Birth Size and Maternal Nutrition During Pregnancy Predict Blood Pressure in Filipino Adults

By Anita Shroff
Northwestern University

Abstract

An adverse uterine environment is thought to program blood pressure (BP) later in life, most likely due to fetal under-nutrition. This study examines the relationships between adult systolic blood pressure (SBP) and diastolic blood pressure (DBP), birth weight, and maternal nutritional status and dietary intake during pregnancy. This study draws on a large sample (n=1632) of Filipino young adults aged 20-22 using data from the Cebu Longitudinal Health and Nutrition Survey (CLHNS), a community-based birth cohort study in Cebu City, Philippines begun in 1983. The baseline maternal survey was given at 30 ± 4 weeks gestation and the children were surveyed at birth and through childhood into adulthood. A series of linear regression models was used to examine the relationship between SBP and DBP to birth outcomes, maternal nutritional status during pregnancy as measured by triceps skin fold thickness, and dietary intake during pregnancy while controlling for socioeconomic status, age, height, BMI of the young adults, and other confounders. Birth weight was inversely related to SBP in males, and birth weight and length were inversely related to DBP in males. Maternal triceps skin fold thickness was inversely related to SBP and DBP in males and to DBP in females. Components of the mother's diet during pregnancy had varying relationships with the young adult BP of male and female offspring. Maternal nutritional status and dietary intake during pregnancy have implications for offspring's BP regulation later in life.

Introduction

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality worldwide. In the Philippines, CVD caused 27% of all deaths in 2002 (WHO 2005). The risk factors for CVD in adulthood have been widely studied. They include high blood pressure, smoking, lack of physical activity, age, and stress (National Heart Lung and Blood Institute, 2009). More recently, studies have been examining causes of CVD that emerge during early development. These studies attempt to explain whether an individual's susceptibility to chronic disease later in life can be "programmed" by early life experiences, specifically those *in utero*. It is hypothesized that the intrauterine nutritional environment affects fetal growth, which influences the expression of cardiovascular disease later in life. Thus far, study results have been inconsistent in that they have found direct, inverse, or no relationship between birth weight or birth length and CVD. Studies examining the relationships between maternal nutrition status during pregnancy, maternal dietary intake during pregnancy, and CVD in adulthood have not gathered consistent results either. Therefore, the programming of CVD is still debatable.

In this study, we use data from the Cebu Longitudinal Health and Nutrition Survey (CLHNS). We aim to examine the relationship between size at birth and systolic blood pressure (SBP) and diastolic blood pressure (DBP) in Filipino young adults, before and after taking into account maternal nutritional status and dietary intake during pregnancy, using BP as a measure of risk for CVD. This comprehensive, longitudinal data allows us to examine the long-term effects and relationships between health, culture, and behavior. Few studies have examined human maternal dietary intake during pregnancy and its effects on long-term health. Lastly, very few longitudinal studies have taken place in countries undergoing the nutrition transition, where changing dietary intake is changing the development of chronic disease. Using the CLHNS data,

we will test the hypothesis that birth weight, maternal dietary intake, and maternal nutritional status during pregnancy predict adult offspring blood pressure.

DOHaD

Developmental Origins of Health and Disease (DOHaD) is a model that explains how CVD and early life experiences, such as birth outcomes, maternal dietary intake and nutritional status during pregnancy, could be related. Much research has shown that early life events that determine susceptibility to disease in adulthood do not occur specifically *in untero* during the period of fetal development, but can also occur throughout the period of developmental plasticity. The word "developmental" rather than "fetal" encompasses both the fetal period and the period of infancy. Secondly, DOHaD incorporates ideas about disease, and also about health (Gluckman and Hanson, 2006). DOHaD can be used in public health and health education programs and presents a new way of thinking about how early life events affect adult health and disease.

Programming can be defined as the "permanent or long-term change to the physiology, morphology, or metabolism of a fetus in response to a specific insult or stimulus at a critical period in development" (Langley-Evans et al. 1998). Thus, events early in life, such as nutritional deficiency *in utero*, play a strong role in impacting susceptibility to the metabolic syndrome which leads to chronic, non-communicable diseases later in life including non insulin-dependent diabetes mellitus, coronary heart disease, and hypertension (Harding, 2001; Philips 1998; Barker and Martyn, 1992; Kuzawa 2004). For example, cardiovascular disease could be the outcome of short-term fetal physiological adjustments to under-nutrition that are crucial to survival, but have harmful effects in life post-reproduction (Godfrey and Barker 2001).

Programming of tissues and organs occurs during a "critical period" during development when physiological changes can become fixed. During this time, fetuses are "plastic" and are subject to environmental influences (Godfrey and Barker 2001). The critical period occurs at various stages throughout embryonic and fetal development with different organs and tissues developing during different critical periods (Symonds et al. 2006).

Several ideas in relation to life history theory can explain the relationship between early life events and the development of disease in adulthood. These include the thrifty phenotype, predictive adaptive response, environmental mismatch, or developmental programming. All of these theories claim that fetuses receive signals from their environment, and adapt to their environment, which will help them better cope with environmental stressors later in life (Silveira et al. 2007). In particular, adapted fetuses will be better prepared to cope with the stressors that the mother encountered during her pregnancy about which the "signal" is conveyed to the fetus. However, if the postnatal environment differs from the predicted environment, the adaptation can actually be harmful and there is increased risk of disease. For example, low birth weight babies who are born into an affluent environment are at a higher risk for coronary heart disease, hypertension, and type 2 diabetes (Bateson et al. 2004). This possibly occurs because their bodies have been programmed metabolically for a low resource environment but they experience nutritional abundance.

Much animal model data and human epidemiological data support DOHaD, however, the exact biological mechanisms are not yet fully understood. Recently, epigenetic mechanisms to explain DOHaD have come into view. Epigenetics is the study of heritable changes in DNA caused by reasons other than alterations in the DNA sequence. These changes include histone modification, DNA methylation, and DNA-binding proteins. The epigenetic hypothesis for the

development of disease states that early environmental stressors and influences trigger epigenetic changes, which affect chronic disease risk. For example, DNA hypo- and hypermethylation has been implicated early in atherogenesis, the formation of plaques that leads to CVD (Waterland and Michels 2007).

Birth Weight and Maternal Nutrition and Diet During Pregnancy

Nutrition is a major programming stimulus and the mechanisms by which maternal under- or over-nutrition lead to low birth weight may contribute to understandings of DOHaD. Maternal under-nutrition could result from low intake of nutrients due to restricted food access or famine. Inadequate or improper maternal nutrition leads to intrauterine growth retardation (IUGR), which manifests as thinness and decreased birth length and weight, which lead to increased BP later in life. However, a distinction must be made between maternal nutrition and fetal nutrition, which is the total supply of metabolic substrates to the fetus (Harding 2001). According to Harding, fetal growth is at the end of a chain of events including maternal diet, maternal metabolic and endocrine status, placenta transport, umbilical blood flow, and finally fetal uptake. If the chain of events has a large margin of error, changes in maternal diet may not influence fetal growth strongly (Harding 2001). Hence, the fetus may be buffered from maternal environment.

