0.162) | 0.279 | -0.135 (0.159) | 0.401 |
| | BMI | 1.060 (0.093) | <0.001 | 1.207 (0.296) | <0.001 | 1.058 (0.095) | <0.001 | 1.021 (0.098) | <0.001 |
| | Stunted | | | -0.073 (0.205) | 0.721 | | | -0.192 (0.214) | 0.373 |
| | BMI × Stunted | | | -0.116 (0.210) | 0.582 | | | | |
| | SHR | | | | | 0.160 (0.112) | 0.158 | 0.194 (0.117) | 0.103 |
| | BMI × SHR | | | | | -0.029 (0.103) | 0.777 | | |
| | R ² adj | 0.699 | | 0.690 | | 0.699 | | 0.703 | |
| AFI ^h | Constant | 0.613 (0.163) | <0.001 | 0.605 (0.342) | 0.083 | 0.363 (0.261) | 0.169 | 0.383 (0.257) | 0.413 |
| | BMI | 0.804 (0.148) | <0.001 | 0.765 (0.476) | 0.114 | 0.797 (0.153) | <0.001 | 0.781 (0.159) | <0.001 |
| | Stunted | | | 0.111 (0.329) | 0.738 | | | -0.070 (0.346) | 0.838 |
| | BMI × Stunted | | | 0.041 (0.337) | 0.904 | | | | |
| | SHR | | | | | 0.225 (0.180) | 0.217 | 0.235 (0.189) | 0.221 |
| | BMI × SHR | | | | | -0.020 (0.165) | 0.903 | | |
| | R ² adj | 0.336 | | 0.313 | | 0.332 | | 0.332 | |

^aAll variables except stunting and percent body fat are age- and sex-specific z-scores calculated from Frisancho's comprehensive reference (2008).

^bUMA, upper arm muscle area (Frisancho, 2008).

^cBMI, body mass index.

^dStunting defined as height-for-age below the 5th percentile of Frisancho's comprehensive reference (2008).

^eNonstunted was set as the reference.

^fSHR, sitting height ratio.

^gAFA, arm fat area (Frisancho, 2008).

^hAFI, arm fat index, percent of the upper arm that is fat (Frisancho, 2008).

cially increased the %BF estimations of these Maya. This increase in estimated %BF may have artificially inflated the relationship between %BF and BMI found in this study. This is particularly the case since age, sex, weight, and stature are included in both the estimation equations for %BF and as predictor variables in this analysis.

It is also possible that a difference in living conditions between the shantytowns of Sao Paulo and the south of Merida caused a difference in the %BF of these children. Hoffman et al. reported low levels of total body adiposity, with the mean %BF 0.559 *z*-scores below the mean for the Maya of this study. In this study, all families had permanent housing and access to running water inside their property. All the children went to school (4 h a day) and almost all drank purified water (unpublished results). Hoffman's sample was drawn from shantytowns however the living conditions were not described. It is possible that the living conditions of the shantytowns may be worse than those found in the south of Merida. The relatively high standard of living conditions of participants in this study may have led to a higher %BF.

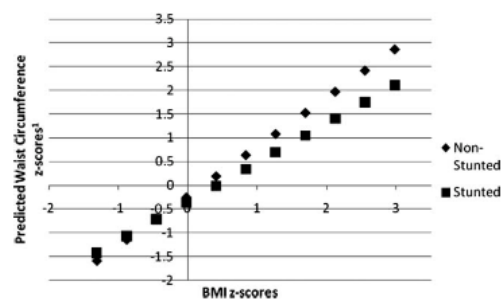


Fig. 2. Children's predicted WC *z*-scores from the interaction of BMI and stunting. ¹WC *z*-scores predicted by the final model for WC, as shown in Table 3, model 4.

It is well established that BMI better predicts %BF within the range of healthy BMIs than at low BMIs (Ellis, 2001). Therefore, when measuring children in transitioning societies who are poor, but still have basic sanitation and can afford to send a child to school, BMI may be an appropriate estimate of adiposity. This is primarily true when more accurate body composition methods are not available such as in medical clinics with limited time, equipment, and funding.

In this study, waist circumference was the best adiposity indicator predicted by BMI with nearly 80% of the variance in WC explained by BMI alone in both mothers' and children's models. BMI has been previously shown to correlate well with abdominal adiposity in the rapidly urbanizing and transitioning society in urban South African children of this age range (Cameron et al., 2009) and adults (Harris et al., 2000). In part this may be due to the influence of frame size on both BMI and WC. In adults, BMI and WC have been shown to be broadly similar in relation to adult CVD risk (Huxley et al., 2010; Satoh et al., 2010). Most studies involving individuals of non-European descent have found that WC has a greater impact on CVD risk than BMI, though the difference in magnitude is small enough that the clinical relevancy is questionable (Huxley et al., 2010).

Stunting did not alter the relationship between BMI and other adiposity indicators. Also, stunting was rarely significant in these models and had a low beta value in every model. This result suggests that BMI can be used in stunted populations in the same way as it is used in non-stunted populations. BMI appears to be a useful estimate of adiposity, especially when used in conjunction with other measures of body composition.

The hypothesis that differences in SHR change the relationship between BMI and adiposity indicators is not supported by this study of urban Mexican Mayans. Although SHR does impact BMI itself, it does not appear to significantly influence BMI as an estimate of adiposity indicators in this sample. This is surprising as the theoretical basis of this assumption is quite strong (Bogin and Beydoun, 2007; Deurenberg et al., 1999; Frisanchi, 2007).

TABLE 5. Estimates of women's body composition in multiple linear regression

| | | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|----------------------|---------------------------|----------------|----------|-----------------|----------|-----------------|----------|-----------------|----------|
| | | <i>B</i> (SE) | <i>P</i> | <i>B</i> (SE) | <i>P</i> | <i>B</i> (SE) | <i>P</i> | <i>B</i> (SE) | <i>P</i> |
| %BF ^a | Constant | 25.085 (3.535) | <0.001 | 19.063 (15.851) | 0.235 | 35.571 (25.574) | 0.170 | 50.893 (25.325) | 0.050 |
| | BMI ^b | 0.585 (0.119) | <0.001 | 0.718 (0.532) | 0.183 | 0.639 (0.125) | <0.001 | 0.670 (0.117) | <0.001 |
| | Stunted ^{c,d} | | | 2.762 (1.098) | 0.015 | | | 2.635 (1.138) | 0.025 |
| | BMI × Stunted | | | -0.650 (0.288) | 0.823 | | | | |
| | SHR ^e | | | | | -0.231 (0.490) | 0.639 | -0.569 (0.492) | 0.253 |
| | BMI × SHR | | | | | 0.247 (0.110) | 0.030 | 0.167 (0.111) | 0.141 |
| | <i>R</i> ² adj | 0.309 | | 0.364 | | 0.361 | | 0.413 | |
| WC (cm) ^f | Constant | 34.763 (3.949) | <0.001 | 11.591 (18.513) | 0.534 | 95.508 (28.838) | 0.022 | 89.306 (28.561) | 0.003 |
| | BMI | 1.815 (0.133) | <0.001 | 2.594 (0.621) | 0.983 | 1.890 (0.137) | <0.001 | 1.905 (0.139) | <0.001 |
| | Stunted | | | 0.027 (1.282) | 0.983 | | | 0.550 (1.288) | 0.671 |
| | BMI × Stunted | | | -0.432 (0.336) | 0.205 | | | | |
| | SHR | | | | | -1.171 (0.554) | 0.040 | -1.075 (0.557) | 0.059 |
| | BMI × SHR | | | | | 0.137 (0.125) | 0.277 | | |
| | <i>R</i> ² adj | 0.781 | | 0.780 | | 0.793 | | 0.789 | |

^a%BF, percent body fat calculated from an bioelectric impedance equation specific to American Indian women (Stolarczyk et al., 1994).

^bBMI, body mass index.

^cStunting for adult women defined as stature below 150 cm.

^dNonstunted was set as the reference.

^eSHR, sitting height ratio.

^fWC, waist circumference.