Few studies have been conducted in humans using measures of maternal nutrition or maternal dietary intake to test the idea that nutrition is a programming stimulus. Animal studies have shown that decreasing protein intake during pregnancy leads to reduced birth weight and increased blood pressure later in life (Langley-Evans et al. 1998). Laura et al. used maternal anthropometric variables (pre-pregnancy weight and BMI) and weight at the end of the pregnancy as a measure of maternal nutrition to show that these variables have positive

associations with SBP and DBP in male and female adolescents in a developing country (Laura et al. 2010). Adair et al. found that higher SBP and DBP in adolescent boys are associated with low maternal fat stores at around 30 weeks gestation. Further, SBP in adolescent boys was inversely related to the percent of the mother's energy intake from protein during late gestation and SBP and DBP were inversely related to mother's percentage of energy intake from fat during pregnancy (Adair et al. 2001).

Birth Weight and Blood Pressure

Human studies on the relationship between birth weight and blood pressure have obtained varied results. A study of 49-year old Swedish men found that adult systolic blood pressure (SBP) was not correlated with birth weight (Siewert-Delle and Ljungman 1998). Another study comparing SBP to either full term or pre term birth weight at 2.5 years found the same result (Bracewell et al. 2007). Conversely, others have found an inverse relationship between birth weight and blood pressure (Davies et al. 2006; Gamborg et al. 2007), including research from the Guangzhou Biobank Cohort Study in a developing Chinese population (Schooling et al. 2010). Further, the Bogalusa Heart Study, a longitudinal study conducted on 16,000 adults and children in Bogalusa, Louisiana, showed that low birth weight was associated with higher SBP in adolescence, and that association was amplified with age from adolescence to adulthood, even after adjusting for race and BMI (Chen et al. 2010). On the other hand, other studies have found no association between birth weight and BP, especially when BP is measured in infancy (Hindmarsh et al. 2010) or in adolescence (Falkner et al. 2003). Thus, findings regarding relationships between birth weight and BP are inconsistent.

Nutrition Transition

This study sheds light on the relationships between health, nutrition and cultural factors. The nutrition transition refers to the range of sociocultural, demographic, as well as biological effects of globalization, which cause a shift in diet and physical activity patterns. In countries undergoing the nutrition transition, diet shifts towards a higher fat and meat content and reduced carbohydrate and fiber content. Physical activity shifts from labor intense agricultural activities to more manufacturing and service jobs, both of which are more sedentary (Popkin 1997). As a result of the transition, people are consuming more energy and activity is becoming idler. These shifts are appealing, but have detrimental effects on health.

The changes in diet and physical activity have caused the incidence and prevalence of obesity to increase, especially in middle- and low-income countries. In 2004, Adair found that in Cebu, the rate of overweight and obesity increased from 6% in 1983-1984 to 35% in 1998-1999, an approximately six-fold increase. This increase corresponds with economic changes exhibited by increased household income and ownership of consumer goods, a shift to foods higher in fat, a reduced burden of household work, and more sedentary jobs. The incidence and prevalence of obesity and overweight is highest in higher SES households, but is increasing in poorer households also.

Increases in income and assets affect diet and activity patterns. In Cebu, women learn skills to set up their own businesses, and one in seven women own their own “sari-sari” shops. The work is mostly sedentary and there is high access to snack foods. Work away from home is associated with higher weight gain in this population. Furthermore, income increases with increased work hours, and higher income is related to dietary changes, such as eating foods with higher fat content (Popkin 2003). Thus, this population is undergoing the nutrition transition.

In Cebu, it was found that increasing socioeconomic status and urbanicity is directly associated with a more obesogenic diet. Urbanicity is rated on an "urbanicity index" and the most urban community would have the greatest population size and density, most communication, transportation, healthcare services, education and market availability (Kelles and Adair 2009). The resulting weight gain due to these changes in Cebu is associated with health consequences such as obesity and risk for hypertension (Popkin 2003). The health trend among Cebu women is consistent with the same trend in other developing countries where increasing obesity is significantly directly associated with BP (Adair 2004). Thus, this population is more likely to develop metabolic syndrome, the metabolic risk factors that predispose individuals to CVD and diabetes, than if globalization and the transition had not occurred.

The nutrition transition can take place in a single lifetime if the individual is born into a pre-westernized diet and physical activity pattern but shifts to a diet higher in fatty foods and low physical activity. When individuals go through the nutrition transition during their life, they are at an especially increased risk for noncommunicable chronic diseases (NCD's). Such individuals encounter a "dual burden". They are underweight during infancy and childhood, but as they undergo the nutrition transition, they are at a higher risk of developing NCD's. The dual burden individuals are actually more likely to develop metabolic syndrome than those who have not undergone a transition, possibly due to DOHaD. The individual as a fetus and an infant adjusts to a "pre-westernized" diet and the changeover to a diet high in fat and processed or fast food distresses the body. It is common for under-nutrition and over-nutrition to coexist in populations experiencing the nutrition transition (Doak et al. 2005; Popkin 2009). Our findings support this idea that the impacts of cultural and dietary change, which are underway in the Philippines and

many similar places around the world, will be worse for individuals who began life with poorer nutrition. Thus, poor early nutrition can exacerbate the negative impacts of gaining weight later in life.

Materials and Methods

Survey Design and Sample

The data for this project come from the Cebu Longitudinal Health and Nutrition Survey (CLHNS), an ongoing, community-level survey that follows a cohort of 3080 women and their infants born between 1983 and 1984. All pregnant women were invited to participate in the surveys as long as they gave birth between that interval. The refusal rate was very low (<3%), and there are no data on those mothers who refused to participate. The women lived in 33 randomly selected communities in Metro Cebu, the second largest metropolitan area of the Philippines. The communities include urban neighborhoods and more isolated rural villages (Adair et al. 2001). The CLHNS data were collected from 1983 to 2007 and the data for this analysis come from data collected from mothers during their third trimester of pregnancy (30±4 weeks in 1983-1984), from their offspring at birth (1983-1984), and from the 2005 survey, when the offspring were between 20 and 22 years of age. Complete blood pressure, anthropometric, environmental, and sociodemographic data were available for 1632 of the initial 3080 infants. Informed consent was obtained from all study participants and the use of human subjects was approved by the institutional review boards at Northwestern University and University of North Carolina.

Data Collection

At baseline, trained interviewers took mothers' anthropometric measures, and mothers were asked to complete a single 24-hour dietary recall. Triceps skin fold thickness and dietary

intakes of protein, fat, and total energy intake at about 30 weeks of gestation were used as measures of maternal nutritional status during pregnancy. The Philippines Food Composition Tables were used to calculate energy and nutrient intakes. The mother's percent of energy from protein, and total energy intake were used in the models. Percent of energy from fat and carbohydrates were not used in the models due to colinearity with the other measures of dietary intake.