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TABLE 6. Estimates of women's upper arm composition in multiple linear regression

| | | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|-------------------------------------|------------------------|-----------------|--------|------------------|-------|-----------------|--------|-----------------|--------|
| | | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P |
| UMA (cm ²) ^a | Constant | 15.097 (6.964) | 0.035 | 44.382 (32.831) | 0.183 | 33.167 (52.516) | 0.531 | 20.580 (52.136) | 0.695 |
| | BMI ^b | 0.733 (0.234) | 0.003 | -0.277 (1.101) | 0.802 | 0.720 (0.249) | 0.006 | 0.749 (0.253) | 0.005 |
| | Stunted ^{c,d} | | | 0.990 (0.274) | 0.665 | | | 1.086 (2.352) | 0.646 |
| | BMI × Stunted | | | 0.563 (0.596) | 0.945 | | | | |
| | SHR ^e | | | | | -0.323 (1.010) | 0.751 | -0.127 (1.017) | 0.901 |
| | BMI × SHR | | | | | -0.277 (0.227) | 0.229 | | |
| R ² adj | | 0.145 | | 0.129 | | 0.136 | | 0.113 | |
| AFA (cm ²) ^f | Constant | -32.817 (2.920) | 0.001 | -41.949 (40.278) | 0.303 | 70.103 (65.531) | 0.290 | 54.395 (62.402) | 0.388 |
| | BMI | 2.408 (0.296) | <0.001 | 2.884 (1.351) | 0.038 | 2.577 (0.310) | <0.001 | 2.509 (0.303) | <0.001 |
| | Stunted | | | -5.642 (2.790) | 0.049 | | | -4.743 (2.815) | 0.092 |
| | BMI × Stunted | | | -0.282 (0.731) | 0.702 | | | | |
| | SHR | | | | | -2.000 (1.260) | 0.119 | -1.600 (1.217) | 0.195 |
| | BMI × SHR | | | | | 0.056 (0.284) | 0.843 | | |
| R ² adj | | 0.556 | | 0.575 | | 0.563 | | 0.588 | |
| AFL ^g | Constant | 19.883 (8.751) | 0.027 | 9.628 (40.681) | 0.814 | 52.259 (65.329) | 0.428 | 59.163 (63.916) | 0.359 |
| | BMI | 1.041 (0.294) | 0.001 | 1.496 (1.364) | 0.278 | 1.148 (0.309) | 0.001 | 1.075 (0.311) | 0.001 |
| | Stunted | | | -4.341 (2.818) | 0.130 | | | -4.002 (2.883) | 0.171 |
| | BMI × Stunted | | | -0.266 (0.739) | 0.720 | | | | |
| | SHR | | | | | -0.672 (1.256) | 0.595 | -0.695 (1.246) | 0.580 |
| | BMI × SHR | | | | | 0.373 (0.283) | 0.194 | | |
| R ² adj | | 0.181 | | 0.190 | | 0.190 | | 0.193 | |

^aUMA, upper arm muscle area (Frisancho, 2008).

^bBMI, body mass index.

^cStunting for adult women defined as stature below 150 cm.

^dNonstunted was set as the reference.

^eSHR, sitting height ratio.

^fAFA, arm fat area (Frisancho, 2008).

^gAFL, arm fat index, percent of the upper arm that is fat (Frisancho, 2008).

According to the literature, SHR does appear to significantly impact BMI and can do so quite substantially in certain groups; however, it may be a relatively small effect when considered alongside other factors. A relatively small sample, with limited variability in both total stature and SHR, such as the one used in this study would have less power to detect these relatively small differences. The overall short stature of this population suggests that all members of the population could have experienced chronically poor conditions regardless of their stunting status. Even the tall individuals in this sample may be at risk for the negative health outcomes associated with short stature since the relationship between stature and mortality is linear (Song and Sung, 2008). The apparent environmentally imposed limit on stature may be over-riding any impact of stunting or body proportions that this study was attempting to investigate. A sample with more variation in stature may be more likely to determine the impact of stunting on BMI. This study can only conclude that in a small, very short sample with limited variability in linear growth, dividing the group into stunted and nonstunted does not influence the use of BMI as a predictor of adiposity indicators. Also BMI alone is not sufficient to adequately estimate adiposity indicators, particularly in a population which is chronically undernourished (stunted). Using BMI alone would give a skewed picture of the overall nutritional status of a dual burdened population as the levels of adiposity are high but the nutrition is not of high enough quality for adequate growth, resulting in short stature.

The small sample and limited variability in stature and SHR greatly reduce the power of this study to find statistical differences. Studies involving a larger sample with greater variability are much more likely to detect statistical differences, particularly if differences actually exist.

Future studies may also measure chronic disease risk factors, as well as adiposity indicators to obtain a better understanding of the relationship between stunting, body proportions, BMI, and chronic disease. Obesity-related chronic diseases are becoming a very large health concern in developing countries, particularly middle income countries such as Mexico, which are still grappling with undernutrition. More fully understanding the relationship between over and undernutrition will help inform greatly needed public health policies.

CONCLUSIONS

In conclusion, the samples of women and children studied have high levels of adiposity and short stature with low variation in stature. This small study found that BMI is appropriate for use to estimate adiposity indicators in this sample of 7–9-year-old urban Mayan children. However, it is recommended that BMI be used in conjunction with other measures such as stature and waist circumference to obtain a more complete estimation of a child's nutritional status. Conversely, BMI is not recommended for use in this sample of adult urban Mayan women. WC may be of value for use in these women to estimate their chronic disease risk, though its validity has not yet been established for this population. The ability of BMI to predict adiposity indicators is not impacted by stunting status or SHR in this sample.