Infant length at birth was measured less than 6 days after birth by trained staff with custom length boards. Infants born in hospitals were measured with hospital scales and birth attendants who were trained in the use of hanging Salter scales measured infants born at home. Gestational age was estimated from the mother's last menstrual period. Body mass index (BMI) was calculated to measure relative weight (Adair et al. 2001).

Anthropometric measurements of the young adults' weight and height, arm, hip, and waist circumferences, and triceps and subscapular skin folds were taken using standard anthropometric techniques (Lohman et al. 1988). The young adults were asked to do two 24-hour dietary recalls on consecutive days. The mean of the recalls was used in the analysis. The young adults' percent of energy from fat, and total energy intake were included in the models. Physical activity was not included in the models because it was not accurately measured. The young adults were asked if they currently smoked, and if they had smoked in the past. Systolic and diastolic blood pressure was measured three times for each individual using a mercury sphygmomanometer and the mean was used in the analysis. The mother's blood pressure in 2005 was measured by the same interviewer, but it was not measured at baseline. Socioeconomic status of the household was estimated by total deflated household income and the possession of household assets, such as air conditioning, or an electric fan. Socioeconomic

variables are not directly related to blood pressure, but affect it more indirectly through other factors such as nutritional status and diet (Adair et al. 2001). Individuals who were not included in this analysis due to missing data did not have significantly different measures of birth length, weight, and blood pressure than those that were included.

Analysis Methods

All analyses were conducted using version 10 of Stata. Here, we stratify our analyses on sex based on previous reports that fetal programming differentially affects male and female fetuses. The outcome variables, SBP and DBP, were treated as continuous variables by ordinary least-squares regression. Variables that were not normally distributed (mother's triceps skin fold thickness, adult offspring's current energy intake, and deflated household income) were log transformed to create a normal distribution. Offspring's sex, offspring's smoking, and mother's vitamin use were treated as nominal variables. Offspring's birth weight (kg), birth length (cm), and current age, height (cm), BMI (kg/m^2), energy intake (kcal), and percent of energy from fat were treated as continuous variables as were mother's triceps skin fold thickness (mm), height (cm), energy intake during pregnancy (kcal), and percent of energy from protein during pregnancy.

A correlation matrix was created to detect any relationships between the outcome and predictor variables (Tables 2 and 3). Upon inspecting the table, we see that SBP and DBP are positively correlated with offspring's adulthood height and BMI, as expected. Thus, during regression analysis, BMI and weight should be included as controls to better understand the relationship between birth weight and adulthood BP.

A series of regression models were created to elucidate the relationships between the outcome and predictor variables. In model one, the relationship between birth outcomes (birth

weight and birth length) and young adults' SBP and DBP were examined while controlling for adulthood age, height, and BMI. Model two adds maternal anthropometric variables during pregnancy (height and triceps skin fold thickness). Model three adds variables measuring maternal dietary intake during pregnancy (total energy intake, percent of energy from protein, and vitamin use). Model four adds variables measuring the young adult's current diet (total energy intake and percent of energy from fat). Model five adds the young adult's lifestyle variables and measures of socioeconomic status (smoking, household income, urbanicity, and assets). Model six adds the mother's current BP.

Results

Table 1 presents descriptive characteristics of the study participants. Compared to age-matched US men (McDowell et al. 2005), the Cebu young men in this sample were shorter (mean height: 162.95 cm vs. 176.0 cm in Cebu and US, respectively) and thinner (mean BMI: 20.99 kg/m^2 vs. 27.8 kg/m^2). Compared to age-matched US women, the Cebu young women in this sample also were shorter (mean height: 151.17 cm vs. 162.10 cm) and thinner (mean BMI: 20.39 kg/m^2 vs. 28.20 kg/m^2). In this sample, 97.05% of males and 97.17% of females had a BMI below the age- and sex-specific median of the US. A total of 11.33% of males and 13.76% of females reported smoking currently, and 8.85% of males and 10.83% of females were born at a low birth weight (<2.5 kg). Prepregnancy BMI data was not collected. The upper limit of a normal, healthy SBP is 120 mm Hg and 15.94% of males and 2.55% of females are above that limit. The upper limit of a normal, healthy DBP is 80 mm Hg and 24.79% of males and 5.48% of females are above that limit.

There were some significant univariate correlations between birth weight and SBP or DBP in males and females. Tables 2 and 3 present correlation matrices for modeled variables and

potential confounders. It is not uncommon for many variables to be intercorrelated. For example, income may be in the pathway of nutritional status as measured by triceps skin fold thickness and height, or BMI, which would cause correlations. Gestational age was taken out of the models because it had no significant main effect and did not modify the effects of birth weight or length. The adolescent's household socioeconomic status and lifestyle factors were not significantly associated with BP and did not change the relationship of the maternal or birth characteristics to BP.

Systolic BP

In males, there was a significant inverse relationship between birth weight and SBP after controlling for age, height, and BMI (Table 4, model 1). In males, lower birth weight predicts higher adulthood blood pressure. Furthermore, lower birth weight in combination with a higher adult BMI puts the individual at an even greater risk for elevated SBP in adulthood (Figure 1). SBP was significantly associated with age, height and BMI in males and with BMI and height in females. In females, there was no significant relationship between birth outcomes and SBP (Table 5, model 1). Mother's triceps skin fold thickness during pregnancy was significantly inversely related to SBP in males but mother's height did not affect the model (Table 4, model 2). The relationship between SBP and birth weight slightly lessened when variables of maternal nutritional status during pregnancy were added. Maternal anthropometric variables during pregnancy were not related to SBP in females (Table 4, model 2).

Relationships between maternal diet during pregnancy and SBP differed in males and females. In males, the mother's total energy intake was directly associated with SBP and the mother's percent of energy from protein was inversely related to SBP (Table 4, model 3). In females, the mother's percent of energy from protein was directly associated with SBP, and the

mother's vitamin use was inversely related to SBP (Table 5, model 3). When birth weight and birth length were removed from the models (not shown), the relationship of the mother's height and mother's skin fold thickness during pregnancy to SBP was approximately unchanged in males and females. Measures of the male's current diet were unrelated to SBP, but females' total energy intake currently was significantly associated with SBP (Table 5, model 4). As stated above, current lifestyle factors and socioeconomic status were not related to SBP (Tables 4 and 5, model 5). Mother's current SBP was significantly related to SBP in males and females (Tables 4 and 5, model 6).

Diastolic BP

In males there was a significant inverse relationship of birth weight and birth length to DBP after controlling for age, height, and BMI (Table 6, model 1). In males, lower birth weight predicts higher adulthood blood pressure. Furthermore, a lower birth weight in combination with a higher adult BMI puts the individual at an even greater risk for elevated DBP in adulthood (Figure 2). DBP was significantly associated with age and BMI in males and with BMI and height in females. In females, there was no significant relationship between birth outcomes and DBP (Table 7, model 1). In males and females, the mother's triceps skin fold thickness during pregnancy was significantly inversely related to DBP but mother's height did not affect the model (Table 6, model 2). In males, the relationship between DBP and birth weight slightly lessened when variables of maternal nutritional status during pregnancy were added (Table 6, model 2).