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LITERATURE CITED

- Asao K, Kao WH, Baptiste-Roberts K, Bandeen-Roche K, Erlinger TP, Brancati FL. 2006. Short stature and the risk of adiposity, insulin resistance, and type 2 diabetes in middle age: the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. *Diabetes Care* 29:1632–1637.
- Aulchenko YS, Struchalin MV, Belonogova NM, Axenovich TI, Weedon MN, Hofman A, Uitterlinden AG, Kayser M, Oostra BA, van Duijn CM, Janssen AC, Borodin PM. 2009. Predicting human height by Victorian and genomic methods. *Eur J Hum Genet* 17:1070–1075.
- Barquera S, Peterson KE, Must A, Rogers BL, Flores M, Houser R, Montterubio E, Rivera-Dommarco JA. 2007. Coexistence of maternal central adiposity and child stunting in Mexico. *Int J Obes (Lond)* 31:601–607.
- Bogin B. 1999. Patterns of human growth. Cambridge: Cambridge University Press.
- Bogin B, Beydoun MA. 2007. The relationship of sitting height ratio to body mass index and fatness in the United States, 1988–1994. *Hum Ecol Special Issue* 15:1–8.
- Bogin B, Varela-Silva MI. 2008. Fatness biases the use of estimated leg length as an epidemiological marker for adults in the NHANES III sample. *Int J Epidemiol* 37:201–209.
- Bogin B, Wall M, MacVean RB. 1992. Longitudinal analysis of adolescent growth of ladino and Mayan school children in Guatemala: effects of environment and sex. *Am J Phys Anthropol* 89:447–457.
- Bowerman B, O'Connell RT. 1990. Linear statistical models: an applied approach. Boston: PWS-Kent Pub. Co. 1012 p.
- Burkhauser RV, Cawley J. 2008. Beyond BMI: the value of more accurate measures of fatness and obesity in social science research. *J Health Econ* 27:519–529.
- Cameron N. 2002. Human growth curve, canalization, and catch-up growth. In: Cameron N, editor. Human growth and development. Amsterdam: Academic Publishers. p 1–20.
- Cameron N, Jones LL, Griffiths PL, Norris SA, Pettifor JM. 2009. How well do waist circumference and body mass index reflect body composition in pre-pubertal children? *Eur J Clin Nutr* 63:1065–1070.
- Cameron N, Wright MM, Griffiths PL, Norris SA, Pettifor JM. 2005. Stunting at 2 years in relation to body composition at 9 years in African urban children. *Obes Res* 13:131–136.
- Case A, Paxson C. 2008. Stature and status: height, ability, and labor market outcomes. *J Polit Econ* 116:499–532.
- Cohen J. 1992. A power primer. *Psychol Bull* 112:115–159.
- Crooks DL. 1994. Growth status of school-age Mayan children in Belize, Central-America. *Am J Phys Anthropol* 93:217–227.
- Deurenberg P, Yap MD, Wang J, Lin FP, Schmidt G. 1999. The impact of body build on the relationship between body mass index and percent body fat. *Int J Obes* 23:537–542.
- Doak CM, Adair LS, Bentley M, Monteiro C, Popkin BM. 2005. The dual burden household and the nutrition transition paradox. *Int J Obes* 29:129–136.
- Doak CM, Adair LS, Monteiro C, Popkin BM. 2000. Overweight and underweight coexist within households in Brazil, China and Russia. *J Nutr* 130:2965–2971.
- Ellis KJ. 2001. Selected body composition methods can be used in field studies. *J Nutr* 131:1589S–1595S.
- Ellis KJ, Abrams SA, Wong WW. 1999. Monitoring childhood obesity: assessment of the weight/height² index. *Am J Epidemiol* 150:939–946.
- Florencio TT, Ferreira HS, Cavalcante JC, Stux GR, Sawaya AL. 2007. Short stature, abdominal obesity, insulin resistance and alterations in lipid profile in very low-income women living in Maceio, north-eastern Brazil. *Eur J Cardiovasc Prev Rehabil* 14:346–348.
- Frankenfield DC, Rowe WA, Cooney RN, Smith JS, Becker D. 2001. Limits of body mass index to detect obesity and predict body composition. *Nutrition* 17:26–30.
- Frisancho AR. 2007. Relative leg length as a biological marker to trace the developmental history of individuals and populations: growth delay and increased body fat. *Am J Hum Biol* 19:703–710.
- 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. 335 p.
- Fuke Y, Okabe S, Kajiwara N, Suastika K, Budhiarta AA, Maehata S, Taniguchi H. 2007. Increase of visceral fat area in Indonesians and Japanese with normal BMI. *Diabetes Res Clin Pract* 77 Suppl 1:S224–S227.
- Gabrielsson BG, Johansson JM, Lonn M, Jernas M, Olbers T, Peltonen M, Larsson I, Lonn L, Sjostrom L, Carlsson B, Carlsson LMS. 2003. High expression of complement components in omental adipose tissue in obese men. *Obes Res* 11:699–708.
- Garrett J, Ruel MT. 2005. The coexistence of child undernutrition and maternal overweight: prevalence, hypotheses, and programme and policy implications. *Matern Child Nutr* 1:185–196.
- Goings S, Nichols J, Loftin M, Stewart D, Lohman T, Tuuri G, Ring K, Pickrel J, Blew R, Stevens J. 2006. Validation of bioelectrical impedance analysis (BIA) for estimation of body composition in Black, White and Hispanic girls. *Int J Body Comp Res* 4:161–167.
- Gunnell DJ, Smith GD, Frankel SJ, Kemp M, Peters TJ. 1998. Socio-economic and dietary influences on leg length and trunk length in childhood: a reanalysis of the Carnegie (Boyd Orr) survey of diet and health in prewar Britain (1937–39). *Paediatr Perinat Epidemiol* 12:96–113.
- Hall DM, Cole TJ. 2006. What use is the BMI? *Arch Dis Child* 91:283–286.
- Harris TB, Visser M, Everhart J, Cauley J, Tyllavsky F, Fuerst T, Zamboni M, Taffe D, Resnick HE, Scherzinger A, Nevitt M. 2000. Waist circumference and sagittal diameter reflect total body fat better than visceral fat in older men and women. The Health, Aging and Body Composition Study. *Ann NY Acad Sci* 904:462–473.
- Hoffman DJ, Roberts SB, Martins PA, de Nascimento C, Sawaya AL. 2000a. Evidence for impaired regulation of energy intake in nutritionally stunted children from the shantytowns of Sao Paulo, Brazil. *Obes Res* 8:775–778.
- Hoffman DJ, Roberts SB, Verreschi I, Martins PA, de Nascimento C, Tucker KL, Sawaya AL. 2000b. Regulation of energy intake may be impaired in nutritionally stunted children from the shantytowns of Sao Paulo, Brazil. *J Nutr* 130:2265–2270.
- Hoffman DJ, Sawaya AL, Coward WA, Wright A, Martins PA, de Nascimento C, Tucker KL, Roberts SB. 2000c. Energy expenditure of stunted and nonstunted boys and girls living in the shantytowns of Sao Paulo, Brazil. *Am J Clin Nutr* 72:1025–1031.
- Hoffman DJ, Sawaya AL, Martins PA, McCrory MA, Roberts SB. 2006. Comparison of techniques to evaluate adiposity in stunted and nonstunted children. *Pediatrics* 117:e725–e732.
- Huxley R, Mendis S, Zheleznyakov E, Reddy S, Ekman J. 2003a. Body mass index, waist circumference and waist:hip ratio as predictors of cardiovascular risk—a review of the literature. *Eur J Clin Nutr* 64:16–22.
- Jenkins CL. 1981. Patterns of growth and malnutrition among preschoolers in Belize. *Am J Phys Anthropol* 56:169–178.
- Kruger HS, Margets BM, Vorster HH. 2004. Evidence for relatively greater subcutaneous fat deposition in stunted girls in the North West Province, South Africa, as compared with non-stunted girls. *Nutrition* 20:564–569.
- Lara-Esqueda A, Aguilar-Salinas CA, Velazquez-Monroy O, Gomez-Perez FJ, Rosas-Peralta M, Mehta R, Tapia-Conyer R. 2004. The body mass index is a less-sensitive tool for detecting cases with obesity-associated co-morbidities in short stature subjects. *Int J Obes* 28:1443–1450.
- Lau DC, Dhillon B, Yan H, Szmikto PE, Verma S. 2005. Adipokines: molecular links between obesity and atherosclerosis. *Am J Physiol Heart Circ Physiol* 288:H2031–H2041.
- Leatherman TL, Goodman A. 2005. Coca-colonization of diets in the Yucatan. *Soc Sci Med* 61:833–846.
- Leatherman TL, 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.
- Leatherman TL, Stillman JT, Goodman AH. 2000. The effects of tourism-led development on the nutritional status of Yucatec Mayan children. Paper presented at the Annual Meeting of the American Association of Physical Anthropologists, April 2000. San Antonio, TX. *Am J Phys Anthropol* 30:207.
- Leitch I. 1951. Growth and health. *Br J Nutr* 5:142–151.
- Leonard WR, Sorensen MV, Mosher MJ, Spitsyn V, Comuzzie AG. 2009. Reduced fat oxidation and obesity risks among the Buryat of Southern Siberia. *Am J Hum Biol* 21:664–670.
- Lohman TG, Caballero B, Himes JH, Hunsberger S, Reid R, Stewart D, Skipper B. 1999. Body composition assessment in American Indian children. *Am J Clin Nutr* 69(4 Suppl):764S–766S.
- Lohman TG, Roche AF, Martorell R. 1988. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics Books.
- Lopez-Alvarenga JC, Montesinos-Cabrera RA, Velazquez-Alva C, Gonzalez-Barranco J. 2003. Short stature is related to high body fat composition despite body mass index in a Mexican population. *Arch Med Res* 34:137–140.
- Malina RM, Pena Reyes ME, Little BB. 2008. Epidemiologic transition in an isolated indigenous community in the Valley of Oaxaca, Mexico. *Am J Phys Anthropol* 137:69–81.
- Malina RM, Pena Reyes ME, Little BB. 2009. Socioeconomic variation in the growth status of urban school children 6–13 years in Oaxaca, Mexico, in 1972 and 2000. *Am J Hum Biol* 21:805–816.
- Martins PA, Hoffman DJ, Fernandes MTB, Nascimento CR, Roberts SB, Sesso R, Sawaya AL. 2004. Stunted children gain less lean body mass and more fat mass than their non-stunted counterparts: a prospective study. *Brit J Nutr* 92:819–825.

- Norgan NG. 1994a. Population differences in body composition in relation to the body mass index. *Eur J Clin Nutr* 48 Suppl 3:S10–S25; discussion S26–S17.
- Norgan NG. 1994b. Relative sitting height and the interpretation of the body mass index. *Ann Hum Biol* 21:79–82.
- Popkin BM. 1996. Understanding the nutrition transition. *Urban Health News* 1(30):3–19.
- Popkin BM, Doak CM. 1998. The obesity epidemic is a worldwide phenomenon. *Nutr Rev* 56(4 Part 1):106–114.
- Rivera JA, Barquera S, Gonzalez-Cossio T, Olaiz G, Sepulveda J. 2004. Nutrition transition in Mexico and in other Latin American countries. *Nutr Rev* 62(7 Part 2):S149–S157.
- Satoh H, Fujii S, Furumoto T, Kishi R, Tsutsui H. 2010. Waist circumference can predict the occurrence of multiple metabolic risk factors in middle-aged Japanese subjects. *Ind Health* 48:447–451.
- Song YM, Sung J. 2008. Adult height and the risk of mortality in South Korean women. *Am J Epidemiol* 168:497–505.
- Stein AD, Wang M, Martorell R, Norris SA, Adair LS, Bas I, Sachdev HS, Bhargava SK, Fall CH, Gigante DP, Victora CG, on behalf of the Cohorts Group. 2010. Growth patterns in early childhood and final attained stature: data from five birth cohorts from low- and middle-income countries. *Am J Hum Biol* 22:353–359.
- Stolarsczyk LM, Heyward VH, Hicks VL, Baumgartner RN. 1994. Predictive accuracy of bioelectrical impedance in estimating body composition of Native American women. *Am J Clin Nutr* 59:964–970.
- Van de Poel E, Hosseini AR, Speybroeck N, Van Ourti T, Vega J. 2008. Socioeconomic inequality in malnutrition in developing countries. *Bull World Health Organ* 86:282–291.
- Van de Poel E, O'Donnell O, Van Doorslaer E. 2007. Are urban children really healthier? Evidence from 47 developing countries. *Soc Sci Med* 65:1986–2003.
- Varela-Silva MI, Azcorra H, Dickinson F, Bogin B, Frisancho AR. 2009. Influence of maternal stature, pregnancy age, and infant birth weight on growth during childhood in Yucatan, Mexico: a test of the intergenerational effects hypothesis. *Am J Hum Biol* 21:657–663.
- Walker SP, Chang SM, Powell CA. 2007. The association between early childhood stunting and weight status in late adolescence. *Int J Obes (Lond)* 31:347–352.