In males, there was no significant relationship between maternal diet during pregnancy and DBP or between the male's current diet and DBP (Table 6, models 3 and 4). In females, there was a significant association between the mother's percent of energy from protein during

pregnancy and DBP, and between the female's current total energy intake and DBP (Table 7, models 3 and 4). Lifestyle factors and socioeconomic status were not related to DBP in males or females (Tables 6 and 7, model 5). Mother's current DBP was significantly related to current DBP in males and females (Tables 6 and 7, model 6).

Discussion

We found an inverse relationship between birth weight and SBP and DBP in males and also between maternal nutritional status during pregnancy and SBP and DBP in males. The effects of the composition of the mother's diet during pregnancy varied between males and females. Also, the mother's current BP was positively associated with the adult offspring's BP. These results suggest that birth weight is a significant predictor for adulthood blood pressure and also provide some evidence for fetal programming of blood pressure.

Consistent with other human studies (Davies et al. 2006; Gamborg et al. 2007; Schooling et al. 2010) this analysis found an inverse relationship of birth weight to SBP in males and an inverse relationship of birth weight and birth length to DBP in males in a sample of 847 males. When birth weight was separated into quartiles and adjusted for confounders, the lowest quartile of birth weight predicted the highest SBP or DBP in adulthood (Figures 3 and 4), which is consistent with literature (Primatesta et al. 2005; Levitt et al. 1999).

After controlling for potential confounding factors, we found a strong inverse relationship between birth weight and SBP and DBP. This is a much more robust change in comparison to the change in a similar age group in the US (Huxley et al. 2002; Davies et al. 2006). This phenomenon is not well understood but could be due to differing components of dietary intake in pregnant mothers in Cebu versus the US or due to differential access to nutritious foods. An analysis similar to this one conducted on the same cohort of individuals when they were

adolescents also found an inverse relationship between birth weight and SBP, but the relationship is weaker than the one we report (Adair et al, 2001). Hence, the findings of this analysis suggest that the inverse relationship between birth weight and SBP is sustained from adolescence through adulthood and that the relationship between birth weight and BP amplifies with age.

Human studies examining maternal nutritional status during pregnancy are few. In a large study of Israeli youth, Laor et al. found that maternal anthropometric variables during pregnancy, including maternal BMI and weight gain, were not related to the offspring's BP at age 17 (Laor et al. 1997), but Godfrey et al found an inverse relationship between mother's triceps skin fold thickness and SBP in a sample of 10-12 year old Jamaican children (Godfrey et al. 1994). Similarly, the study done by Adair and Kuzawa on this same sample also found an inverse relationship between maternal fat stores and SBP and DBP in boys (Adair et al. 2001). Consistent with the Adair finding, this study also uncovered an inverse relationship between mother's triceps skin fold thickness to SBP in males, and to DBP in males and females. This suggests that maternal nutrition status is a fundamental programming stimulus.

As shown in animals through dietary restriction, poor maternal nutrition, which generates an unsatisfactory intrauterine environment, is a causal factor in programming and can lead to higher BP later in life. Animal studies have found that decreasing the amount of protein during pregnancy leads to reduced birth weight and increased blood pressure later in life (Langley-Evans 2001; Woods et al. 2001). Thus, maternal nutrition may be a prime programming stimulus. Our analysis included the mother's total energy intake and the percent of energy intake from protein in the diet during pregnancy. In males, SBP was directly related to the mother's total energy intake and inversely related to the mother's percent of energy intake from protein, which is consistent with the animal model above. In females, SBP and DBP were directly

related to the mother's percent of energy intake from protein, and the mother's vitamin use during pregnancy was inversely related to SBP.

A study by Shiell et al found that Scottish mothers who consumed a high meat, low carbohydrate diet during the second half of their pregnancies had offspring with elevated SBP at 27 to 30 years of age (Shiell et al. 2001). This is consistent with the findings of Campbell et al that a high protein, low carbohydrate diet led to higher SBP (Campbell et al, 1996). In this analysis, the relationships of females' SBP and DBP to protein are consistent with the Shiell and Campbell findings. However, Thone-Reineke et al. found that high maternal protein intake during pregnancy leads to higher SBP in boys, not girls (Thone-Reineke et al. 2006). Thus, human studies of the relationship between maternal protein intake and offspring are inconclusive. This issue would benefit from investigation of the mechanisms, rather than just epidemiological evaluation.

Maternal total energy intake during pregnancy was unrelated to SBP in females and to DBP in both females and males. This proposes that dietary composition may affect fetal programming more than total energy intake. Roseboom et al found similar results in a study of Dutch adults (Roseboom et al. 2001). Decrease in SBP was dependent on an increase in the protein to carbohydrate ratio during the mother's third trimester of pregnancy. Thus, the balance of macronutrient intake influences programming more strongly than total energy intake.

The finding that maternal vitamin use during pregnancy is inversely related to SBP in females could suggest that micronutrient composition of the mother's diet is also playing a role in fetal programming. However, this assumption is limited because since the measures of dietary intake are only based on one 24-hour recall, vitamin use is not a very strong indicator of frequency of use throughout the pregnancy. Furthermore, data about the micronutrient content

of the vitamins were not collected. The role of micronutrient composition of the mother's diet during pregnancy deserves further investigation, particularly because certain micronutrients have a place in the pathway towards cardiovascular disease.

The results of this analysis show that in females, current energy intake has a positive relationship with SBP and DBP. But in both instances, BP also has a positive relationship with the mother's percent of dietary intake from protein during pregnancy. Accordingly, there may be a complex relationship between current diet and fetal programming which compound to elevate blood pressure later in life. However, a closer examination of the beta coefficients from the linear regression analyses shows that female's current total energy intake is a stronger predictor of SBP than the mother's percent of energy intake from protein and it is also more significant (Table 5). When looking at the beta coefficients that predict DBP in females, the coefficient for the female's current total energy intake is still a stronger predictor, but is slightly less significant (Table 7). Thus, in females, current energy intake has a greater influence on BP than maternal dietary composition during pregnancy. This is consistent with the finding that in females, birth weight does not predict blood pressure, and overall, fetal programming is not seen in females in this population.

This study revealed distinct sex differences in the relationship between birth outcomes and BP in adulthood. Recently, studies at the molecular level have begun to uncover the mechanisms connecting birth weight and hypertension. Nephron number is linked to the expression of hypertension, and autopsies have shown that patients who had hypertension had fewer glomeruli in their kidneys. A reduction in nephron number can originate *in utero* due to an adverse uterine environment, and could cause hypertension later in life (Alexander 2006). Both animal and human studies have shown that males have a higher tendency towards decreased

renal function than females, likely due to the ratio of sex steroids. Androgens, such as testosterone, enhance the progression of renal injury, whereas estrogens are protective in vivo (Gilbert and Nijland 2008).

Clinical trials have examined sex differences in renal function and programmed hypertension. A study conducted on 20 to 30 year olds in Norway found that growth restriction *in utero*, high blood pressure, and low renal function were all positively significantly associated. Furthermore, the association was stronger and more consistent in males than in females (Hallan et al. 2008). This is only one mechanism by which hypertension is programmed on a sex-specific basis. Others include utero-placental insufficiency, maternal obesity, or maternal renal compromise (Gilbert and Nijland 2008; Grigore et al. 2008). Further research into these pathways would help elucidate the pathway to programming of disease.

Mother's current BP was found to be a significant predictor of the young adult's current BP, which could suggest some genetic factors influencing BP. It is widely accepted that genetic factors play a role in the pathogenesis of hypertension, and in this post-genome generation, some potential genes that cause hypertension have been identified, such as the angiotensinogen gene (AGT) and the angiotensin converting enzyme (ACE) gene (Dominiczak et al. 2004). However, environmental factors also influence an individual's susceptibility to hypertension so the positive relationship between the mother's BP and child's BP could be a result of mutual environmental factors that were not measured in this study.

The results from this study suggest opportunities for public health interventions. If the role of maternal macronutrient and micronutrient dietary composition during pregnancy and its effects on adulthood offspring BP were elucidated, preventative measures can be initiated in order to change the mother's diet during pregnancy. Moreover, this population lives in a

developing country going through the nutrition transition. Changes in diet and physical activity caused by the nutrition are seen in birth outcomes and measures of nutritional status. The results from this study show that birth outcomes and maternal nutritional status during pregnancy have consequences for BP regulation later in life. Appropriate public health interventions could reduce the burden of non-communicable chronic diseases.

Additionally, this analysis has found similar results as the study done on this same population when the subjects were adolescents (Adair et al. 2001). In that study, low maternal energy stores were inversely related to SBP and DBP in boys, parallel to the results of this analysis. Furthermore, the relationship between birth outcomes and BP in boys was also found in this analysis, but the relationship was intensified in comparison to the adolescent study. Examining amplification of the effects of fetal programming with age merits further investigation.

In conclusion, this study supports the hypothesis that maternal nutritional status during pregnancy influences the offspring's BP regulation in adulthood. Low birth weight and low maternal nutritional status during pregnancy were inversely related to SBP and DBP in males. The composition of the mother's diet during pregnancy affected the young adult's SBP and DBP differently in males and females, but is consistent with animal and human studies. The mechanisms of fetal programming in humans and the influence of macronutrient and micronutrient intake during pregnancy on hypertension warrant further study.

References

Adair L. 2004. "Dramatic rise in overweight and obesity in adult Filipino women and risk of hypertension." *Obesity Research* 12: 1335-1341.

Adair LS, Kuzawa CW, Borja J. 2001. "Maternal energy stores and diet composition during pregnancy program adolescent blood pressure." *Circulation* 104: 1034-1039.

Alexander B.T. 2006. "Fetal programming of hypertension." *American Journal of Physiology-Regulatory, Integrative and Comparative Phsiology* 290: R1-R10.

Barker D.J.P. 1995. Fetal origins of coronary heart disease. *British Medical Journal* 311: 171-174.

Barker D.J.P, Martyn C.N. 1992. The maternal and fetal origins of cardiovascular disease. *Journal of Epidemiology and Community Health* 46: 8-11.

Barker DJP, Osmond C, Golding J, Kuh D, Wadsworth MEJ. 1989. "Growth *in untero*, blood pressure in childhood and adult life, and mortality from cardiovascular disease." *British Medical Journal* 298: 564-567.

Bateson P, Barker D, Clutton-Brock T, Deb D, D'Udine B, Foley R, Gluckman P, Godfrey K, Kirkwood T, Lahr M, McNamara J, Metcalfe N, Monaghan R, Spencer H, Sultan S. 2004. "Developmental plasticity and human health." *Nature* 430: 419-421.

Bracewell MA, Hennessy EM, Wolke D, Marlow N. 2007. "The EPICure study: growth and blood pressure at 6 years of age following extremely preterm birth." *Fetal and neonatal edition British Paediatric Association*:10.1136/adc.2007.118596.

Campbell D.M., Hall M.H., Barker D.J.P., Cross J., Shiell A.W., Godfrey K.M. 1996. "Diet in pregnancy and the offspring's blood pressure 40 years later. *British Journal of Obstetrics and Gynecology* 103: 273-280.

Cebu Longitudinal Health and Nutrition Survey. UNC Carolina Population Center. <http://www.cpc.unc.edu/projects/cebu>.

Chen W., Srinivasan S., Berenson G. 2010. "Amplification of the association between birthweight and blood pressure with age: the Bogalusa heart study." *Journal of Hypertension* 28: 2046-2052.

Danese A., Pariante C.M., Caspi A., Taylor A., Poulton R. 2007. "Childhood maltreatment predicts adult inflammation in a life-course study." *Proceedings of the National Academy of Sciences* 104: 1319-1324.

Davies A.A., Smith G.D., May M.T., Ben-Shlomo Y. 2006. "Association between birth weight and blood pressure is robust, amplifies with age, and may be underestimated." *Journal of Hypertension* 48: 431-436.

Doak C.M., Adair L.S., Bentley M., Monteiro C., Popkin B.M. 2005. "The dual burden household and the nutrition transition paradox." *International Journal of Obesity* 29: 129-136.

Dominiczak A., Brain N., Charchar F., McBride M., Hanlon N., Lee, W.K. 2004. "Genetics of hypertension: lessons learnt from mendelian and polygenic syndromes." *Clinical and Experimental Hypertension* 26: 611-620.

Falkner B., Hulman S., Kushner H. 2003. "Effect of birth weight on blood pressure and body size in early adolescence." *Journal of Hypertension* 43: 203-207.

Gamborg M., Bybert L., Rasmussen F., et al. 2007. "Birth weight and systolic blood pressure in adolescence and adulthood: meta-regression analysis of sex- and age-specific results from 20 Nordic studies." *American Journal of Epidemiology* 166: 634-645.

Gilbert J., Nijland M. 2008. "Sex differences in the developmental origins of hypertension and cardiorenal disease." *American Journal of Physiology- Regulatory, Integrative and Comparative Phsiology* 295: R1941-R1952.

Gluckman P., Hanson M. 2006. Developmental Origins of Health and Disease. Cambridge: University Press.

Godfrey K., Barker D.J.P. 2001. "Fetal programming and adult health." *Public Health Nutrition* 4(2B): 611-624.

Godfrey K.M., Forrester T., Barker D.J. 1994. "Maternal nutritional status in pregnancy and blood pressure in childhood." *Brititsh Journal of Obstetrics and Gynecology* 101: 398-403.

Grigore D., Ojeda N., Alexander B. 2008. "Sex differences in the fetal programming of hypertension." *Gender Medicine* 5, Suppl A: S121-S132.