2. Article in press

Fat free mass explains the relationship between stunting and energy expenditure in urban Mexican Maya children

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Abstract

Background: Childhood stunting has been associated with an increased risk of obesity in adulthood, but the causes are unclear. We hypothesize that stunting significantly reduces both resting and activity energy expenditure.

Aim: To assess and describe energy expenditure of low socio-economic Maya children and to determine whether stunting is independently related to energy expenditure after controlling for lean mass.

Subjects and Methods: 33 urban Maya children, 17 boys, aged 7-9 years, living in Merida, Mexico were measured for height, weight and bioelectrical impedance analysis (BIA). Body composition was estimated from BIA. Energy expenditure was measured for one week using the Actiheart (combined heart rate and accelerometer).

Results: Stunting (height-for-age below the 5th percentile of NHANES III based references) affected 35% of these physically active children. Using multiple linear regression analysis, greater lean body mass predicted higher resting and activity energy expenditure. Stature was not a significant predictor of resting energy expenditure. A lower 'height-for-age z-score', but not stunting as a categorical variable, significantly predicted lower activity energy expenditure.

Conclusion: Our hypothesis that stunting reduces total energy expenditure (resting + active) in children is not supported. Rather children with shorter stature and less lean body mass have lower total energy expenditure. Complex interactions between body size, body composition and metabolic activity appear to elevate the risk for later life obesity in these Maya children.

Introduction

Middle-income countries, such as Mexico and Brazil, are currently experiencing a rise in obesity levels among their poor (Mendez et al. 2005; Popkin et al. 2012). It is possible that this rise in obesity is following the pattern shown in populations in high income countries as globalized diets and lifestyles are adopted by the populations of lower income nations (Adair and Popkin 2005; Barquera et al. 2008; Caballero 2007; Popkin et al. 2012). However, additional causes for this rise in obesity may exist. Trends toward urbanisation, including rural-to-urban migration, with associated changes in physical activity are a possible cause (Katzmarzyk and Mason 2009; Monda et al. 2007; Parra et al. 2009). In addition, the most impoverished segments of lesser-developed nations may have experienced a relatively rapid and acute shift from general protein-energy under-nutrition to a more complex mix of under-supply of specific amino acids, vitamins and (Delisle 2008). There is indirect evidence from research with adults indicating that this change in the nature of malnutrition is accompanied by metabolic shifts, such as reduced fat oxidation with increased risk of overweight/obesity (Florencio et al. 2003; Leonard et al. 2009).

Low socioeconomic status (SES), urban, Maya children are the focus of this study. Maya living in the Mexican Yucatan Peninsula are currently undergoing nutrition transition (Leatherman and Goodman 2005; Leatherman et al. 2010) and are a double burdened population, with simultaneously high rates of stunting and overweight and obesity (Azcorra et al. 2009; Dickinson 1997; Varela-Silva et al. 2012).

Stunted and short height-for-age individuals may be at an increased risk of overweight or obesity (OW/OB) during the growing years compared with their non-stunted peers (Bogin and Loucky 1997; Corvian et al. 2008; Hoffman et al. 2007; Mardones et al. 2008; Martins et al. 2004; Smith et al. 2003; Walker et al. 2007). Changes in resting metabolic rate (RMR) occur during the first five years of life (Grillo et al. 2005) the same period that stunting most often occurs (Fox and Hillsdon 2007). A decreased metabolism could be an efficient method of adapting to energy requirements in response to low caloric availability. Yet a slowing of the metabolism places individuals at an increased risk of weight gain in conditions of caloric abundance. Preliminary evidence suggests that metabolic shifts may be associated with the risk for obesity in stunted children (Grillo et al. 2005; Hoffman et al. 2000b; Soares-Wynter and Walker 1996; Wren et al. 1997) but the exact conditions this relationship requires have yet to be fully understood. The present study attempts to objectively estimate urban Maya children's RMR and determine whether it is influenced by stunting status.

The changes in behaviour that occur as part of urbanisation and modernisation may play a substantial role in nutrition-related, non-communicable diseases. The behavioural transition, associated with modernisation and the nutrition transition, is characterised by an increase in sedentary activity (Onywera 2010) and reduced occupational physical activity (Huneault et al. 2011; Popkin et al. 2002) in adults. Low levels of activity energy expenditure (AEE) and physical activity are risk factors for obesity (Fox and Hillsdon 2007; Gomez et al. 2007; Monda et al. 2008).

The relationship between physical activity and adiposity or OW/OB in children living in developing countries has been less studied compared to adults in the same environments and children in developed countries (Department of Health 2004). Research with Latin American and Mexican children has found an increase in sedentary behaviour, such as television viewing (Hernandez et al. 1999; Malina et al. 2008; Sauri Bazán 2003). However frequent participation in sedentary activities does not necessarily result in low physical activity levels (Biddle et al. 2004; Malina et al. 2008). Thus, it is not clear what the relationship is between children's physical activity and adiposity or OW/OB in the nutrition transition. This present study presents objective measurements of the physical activity and AEE of urban Maya children and classifies them for risk of negative health outcomes.

This is one of the first studies to objectively assess energy expenditure using combined heart rate monitoring and accelerometry, the Actiheart, in free-living children in developing countries. A previous study has been performed in adolescents in urban Brazil (Victora et al. 2008). The Actiheart allows habitual physical activity to be assessed objectively and accurately (Assah et al. 2011; Corder et al. 2005) for up to 3 weeks at a time (CamNtech 2009). The Actiheart was designed to minimize high discomfort and burden on the participant and has been validated against double-labelled water (Catalano and Ehrenberg 2006) and indirect calorimetry (Brage et al. 2005; Corder et al. 2005). This makes it a very good tool for assessing free-living activity.

The purpose of this article is to report the physical activity levels of low SES, urban Maya children and to assess whether chronic under-nutrition (stunting) impacts their energy expenditure. We hypothesize that stunting significantly reduces both resting and activity energy expenditure, independently of lean mass.

Methods

Sample

The study design was cross-sectional and conducted between March and July 2010. This sample was composed of 7.00-9.99 year old urban Maya school children living in Merida, Yucatan, Mexico.

Recruitment

Schools located in the low SES neighbourhoods of southern Merida, known to contain a relatively high proportion of Maya families, were selected. Children of Maya ethnicity, aged 7-9 years-old, were identified from school lists by having two Maya surnames, one from their mother and one from their father. The mothers were then invited to group information sessions at their children's schools where the nature of the study was verbally explained, in Spanish, and information sheets, in Spanish, were provided. The Maya living in the south of Merida were highly acculturated and all were comfortable speaking Spanish.

Written informed consent was obtained from the mothers and verbal assent from the children. Ethical clearance was obtained from the Loughborough University

Ethics Committee in the U.K. (approval number: R09-P145) and the Bioethical Committee of CINVESTAV in Mexico. The Bioethical Committee of CINVESTAV does not give ethical approval numbers.

F. *Anthropometry*

Children underwent anthropometric measurements for stature (Gordon et al. 1988), weight, waist circumference (WC) (Callaway et al. 1988) and skinfolds (triceps and sub-scapular) (Harrison et al. 1988) using standard techniques. Body mass index (BMI) was calculated by dividing the child's weight in kilograms by their statures in metres squared. The technical error of measurement for anthropometry was not calculated. The research team received careful training both before and during the fieldwork.

The Comprehensive sex- and age-specific reference charts (Frisancho 2008) were used to calculate z-scores for BMI and WC. The Comprehensive reference was chosen as it was created using data from the Third National Health and Nutrition Examination Survey (NHANES III) from the United States. NHANES III is a stratified, nationally representative sample of the US population, which over-sampled ethnic minorities. The percentage of participants according to ethnicity is: White (European-American) 34.4%, Black (African-American) 26.8%, Hispanic/Latino (mostly Mexican-American and Cuban) 27.0% and Other 4.3%, with 7.5% missing an ethnic affiliation. NHANES III is, perhaps, the largest sampled, statistically validated growth reference that includes Mexican children. As such, it is the most appropriate for use with a Mexican population such as the Maya.

Children were classified as being stunted if their height-for-age was below the 5th percentile.

G. *Bioelectrical impedance analysis*

Body composition was measured using bioelectric impedance analysis (BIA) with a BioScan 916 by (Maltron, UK). Percent body fat (%BF) was calculated using the impedance and reactance values with equations specific for American Indian children (Equation 1) (Lohman et al. 19999) as the Maya are an indigenous American group. %BF was converted into kilograms of body fat, which was used to calculate fat free mass (FFM). %BF was compared against age- and sex-specific

reference curves (Ogden et al. 2011). The technical error of measurement for BIA was not calculated. The research team received careful training in BIA both before and during the fieldwork.

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

Definitions: Sex coded 1 for girls, 0 for boys. Weight is in kg. Skinfold thicknesses are in mm. Resistance and reactance are in ohms. Stature is in m.

H. *Energy expenditure*

Physical activity of the children was measured for 7 days using an Actiheart, a combined heart rate and accelerometer (Corder et al. 2007; Corder et al. 2005; Wilson et al. 2011). Children were included in the analysis if they had at least 3 days usable data that included a minimum of 2 weekdays and 1 weekend day. A day was defined as a minimum of 10 hours of usable data during waking hours (Ward et al. 2005). Data was collected and analysed in minute-long epochs or sampling interval.

Energy expenditure was estimated using branched equation modelling in Actiheart software v.4.52. Data were cleaned by removing extended periods (>5 minutes) in which the heart rate data was missing and also when there was a mismatch in the heart rate and accelerometry data, for example, extended periods of high accelerometry counts and low heart rates (but not the reverse). The software performed straight line interpolation for periods of missing heart rate data lasting up to 5 minutes, allowing energy expenditure to be estimated for these periods. The period of data removal was extended if 5 minutes of missing heart rate data was preceded or followed by one minute of heart rate data and then another period of missing heart rate data.

Resting energy expenditure (REE), activity energy expenditure (AEE) and total energy expenditure (TEE) were used in the analyses. All variables were calculated using predictive equations with an external reference curve created using 13 year old British children (Corder et al. 2005). The error introduced into the sample through using an external group calibration curve is likely similar across the sample, as the sample is fairly genetically homogeneous and has experienced similar chronic

environmental conditions. However the error introduced by this method of estimation is probably too high for direct comparisons with external samples.

REE was calculated using the Schofield equation (Schofield 1985). Sleeping heart rate was individually calculated for each child using the average heart rate during extended periods (>2 hours) of negligible accelerometry counts during night hours (12 pm to 9 am). AEE was calculated when the heart rate was above sleeping heart rate. TEE was calculated as the additive combination of REE, AEE and diet induced thermogenesis.

For assessment of the level of physical activity in which the children engaged, metabolic equivalent (MET) was calculated by the Actiheart software. The time at each MET was used to classify the activity level of each minute-long epoch as low (MET<3) or moderate-to-vigorous (MET ≥3).

Statistical analysis

Normality of the distribution of all variables was checked using Kolmogorov-Smirnov test and skewness and kurtosis. Independent *t*-tests were used to compare chronic nutritional status (stunted v. non-stunted) and sex with anthropometric, body composition and energy expenditure variables. Energy expenditure variables were linearly regressed onto BMI z-score and WC z-scores and body composition variables. Independent *t*-tests were used to compare included and excluded children for anthropometric and body composition measures. Energy expenditure variables were not compared between included and excluded children because of the unreliability of the excluded measures.

Stepwise multiple linear regressions using the enter method were performed with measures of energy expenditure (REE, AEE and TEE) as the dependent variables. The independent variables were added in three steps: 1) FFM, 2) height-for-age z-score or stunting and 3) sex. Normality of the residuals were checked

All analyses were undertaken using PASW (SPSS) v.18.0. Significance was set *a priori* at $p<0.05$.

Results

From the 58 children recruited, 33 were included in the final analysis. Children were excluded for incomplete Actiheart data ($n=24$) and health problems ($n=1$). The reason for missing Actiheart data was primarily poor electrode adherence caused by high rates of sweating in the hot, humid climate. This resulted in missing heart rate data or the device falling off of the skin (Wilson et al. 2011). Also several Actihearts were broken during the course of the week. See Wilson *et al.* (2011) for a more complete description of the logistical difficulties encountered when attempting to use Actihearts in children. The included children were not statistically significantly different from excluded children for any measures of anthropometry or body composition (Table 1).

Table 1 about here

Overall these urban Mexican Maya children were short and except for the non-stunted girls had low weight-for-age z-scores (Table 2). Stunted children were significantly smaller in stature and weight, with lower estimated FFM and FM than their non-stunted peers. BMI z-scores did not significantly differ between the stunted and non-stunted children. Using United States references based upon the NHANES III (1999-2004), the mean %BF of the stunted girls is about the 17th percentile, at about the 61st percentile for the non-stunted girls and both stunted and non-stunted boys are at about the 53rd percentile (Ogden et al. 2011).

Table 2 about here

Stunted children had lower levels of, REE, AEE and TEE (Table 3) compared to non-stunted children. Stunted children spent significantly more time in light physical activity and less time per day in moderate-to-vigorous physical activity (MVPA) than non-stunted children. No significant differences in REE, AEE or TEE were found between the sexes.

Overall, this sample of children was highly active, spending an average of 120 minutes per day in MVPA. However girls and stunted children spent significantly less time in MVPA compared to boys and non-stunted children, respectively. Of the five children who did not spend 60 minutes per day in MVPA, all were girls and four were

stunted. The variation in time spent at each level of physical activity was high, with a range of 20-312 minutes per day spent in moderate-to-vigorous physical activity.

Table 3 about here

In multiple linear regression models (Table 4), kilograms of FFM was the largest predictor of all measures of energy expenditure (REE, AEE and TEE), explaining between 33 and 81% of the variance in energy expenditures (Table 4). Boys had significantly higher measures of all measures of energy expenditure than girls. Neither height-for-age z-scores nor stunting were significant predictors of REE. Height-for-age z-score but not stunting was a significant predictor for AEE and TEE.

Table 4 about here

1 **Discussion**

Our hypothesis that stunting, *per se*, is associated with lower estimated energy expenditure (resting + activity) in children is not supported by the current study. Rather children with short stature, as a continuous variable, and lower fat free mass (FFM) have lower total energy expenditure (TEE) compared to taller children with higher FFM. This study of Maya children extends the understanding of the relationship between chronic under-nutrition and obesity risk by adjusting for the primary determinant of resting energy expenditure (REE), lean tissue. For these Maya children the lower absolute amount of FFM in the stunted children may mitigate other factors and result in a lower REE. The stunted children also have lower activity energy expenditure (AEE), a potential behavioural modification. The differences in behaviour according to stature are clearly revealed by the Actiheart data, which show that the stunted children engage in significantly less moderate-to-vigorous activity than the non-stunted children. This finding of lower REE and AEE leading to lower TEE in stunted Maya children can be considered in combination with previous findings that Brazilian stunted children have a decreased ability to regulate energy intake (Hoffman et al. 2000a) and gain more fat than their non-stunted peers

(Martins et al. 2004). These studies suggest that physiologic shifts may occur with stunting, increasing the risk of excess weight gain in stunted individuals, which may lead to individual double burden. Findings from indigenous adult Siberians, implicate reduced fat oxidation as one of these physiological shifts (Leonard et al. 2009). We did not measure directly metabolic activity at the physiological level in these urban Maya children, and this needs to be done to better evaluate our findings in light of past research.

A statistically significant interaction between FFM, sex and height-of-age z-score was observed in all regression models. Compared with non-stunted boys and girls, stunted girls had a significantly lower REE and TEE than stunted boys (Table 3). Stunted girls also had 49% lower MVPA than non-stunted girls (60 vs. 116.7 MET). In contrast, stunted boys had 31% lower MVPA than non-stunted boys (113.6 vs. 164.25 MET).

Other studies have found that stunting has effects on energy expenditure. Soares-Wynter and Walker (1996) found lower REE in stunted Jamaican children aged 7-8 years old but did not report sex composition of the sample or differences between male and female children. Our findings are similar to those of Grillo et al. (2005) who found in a case-controlled study in the shantytowns of Sao Paulo, Brazil that stunted girls had lower resting energy expenditure than age- and weight-for-height matched, non-stunted girls. In the same Brazilian cohort, Martins et al. (2004) reported that stunted children had an increased %BF and reduced %FFM compared to their non-stunted, age- and weight-for-height matched peers. Our new results find a trend toward just the opposite for body composition in girls.