Hallan S., Euser A.M., Irgens L.M., Finken M.J., Holmen J., Dekker F.W. 2008. "Effect of intrauterine growth restriction on kidney function at young adult age: the Nord Trondelag Health (HUNT 2) study." *American Journal of Kidney Disease* 51: 10-20.

Harding J.E. 2001. "The nutritional basis of the fetal origins of adult disease." *International Journal of Epidemiology* 30: 15-23.

Hindmarsh P., Bryan S., Geary M., Cole T. 2010. "Effects of current size, postnatal growth, and birth size on blood pressure in early childhood." *Journal of Pediatrics* doi:10.1542/peds.2010-0358.

Huxley R., Neil A., Colins R. 2002. "Unraveling the fetal origins hypothesis: is there really an inverse association between birthweight and subsequent blood pressure?" *Lancet Oncology* 360: 659-665.

Kelles A., Adair L. 2009. "Offspring consume a more obesogenic diet than mother in response to changing socioeconomic status and urbanization in Cebu, Philippines." *International Journal of Behavior Nutrition and Physical Activity* 6: 47-58.

Kramer M.S. "Isocaloric balanced protein supplementation in pregnancy (Cochrane Review). The Cochrane Library. Issue 4. Oxford: Update Software, 1999.

Kuzawa C. 2004. "Modeling fetal adaptation to nutrient restriction: testing the fetal origins hypothesis with a supply-demand model." *Journal of Nutrition* 134: 194-200.

Kuzawa C., Adair L. 2003. "Lipid profiles in adolescent Filipinos: relation to birth weight and maternal energy status during pregnancy." *American Journal of Clinical Nutrition* 77: 960-966.

Lackland D., Egan B., Ferguson P. 2007. "Low birth weight as a risk factor for hypertension." *Journal of Clinical Hypertension* 5: 133-136.

Langley-Evans S. 2001. "Fetal programming of cardiovascular function through exposure to maternal undernutrition." *Proceedings of the National Academy of Sciences* 60: 505-513.

Langley-Evans S., Gardner D., Welham S. 1998. "Intrauterine programming of cardiovascular disease by maternal nutritional status." *Journal of Nutrition* 14: 39-47.

Laor A., Stevenson D., Shelmer J., Gale R, Seidman D. 1997. "Size at birth, maternal nutritional status in pregnancy, and blood pressure at age 17: population based analysis." *British Medical Journal* 315: 449-453.

Laura H., Menezes A., Noal R., Hallal P., Araujo C. 2010. "Maternal anthropometric characteristics in pregnancy and blood pressure among adolescents: 1993 live birth cohort, Pelotas, southern Brazil." *BMC Public Health*: 10: 434-450.

Leavitt N., Steyn K., De Wet T., Morrell C., Edwards R., Ellison G., Cameron N. 1999. "An inverse relation between blood pressure and birth weight among 5 year old children from Soweto, South Africa." *Journal of Epidemiology and Community Health* 53: 264-268.

Lindsay R.S., Lindsay M.R., Edwards C.R.W., Seckl J.R. 1996. "Inhibition of 11 beta-hydroxysteroid dehydrogenase in pregnant rats and the programming of blood pressure in the offspring." *Journal of Hypertension* 27: 1200-1204.

Lohman, T. G., Roche, A. F. & Martorell, R. 1988. *Anthropometric standardization reference manual*. Human Kinetics Books.

McCance R.A., Widdowson E.M. 1974. “The determinants of growth and form.” *Proceedings of the Royal Society* 185: 1-17.

Metcoff J., Cole T.J., Luff R. 1981. “Fetal growth retardation induced by dietary imbalance of threonine and dispensable amino acids, with adequate energy and protein-equivalent intakes, in pregnant rats.” *Journal of Nutrition* 111: 1411-1424.

National Heart, Lung and Blood Institute. *Heart Disease Risk Factors.* Accessed February 2011. <http://www.nhlbi.nih.gov/health/dci/Diseases/hd/hd_risk.html>.

Pauletto P., Rattazzi M. 2006. “Inflammation and hypertension: the search for a link.”*Nephrology Dialysis and Transplantation* 21: 850-853.

Phillips D.I. 1998. “Birth weight and the future development of diabetes. A review of the evidence.” *Diabetes Care* 21: B150-55.

Pingali P. 2006. “Westernization of Asian diets and the transformation of food systems: Implications for research and policy.” *Food Policy* 32: 281-298.

Popkin B. 1997. “The nutrition transition and its health implications in lower-income countries.” *Public Health Nutrition* 1: 5-21.

Popkin B. 2003. “The nutrition transition in the developing world." *Development Policy Rev*iew 21: 581-597.

Popkin B.M. 2009. “The nutrition transition in low-income countries: an emerging crisis.:” *Nutrition Review* 52: 285-298.

Primatesta P., Falaschetti E., Poulter N. 2005. “Birth weight and blood pressure in childhood: Results from the health survey for England.” *Journal of Hypertension* 45: 75-79.

Roseboom T.J., van der Meulen J.H., van Montfrans G.S. 2001. “Maternal nutrition during gestation and blood pressure in later life.” *Journal of Hypertension* 19: 29-34.

Schooling C.M., Jiang C.Q., Lam T.H., Cowling B.J., Yeung S.A., Zhang W.S., Cheng K.K., Leung G.M. 2010. “Estimated birth weight and adult cardiovascular risk factors in a developing southern Chinese population: a cross sectional study.” *BMC Public Health* 10: 270.

Shiell A., Campbell-Brown M., Haselden S., Robinson S., Godfrey K., Barker D. 2001. “High-meat, low-carbohydrate diet in pregnancy: relation to adult blood pressure in the offspring.” *Journal of Hypertension* 38: 1282-1288.

Siewert-Delle A., Ljungman S. 1998. “The impact of birth weight and gestational age on blood pressure in adult life.” *American Journal of Hypertension* 11: 946-953.

Sigelman C., Rider E. 2009. “Life-Span Human Development.” Wadsworth: Cengage Learning.

Silveira P., Portella A., Goldani M., Barbieri M. 2007. “Developmental origins of health and disease (DOHaD).” *Journal of Pediatrics* 83: 494-504.

Symonds M., Stephenson T., Gardner S., Budge H. 2006. “Long-term effects of nutritional programming of the embryo and fetus: mechanisms and critical windows.” *Reproduction, Fertility and Development* 19: 53-63.

Thone-Reineke C., Kalk P., Dorn M., Klaus S., Simon K., Pfab T., Godes M., Persson P., Unger T., Hocher B. 2006. “High-protein nutrition during pregnancy and lactation programs blood pressure, food efficiency, and body weight of the offspring in a sex-dependent manner.” *American Journal of Physiology- Regulatory, Integrative and Comparative Physiology* 291: 1025-1030.

Tzoulaki I., Jarvelin M., Hartikainen A., et al. 2008. “Size at birth, weight gain over the life course, and low-grade inflammation in young adulthood: northern Finland 1966 birth cohort study.” *European Heart Journal* 29: 1049-1056.

McDowell M.A., Fryar C.D., Hirsch R., Ogden C.L. 2005. Anthropometric reference data for children and adults: US population, 1999-2002. Adv Data 361:1-5.

Waterland R., Michels K. 2007. "Epigenetic epidemiology of the developmental origins hypothesis." *Annual Review of Nutrition* 27: 363-388.

World Health Organization 2005. "Preventing Chronic Diseases: A Vital Investment: WHO Global Report."

Woods L., Ingelfinger J., Nyengaard J., Rasch R. 2001. "Maternal protein restriction suppresses the newborn rennin-angiotensin system and programs adult hypertension in rats." *Pediatric Research* 49: 460-467

Wu G., Bazer F., Cudd T., Meininger C., Spencer T. 2004. "Maternal nutrition and fetal development."*Journal of Nutrition* 134: 2169-2172.

Appendix

	Males	Females
	n=847	n=785
Offspring		
Age	20.94 ± 0.33	20.93 ± 0.35
Height, cm	162.95 ± 5.89	151.17 ± 5.51
Weight, kg	55.85 ± 9.33	46.65 ± 8.00
BMI, kg/m^2	20.99 ± 3.04	20.39 ± 3.15
Birth weight, g	3.03 ± 0.42	2.98 ± 0.41
Birth length, cm	49.36 ± 2.03	48.88 ± 1.94
SBP, mm Hg	112.08 ± 10.85	99.25 ± 10.06
DBP, mm Hg	76.52 ± 9.29	67.88 ± 8.51
Mothers of Index children (all)		
During Pregnancy		
Height, cm	150.65 ± 5.01	
Triceps skin fold thickness, mm	2.62 ± 0.34	
Current		
DBP, mm Hg	79.32 ± 13.02	
SBP, mm Hg	118.8 ± 20.32	

Table 1. Descriptive statistics.

	SBP	DBP	Birth Weight	Age	Height	BMI	Mother's Triceps	Mother's Height	Mother's kcal	Mother's % protein	Index Child's kcal	Income
SBP	1.00											
DBP	0.67*	1.00										
Birth Weight	0.03	0.03	1.00									
Age	0.02	0.00	0.05	1.00								
Height	0.09*	0.08*	0.30*	0.02	1.00							
BMI	0.34*	0.26*	0.11*	-0.03	-0.02	1.00						
Mother's Triceps	0.00	-0.02	0.21*	0.01	0.16*	0.12*	1.00					
Mother's Height	0.01	0.02	0.21*	-0.02	0.47*	-0.02	0.22*	1.00				
Mother's kcal	0.00	0.05	0.04	-0.09*	0.12*	0.04	0.20	0.14*	1.00			
Mother's % protein	0.04	0.05	0.06	0.05	0.00	-0.03	0.10	0.03*	-0.05	1.00		
Index Child's kcal	0.05	0.07	0.08*	-0.01	0.14*	-0.04	0.08	0.13*	0.21*	0.01	1.00	
Income	-0.05	0.00	0.10*	0.03	0.11*	-0.07*	0.16	0.08*	0.20*	0.01	0.11*	1.00

* P≤0.05

Table 2. Correlation matrix: females

	SBP	DBP	Birth Weight	Age	Height	BMI	Mother's Triceps	Mother's Height	Mother's kcal
SBP	1								
DBP	0.60*	1							
Birth Weight	-0.02	-0.05	1						
Age	0.08*	0.05	-0.00	1					
Height	0.11*	0.003	0.22*	0.07*	1				
BMI	0.33*	0.29*	0.08*	-0.04	0.07*	1			
Mother's Triceps	0.00	0.00	0.15*	0.05	0.11*	0.21*	1		
Mother's Height	0.08*	0.04	0.17*	0.05	0.50*	0.11*	0.15*	1	
Mother's kcal	0.07*	0.06	0.04	-0.04*	0.13*	0.10*	0.22	0.13*	1
Mother's % protein	-0.09*	-0.03	-0.00	0.01	-0.01	0.06	0.03	-0.02	-0.04
Index Child's kcal	0.04	0.06	-0.00*	-0.08	0.11*	0.19*	0.14	0.09*	0.13*
Income	0.07*	0.08*	0.07	-0.01	0.11*	0.15*	0.19	0.09*	0.17*

*P≤0.05

Table 3. Correlation matrix: males.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Birth						
Weight, kg	-2.72˘	-2.61˘	-2.74˘	-2.82‡	-2.85‡	-2.57˘
Length, cm	0.30	0.35	0.36	0.37	0.37	0.34
Adult Offspring's						
Age	2.79‡	2.94‡	3.11‡	3.02‡	3.12‡	3.31‡
Height, cm	0.14˘	0.13*	0.13*	0.14*	0.13*	0.13*
BMI, kg/m^2	1.18‡	1.24‡	1.28‡	1.29‡	1.27‡	1.26‡
Mother's During Pregnancy						
Triceps, mm		-3.01‡	-3.19‡	-3.09‡	-3.30‡	-3.64
Height, cm		0.04	0.03	0.04	0.03	0.03
Energy intake, kcal			0.0008*	0.0009*	0.0008	0.0009*
% energy from protein			-0.26‡	-0.26‡	-0.27‡	-0.28‡
Vitamin use			-1.13	-1.13	-1.23*	-1.11
Adult Offspring's						
Energy intake, kcal				-0.79		
% energy from fat				-0.002		
Smoking					-0.77	
Household income					0.55	
Assets					-0.06	
Urbanicity					-0.01	
Mother's						
Current SBP, mm Hg						0.05‡
Model R^2	0.126	0.133	0.146	0.157	0.1443	0.156

*P≤0.10
˘P≤0.05
‡P≤0.01

Table 4. SBP in CLHNS Adult Males: ß-Coefficients from Linear Regression Models

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Birth						
Weight, kg	-0.42	-0.24	-0.47	-0.60	-0.54	-0.59
Length, cm	-0.23	-0.21	-0.20	-0.19	-0.19	-0.21
Current						
Age	0.84	0.83	0.74	0.72	0.81	0.58
Height, cm	0.20‡	0.23‡	0.24‡	0.24‡	0.24‡	0.24‡
BMI, kg/m^2	1.08‡	1.10‡	1.10‡	1.10‡	1.10‡	1.11‡
Mother's						
Triceps, mm		-1.55	-1.42	-1.35	-1.29	-1.62
Height, cm		-0.03‡	-0.04	-0.05	-0.04	-0.05
Energy intake, kcal			0.00004	-0.0001	-0.00004	-0.00009
% energy from protein			0.15*	0.15*	0.17ˇ	0.13*
Vitamin use			-1.63ˇ	-1.74ˇ	-1.64ˇ	-1.74ˇ
Index Child						
Energy intake, kcal				1.69ˇ	1.64ˇ	1.43ˇ
% energy from fat				-0.02		
Smoking					0.21	
Household income					0.06	
Assets					-0.31	
Urbanicity					0.006	
Mother's						
Current SBP, mm Hg						0.03‡
Model R^2	0.124	0.127	0.126	0.142	0.145	0.149