The AEE data from these Maya children also stand in contrast to the same stunted Brazilian children, which found equivalent AEEs in stunted and non-stunted children (Hoffman et al. 2000a). Another study of rural indigenous children and urban children, all from poor families, in Oaxaca, Mexico found that stunted children exhibited similar fitness levels to non-stunted, adequately nourished children (Malina et al. 2011). It appears that the Mexican Maya children of our study have a different relationship between stunting and physical activity levels from the children in Sao Paulo and Oaxaca. From these data, it is not possible to know whether the lower levels of physical activity in the stunted Maya are driven by biological or social processes.

These urban Maya children can be considered very active, with 85% (n=28) spending at least one hour per day in moderate-to-vigorous physical activity.

Recommendations are that children should be moderately to vigorously active for a minimum of 60 minutes a day (Department of Health 2004) for independent reduction of chronic disease risk (Balas-Nakash et al. 2010; Bell et al. 2007). Even the stunted girls achieved on average 60 minutes per day of MVPA.

It is notable that the only children who failed to spend an hour a day in MVPA were girls. That the majority of these girls (4 of 5) were stunted is suggestive of a biosocial or biocultural relationship related to the sex of the child. It is likely that this sample of girls will become less active in adolescence (Dumith et al. 2011), increasing their risk for obesity in adulthood. The mothers of these children had very high levels of obesity (Varela-Silva et al. 2012). Women who are both stunted and overweight place their own children at risk of negative health outcomes. Stunted mothers are more likely to have children who are low birth weight (Victora et al. 2008), who die before their fifth birthday (Monden and Smits 2009) and who are stunted (Delisle 2008). Overweight mothers are more likely to miscarry, have gestational diabetes and have children who are overweight (Catalano and Ehrenberg 2006). Thus a population with a large number of women with individual double burden faces a public health concern in for multiple generations facing cycles of ill health increasing the risk for the cycle of poverty to continue (Harper et al. 2003).

. The applicability of the results of this study to other human groups is limited by the small sample size, the use of a group calibration curve for the energy expenditure estimation and a BIA predictive equation that was not specific to the Maya. Some strengths of this study are the variety of data collected simultaneously, especially the use of an objective and well-validated instrument, the Actiheart, to estimate energy expenditure. The cost and participant burden of traditional methods of objectively measuring energy expenditure have tended to limit their use in low income groups of developing countries, with self-reported physical activity being the preferred method.

Conclusion

Complex interactions between body size, body composition and metabolic activity appear to elevate the risk for current and later life obesity in this sample of Maya children. These children were found to be highly active but girls and stunted children exhibited the lowest level of physical activity. In this sample, shorter children were less active than taller children, and stunted children spent significantly less time in MVPA than non-stunted children. The effect of stature seems to be mediated via the lower FFM of the shorter/stunted children and not primarily due to the stunting. Even so, an important point to make here is that lower FFM is associated with short stature/stunting and public health workers and policy planners may still use short stature as a proxy for higher risk of negative health outcomes.

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Literature cited

- Adair LS, Popkin BM. 2005. Are child eating patterns being transformed globally? *Obes Res* 13:1281-1299.
- Assah FK, Ekelund U, Brage S, Wright A, Mbanya JC, Wareham NJ. 2011. Accuracy and validity of a combined heart rate and motion sensor for the measurement of free-living physical activity energy expenditure in adults in Cameroon. *Int J Epidemiol* 40:112-120.
- Azcorra H, Dickinson F, Rothenberg SJ. 2009. Family Migration and Physical Growth in Merida, Yucatan, Mexico. *Am J Hum Biol* 21:398-400.
- Balas-Nakash M, Benitez-Arciniega A, Perichart-Perera O, Valdes-Ramos R, Vadillo-Ortega F. 2010. The effect of exercise on cardiovascular risk markers in Mexican school-aged children: comparison between two structured group routines. *Salud Publica Mex* 52:398-405.

- Barquera S, Hernandez-Barrera L, Tolentino ML, Espinosa J, Ng SW, Rivera JA, Popkin BM. 2008. Energy Intake from Beverages Is Increasing among Mexican Adolescents and Adults. *J Nutr* 138:2454-2461.
- Bell LM, Watts K, Siafarikas A, Thompson A, Ratnam N, Bulsara M, Finn J, O'Driscoll G, Green DJ, Jones TW and others. 2007. Exercise alone reduces insulin resistance in obese children independently of changes in body composition. *J Clin Endocrinol Metab* 92:4230-4235.
- Biddle SJ, Gorely T, Stensel DJ. 2004. Health-enhancing physical activity and sedentary behaviour in children and adolescents. *J Sports Sci* 22:679-701.
- Bogin B, Loucky J. 1997. Plasticity, political economy, and physical growth status of Guatemala Maya children living in the United States. *Am J Phys Anthropol* 102:17-32.
- Brage S, Brage N, Franks PW, Ekelund U, Wareham NJ. 2005. Reliability and validity of the combined heart rate and movement sensor Actiheart. *Eur J Clin Nutr* 59:561-570.
- Caballero B. 2007. The global epidemic of obesity: an overview. *Epidemiol Rev* 29:1-5.
- Callaway C, Cameron Chumlea W, Bouchard C, Himes H, Lohman T, Martin A, Mitchell C, Mueller W, Roche A, Seefeldt V. 1988. Circumferences. In: Lohman T, Roche A, and Martorell R, editors. *Anthropometric standardization reference manual*. Champaign, IL: Human Kinetics Books. p 39-54.
- CamNtech. 2009. The Actiheart User Manual: v. 4.0.34. Cambridge: CamNtech Ltd. 125 p.
- Catalano P, Ehrenberg H. 2006. The short- and long-term implications of maternal obesity on the mother and her offspring. *BJOG* 113:1126-1133.
- Corder K, Brage S, Mattocks C, Ness A, Riddoch C, Wareham NJ, Ekelund U. 2007. Comparison of two methods to assess PAEE during six activities in children. *Med Sci Sports Exerc* 39:2180-2188.
- Corder K, Brage S, Wareham NJ, Ekelund U. 2005. Comparison of PAEE from combined and separate heart rate and movement models in children. *Med Sci Sports Exerc* 37:1761-1767.
- Corvalan C, Uauy R, Flores R, Kleinbaum D, Martorell R. 2008. Reductions in the energy content of meals served in the Chilean National Nursery School Council Program did not consistently decrease obesity among beneficiaries. *J Nutr* 138:2237-2243.
- Delisle HF. 2008. Poverty: the double burden of malnutrition in mothers and the intergenerational impact. *Ann N Y Acad Sci* 1136:172-184.
- Department of Health. 2004. At least five a week. In: Department of Health, Physical Activity, Health Improvement and Prevention, editor. London. Gateway reference 2389. April 29.
- Dickinson F. 1997. Desnutrición y obesidad en poblaciones Yucatecas. In: Aréchiga Viramontes J, and Bertran Vilá M, editors. *Significación Sociocultural de la Variación Morfológica*. México, D. F.: Universidad Nacional Autónoma de México. p 69-88.
- Dumith SC, Gigante DP, Domingues MR, Kohl HW, 3rd. 2011. Physical activity change during adolescence: a systematic review and a pooled analysis. *Int J Epidemiol* 40:685-698.
- Florencio TT, Ferreira HS, Cavalcante JC, Luciano SM, Sawaya AL. 2003. Food consumed does not account for the higher prevalence of obesity among

- stunted adults in a very-low-income population in the Northeast of Brazil (Maceio, Alagoas). *Eur J Clin Nutr* 57:1437-1446.
- Fox KR, Hillsdon M. 2007. Physical activity and obesity. *Obes Rev* 8 Suppl 1:115-121.
- 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. 335 p.
- Gomez LF, Parra DC, Lobelo F, Samper B, Moreno J, Jacoby E, Lucumi DI, Matsudo S, Borda C. 2007. Television viewing and its association with overweight in Colombian children: results from the 2005 National Nutrition Survey: a cross sectional study. *Int J Behav Nutr Phys Act* 4:41.
- Gordon C, Chumlea C, Roche A. 1988. Stature, recumbent length and weight. In: Lohman T, Roche A, and Martorell R, editors. Anthropometric standardization reference manual (abridged edition). Champaign, IL: Human Kinetics. p 3-8.
- Grillo LP, Siqueira AFA, Silva AC, Martins PA, Verreschi ITN, Sawaya AL. 2005. Lower resting metabolic rate and higher velocity of weight gain in a prospective study of stunted vs nonstunted girls living in the shantytowns of Sao Paulo, Brazil. *Eur J Clin Nutr* 59:835-842.
- Harper C, Marcus R, Moore K. 2003. Enduring poverty and the conditions of childhood: Lifecourse and intergenerational poverty transmissions. *World Dev* 31:535-554.
- Harrison G, Buskirk E, Carter J, Johnston F, Lohman T, Pollock M, Roche A, Wilmore J. 1988. Skinfold thicknesses and measurement techniques. In: Lohman T, Roche A, and Martorell R, editors. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics Books. p 55-70.
- Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM, Parra-Cabrera S. 1999. Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico city. *Int J Obes Relat Metab Disord* 23:845-854.
- Hoffman DJ, Martins PA, Roberts SB, Sawaya AL. 2007. Body fat distribution in stunted compared with normal-height children from the shantytowns of Sao Paulo, Brazil. *Nutr* 23:640-646.
- Hoffman DJ, Roberts SB, Verreschi I, Martins PA, de Nascimento C, Tucker KL, and Sawaya AL. 2000a. Regulation of energy intake may be impaired in nutritionally stunted children from the shantytowns of Sao Paulo, Brazil. *J Nutr* 130:2265-2270.
- Hoffman DJ, Sawaya AL, Coward WA, Wright A, Martins PA, de Nascimento C, Tucker KL, Roberts SB. 2000b. Energy expenditure of stunted and nonstunted boys and girls living in the shantytowns of Sao Paulo, Brazil. *Am J Clin Nutr* 72:1025-1031.
- Huneault L, Mathieu ME, Tremblay A. 2011. Globalization and modernization: an obesogenic combination. *Obes Rev* 12:e64-72.
- Katzmarzyk PT, Mason C. 2009. The physical activity transition. *J Phys Act Health* 6:269-280.
- Leatherman TL, Goodman A. 2005. Coca-colonization of diets in the Yucatan. *Soc Sci Med* 61:833-846.
- Leatherman TL, 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.

- Leonard WR, Sorensen MV, Mosher MJ, Spitsyn V, Comuzzie AG. 2009. Reduced Fat Oxidation and Obesity Risks Among the Buryat of Southern Siberia. *Am J Hum Biol* 21:664-670.
- Lohman TG, Caballero B, Himes JH, Hunsberger S, Reid R, Stewart D, Skipper B. 1999. Body composition assessment in American Indian children. *Am J Clin Nutr* 69:764S-766S.
- Malina RM, Pena Reyes ME, Tan SK, Little BB. 2011. Physical fitness of normal, stunted and overweight children 6-13 years in Oaxaca, Mexico. *Eur J Clin Nutr* 65:826-834.
- Malina RM, Reyes ME, Tan SK, Little BB. 2008. Physical activity in youth from a subsistence agriculture community in the Valley of Oaxaca, southern Mexico. *Appl Physiol Nutr Metab* 33:819-830.
- Mardones F, Villarreal L, Karzulovic L, Barja S, Arnaiz P, Taibo M, Mardones-Restat F. 2008. Association of perinatal factors and obesity in 6-to 8-year-old Chilean children. *Int J Epidemiol* 37:902-910.
- Martins PA, Hoffman DJ, Fernandes MTB, Nascimento CR, Roberts SB, Sesso R, Sawaya AL. 2004. Stunted children gain less lean body mass and more fat mass than their non-stunted counterparts: a prospective study. *Brit J Nutr* 92:819-825.
- Mendez MA, Monteiro CA, Popkin BM. 2005. Overweight exceeds underweight among women in most developing countries. *Am J Clin Nutr* 81:714-721.
- Monda KL, Adair LS, Zhai F, Popkin BM. 2008. Longitudinal relationships between occupational and domestic physical activity patterns and body weight in China. *Eur J Clin Nutr* 62:1318-1325.
- Monda KL, Gordon-Larsen P, Stevens J, Popkin BM. 2007. China's transition: The effect of rapid urbanization on adult occupational physical activity. *Soc Sci Med* 64:858-870.
- Monden CWS, Smits J. 2009. Maternal Height and Child Mortality in 42 Developing Countries. *Am J Hum Biol* 21:305-311.
- Ogden CL, Li Y, Freedman DS, Borrud LD, Flegal KM. 2011. Smoothed Percentage Body Fat Percentiles for U.S. Children and Adolescents, 1999-2004. *Natl Health Stat Report* 43:1-7.
- Onywera VO. 2010. Childhood obesity and physical inactivity threat in Africa: strategies for a healthy future. *Glob Health Promot* 17:45-46.
- Parra DC, Lobelo F, Gomez LF, Rutt C, Schmid T, Brownson RC, Pratt M. 2009. Household motor vehicle use and weight status among Colombian adults: Are we driving our way towards obesity? *Prev Med* 49:179-183.
- Popkin BM, Adair LS, Ng SW. 2012. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev* 70:3-21.
- Popkin BM, Lu B, Zhai F. 2002. Understanding the nutrition transition: measuring rapid dietary changes in transitional countries. *Public Health Nutr* 5:947-953.
- Sauri Bazán MC. 2003. Publicidad televisiva, hábitos alimentarios y salud en adolescentes de la ciudad de Mérida Mérida, México: Centro de Investigación y de Estudios Avanzados del I. P. N.
- Schofield WN. 1985. Predicting basal metabolic rate, new standards and review of previous work. *Hum Nutr Clin Nutr* 39:5-41.
- Smith PK, Bogin B, Varela-Silva MI, Loucky J. 2003. Economic and anthropological assessments of the health of children in Maya immigrant families in the US. *Econ Hum Biol* 1:145-160.

- Soares-Wynter SY, Walker SP. 1996. Resting metabolic rate and body composition in stunted and nonstunted children. *Am J Clin Nutr* 64:137-141.
- Varela Silva MI, Dickinson F, Wilson H, Azcorra H, Griffiths P, Bogin B. 2012. The nutritional dual-burden in developing countries. How is it assessed and what are the health implications? *Coll Antropol* 39-45.
- Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, Sachdev HS. 2008. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* 371:340-357.
- Walker SP, Chang SM, Powell CA. 2007. The association between early childhood stunting and weight status in late adolescence. *Int J Obes (Lond)* 31:347-352.
- Ward DS, Evenson KR, Vaughn A, Rodgers AB, Troiano RP. 2005. Accelerometer use in physical activity: best practices and research recommendations. *Med Sci Sports Exerc* 37:S582-588.
- Wilson H, Dickinson F, Griffiths P, Bogin B, Varela-Silva MI. 2011. Logistics of using the Actiheart physical activity monitors in urban Mexico among 7- to 9-year-old children. *Am J Hum Biol* 23:426-428.
- Wren RE, Blume H, Mazariegos M, Solomons N, Alvarez JO, Goran MI. 1997. Body composition, resting metabolic rate, and energy requirements of short- and normal-stature, low-income Guatemalan children. *Am J Clin Nutr* 66:406-412.

Table 4 Comparison of body composition and anthropometric measurements of included and excluded urban Maya children

| | Included | Excluded | All |
|------------------------------------|-----------------|-----------------|---------------|
| N (%) | 33 | 25 | 58 (100) |
| Boys, n (%) | 17 (51.5) | 14 (56.0) | 31 (53.4) |
| Age | 8.34 (0.82) | 8.52 (0.75) | 8.42 (0.79) |
| Stature (cm) | 121.41 (6.96) | 123.08 (6.64) | 122.13 (6.82) |
| Stature z-score¹ | -1.19 (0.92) | -1.10 (0.79) | -1.15 (0.86) |
| Weight (kg) | 26.14 (6.96) | 27.85 (5.76) | 26.87 (6.47) |
| Weight z-score¹ | -0.52 (0.89) | -0.35 (0.88) | -0.45 (0.88) |
| BMI (kg/m²) | 17.48 (2.97) | 18.28 (3.19) | 17.83 (3.07) |
| BMI z-score¹ | 0.45 (0.90) | 0.68 (1.01) | 0.56 (0.95) |
| WC (cm) | 58.90 (7.87) | 60.93 (7.70) | 59.77 (7.80) |
| WC z-score¹ | 0.28 (0.75) | 0.43 (0.84) | 0.34 (0.79) |
| FFM (kg)² | 18.43 (2.96) | 19.58 (2.83) | 18.96 (2.95) |
| FM (kg)² | 7.71 (4.40) | 8.27 (3.32) | 8.00 (3.97) |
| %FFM² | 72.19 (7.53) | 71.38 (6.31) | 71.84 (6.98) |
| %BF² | 27.81 (7.53) | 28.62 (6.31) | 28.23 (7.03) |

¹Age and sex specific z-scores based upon NHANES III using Frisancho's Comprehensive reference (2008).

²Calculated using an equation for American Indian children including bioelectrical impedance and anthropometry (Lohman et al 1999).

No significant differences found between included and excluded using independent t-tests

Table 5 Anthropometric and body composition variables for urban Mexican Maya children

| | Stunted | | Non-Stunted | | All |
|--------------------------------------|---------------|---------------|---------------|---------------|---------------|
| | Boys | Girls | Boys | Girls | |
| N (%) | 5 (15.2) | 6 (18.2) | 12 (36.4) | 10 (30.3) | 33 (100) |
| Age | 8.24 (0.93) | 8.27 (0.96) | 8.20 (0.86) | 8.60 (0.71) | 8.34 (0.82) |
| Stature (cm)^a | 114.46 (4.25) | 115.23 (7.91) | 123.47 (4.22) | 126.12 (5.02) | 121.41 (6.96) |
| Stature z-score^{2,a} | -2.38 (0.53) | -2.02 (0.45) | -0.71 (0.68) | -0.66 (0.57) | -1.19 (0.92) |
| Weight (kg)^a | 22.56 (3.27) | 20.89 (4.77) | 26.15 (5.11) | 31.05 (8.37) | 26.14 (6.96) |
| Weight z-score^{2,a} | -1.17 (0.58) | -1.17 (0.41) | -0.49 (0.85) | 0.17 (0.79) | -0.52 (0.89) |
| BMI (kg/m²) | 17.13 (1.50) | 15.60 (1.91) | 17.06 (2.53) | 19.28 (3.79) | 17.48 (2.98) |
| BMI z-score² | 0.48 (0.58) | -0.25 (0.73) | 0.44 (0.91) | 0.87 (0.95) | 0.45 (0.90) |
| FFM^{3,a} | 16.51 (1.84) | 15.88 (3.13) | 18.98 (1.79) | 20.24 (3.12) | 18.43 (2.96) |
| %FFM³ | 73.81 (7.49) | 76.37 (3.71) | 73.79 (7.18) | 66.96 (7.69) | 7.71 (4.40) |
| FM^{3,a} | 6.05 (2.46) | 5.01(1.81) | 7.17 (3.75) | 10.81 (5.45) | 72.19 (7.53) |
| %BF³ | 26.19 (7.50) | 23.63 (3.71) | 26.21 (7.18) | 33.04 (7.69) | 29.81 (7.53) |

¹Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference using NHANES III (2008).

²Age and sex specific z-scores based upon Frisancho's Comprehensive reference (2008).

³Calculated using an equation for American Indian children including bioelectrical impedance and anthropometry (Lohman et al 1999).

^aSignificant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.01

Table 6 Energy expenditure variables for urban Mexican Maya children

| | | Stunted | | Non-Stunted | | All |
|--|------------------------------|---------------------|---------------------|-----------------------|----------------------|----------------------|
| | | Boys | Girls | Boys | Girls | |
| Resting energy expenditure² | kJ/day^b | 4221.00 (315.23) | 3813.17 (384.53) | 4542.25 (437.84) | 4615.90 (615.36) | 4383.33 (547.15) |
| | kJ/kg/day^b | 188.89 (15.02) | 185.99 (18.34) | 176.31 (15.05) | 153.24 (19.44) | 172.98 (21.50) |
| Activity energy expenditure² | kJ/day^b | 2022.20 (257.92) | 2340.50 (425.36) | 3711.67 (955.11) | 3411.60 (799.06) | 3272.27 (879.21) |
| | kJ/kg/day^d | 135.97 (15.19) | 114.26 (13.51) | 143.10 (32.08) | 112.09 (18.51) | 127.38 (26.78) |
| Total energy expenditure² | kJ/day^b | 8060.40 (594.34) | 6859.83 (858.29) | 9,171.00 (1393.84) | 8919.50 (1487.21) | 8506.30 (1485.79) |
| | kJ/kg/day^d | 360.95 (32.28) | 333.62 (30.23) | 354.90 (42.56) | 294.81 (36.81) | 33373 (45.13) |
| MET (min)³ | Light | 1327.00 | 1380.33 | 1275.92 | 1323.10 | 1316.94 |
| | (<3)^{a,c} | (29.65) | (40.81) | (70.63) | (52.93) | (65.46) |
| | Mod-Vig | 113.60 | 60.00 | 164.25 | 116.70 | 123.21 |
| | (≥3)^{a,c} | (29.84) | (40.09) | (70.30) | (52.74) | (65.21) |

¹Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference using NHANES III (2008).

²Calculated in the Actiheart software using simultaneous heart rate and accelerometry data and an external reference curve (Corder 2005).

³Average number of minutes per day spent at each MET level

^a Significant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.05

^b Significant difference found between stunted and non-stunted children using an independent *t*-test, *p*<0.01

^c Significant difference found between the sexes using an independent *t*-test, *p*<0.05

^d Significant difference found between the sexes using an independent *t*-test, *p*<0.01

Appendices

Table 7 Estimated measures of energy expenditure predicted by absolute fat free mass, stature and sex in urban Mexican Maya children using multiple linear regression

| | Resting energy expenditure (kJ/day) | | | | | | Activity energy expenditure (kJ/day) | | | | | | Total energy expenditure (kJ/day) | | | | | |
|-----------------------------------|-------------------------------------|--------|---------------------|--------|---------------------|--------|--------------------------------------|--------|----------------------|-------|---------------------|-------|-----------------------------------|--------|----------------------|--------|----------------------|--------|
| | Model 1 | | Model 2 | | Model 3 | | Model 1 | | Model 2 | | Model 3 | | Model 1 | | Model 2 | | Model 3 | |
| | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P | B (SE) | P |
| Constant | 1297.51 (264.34) | <0.001 | 1675.07 (367.88) | <0.001 | 1716.65 (338.84) | <0.001 | -10.10 (798.22) | 0.990 | 1526.13 (1078.69) | 0.167 | 1646.29 (994.72) | 0.109 | 1430.70 (1087.01) | 0.198 | 3556.42 (1465.73) | 0.021 | 3736.13 (1323.30) | 0.009 |
| FFM (kg)^{1,2} | 167.48 (14.17) | <0.001 | 152.23 (17.46) | <0.001 | 154.77 (16.09) | <0.001 | 178.15 (42.79) | <0.001 | 116.08 (51.18) | 0.031 | 123.42 (47.33) | 0.014 | 384.02 (58.27) | <0.001 | 298.14 (69.55) | <0.001 | 309.11 (62.84) | <0.001 |
| Height z-score³ | | | 81.35 (56.13) | 0.158 | 78.29 (51.65) | 0.140 | | | 330.99 (164.57) | 0.053 | 322.15 (151.93) | 0.043 | | | 458.00 (223.62) | 0.049 | 444.79 (201.71) | 0.036 |
| Sex⁴ | | | | | -189.81 (74.76) | 0.017 | | | | | -548.45 (219.92) | 0.019 | | | | | -820.22 (29.98) | 0.009 |
| R² adj | 0.813 | | 0.819 | | 0.847 | | 0.338 | | 0.397 | | 0.486 | | 0.570 | | 0.610 | | 0.683 | |
| Constant | 1297.51 (264.34) | <0.001 | 1322.66 (341.02) | 0.001 | 1310.45 (314.92) | <0.001 | -10.10 (798.22) | 0.990 | 851.06 (997.89) | 0.400 | 818.43 (937.75) | 0.390 | 1430.70 (1087.01) | 0.198 | 2415.07 (1371.99) | 0.089 | 2365.24 (1263.20) | 0.071 |
| FFM (kg) | 167.48 (14.17) | <0.001 | 166.35 (17.24) | <0.001 | 171.50 (16.05) | <0.001 | 178.15 (42.79) | <0.001 | 139.302 (50.45) | 0.010 | 153.08 (47.80) | 0.003 | 384.02 (58.27) | <0.001 | 339.62 (69.36) | <0.001 | 360.65 (64.39) | <0.001 |
| Stunted^{5,6} | | | -12.74 (106.43) | 0.906 | 22.48 (99.29) | 0.822 | | | -436.32 (311.43) | 0.171 | -342.24 (295.65) | 0.256 | | | -498.75 (428.18) | 0.253 | -355.09 (398.26) | 0.380 |
| Sex | | | | | -194.98 (78.38) | 0.019 | | | | | -520.80 (233.40) | 0.034 | | | | | -798.56 (314.40) | 0.017 |
| R² adj | 0.813 | | 0.806 | | 0.835 | | 0.338 | | 0.358 | | 0.433 | | 0.570 | | 0.575 | | 0.640 | |

¹Fat free mass expressed as kilograms of body weight

²Calculated using an equation for American Indian children including bioelectrical impedance and anthropometry

³Height-for-age z-scores calculated using the age and sex specific curves of Frisancho's Comprehensive reference (2008).

⁴Boys set as reference

⁵Non-stunted set as reference

⁶Stunted defined as height-for-age less than the 5th percentile of Frisancho's Comprehensive reference (2008).