*P≤0.10
ˇP≤0.05
‡P≤0.01

Table 5. SBP in CLHNS Adult Females: ß-Coefficients from Linear Regression Models

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Birth						
Weight, kg	-2.50‡	-2.48‡	-2.41ˇ	-2.41ˇ	-2.40ˇ	-2.60ˇ
Length, cm	0.33*	0.37*	0.34*	0.34*	0.35*	0.35
Current						
Age	1.63*	1.70*	1.80ˇ	1.82ˇ	1.91ˇ	3.25‡
Height, cm	-0.03	-0.05	-0.06	-0.06	-0.07	0.13*
BMI, kg/m^2	0.90‡	0.93‡	0.93‡	0.92‡	0.91‡	1.27‡
Mother's						
Triceps, mm		-1.57*	-1.78*	-1.87*	-2.18ˇ	-3.42‡
Height, cm		0.05	0.04	0.04	0.04	0.03
Energy intake, kcal			0.0006	0.0006	0.0004	0.0009*
% energy from protein			-0.11	-0.11	-0.13*	-0.27‡
Vitamin use			-0.07	-0.11	-0.29	-1.19
Index Child						
Energy intake, kcal				0.03		
% energy from fat				0.01		
Smoking					0.21	
Household income					0.30	
Assets					0.23	
Urbanicity					0.01	
Mother's						
Current SBP, mm Hg						0.64ˇ
Model R^2	0.095	0.099	0.103	0.104	0.108	0.151

*P≤0.10
ˇP≤0.05
‡P≤0.01

Table 6. DBP in CLHNS Adult Males: ß-Coefficients from Linear Regression Models

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Birth						
Weight, kg	0.03	0.21	0.23	0.14	0.10	0.14
Length, cm	-0.19	-0.17	-0.20	-0.19	-0.19	-0.20
Current						
Age	0.20	0.21	0.25	0.25	0.11	0.13
Height, cm	0.15‡	0.16‡	0.16‡	0.15ˇ	0.15ˇ	0.16‡
BMI, kg/m^2	0.71‡	0.74‡	0.74‡	0.75‡	0.76‡	0.75‡
Mother's						
Triceps, mm		-1.69*	-2.10ˇ	-2.11ˇ	-2.01ˇ	-2.25ˇ
Height, cm		-0.01	-0.01	-0.02	-0.03	-0.34
Energy intake, kcal			0.0006	0.0004	0.0004	0.0004
% energy from protein			0.15ˇ	0.15ˇ	0.16ˇ	0.14ˇ
Vitamin use			-0.07	-0.19	-0.15	-0.13
Index Child						
Energy intake, kcal				1.10*	1.20*	1.16*
% energy from fat				0.004		
Smoking					0.44	
Household income					0.22	
Assets					-0.0002	
Urbanicity					-0.01	
Mother's						
Current SBP, mm Hg						0.04ˇ
Model R^2	0.078	0.083	0.091	0.081	0.096	0.101

*P≤0.10
ˇP≤0.05
‡P≤0.01

Table 7. DBP in CLHNS Adult Females: ß-Coefficients from Linear Regression Models

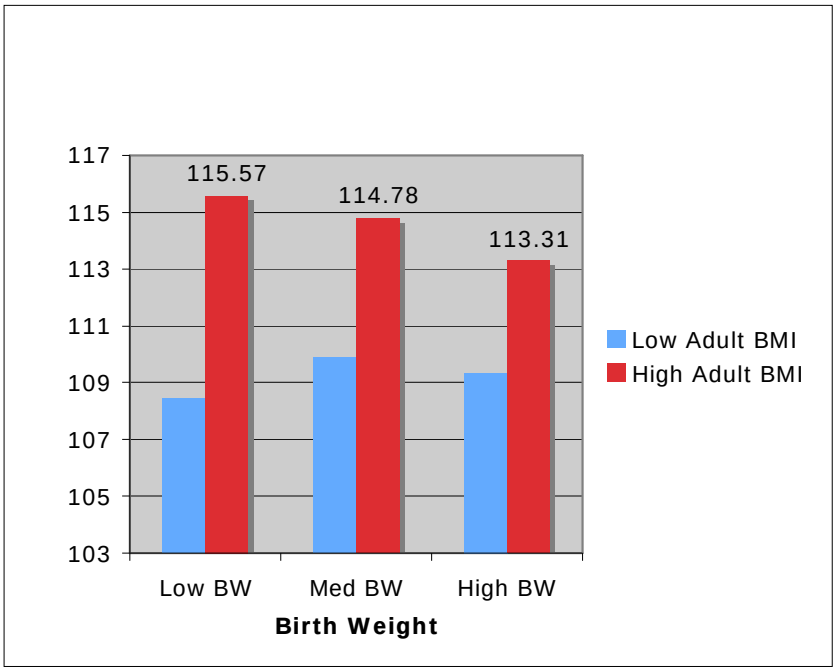

Figure 1. SBP vs. Birth Weight and Adult BMI in Males

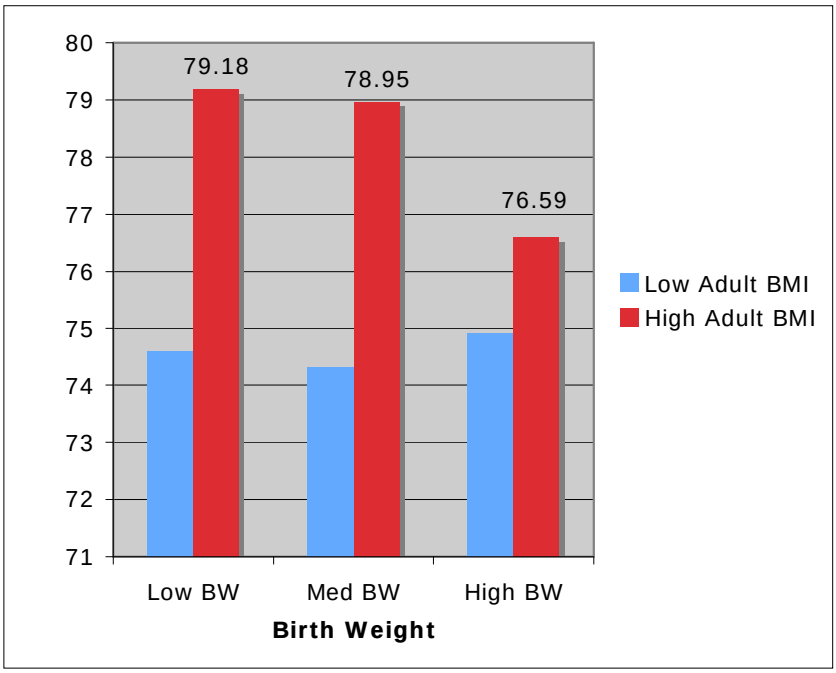

Figure 2. DBP vs. Birth Weight and Adult BMI in Males

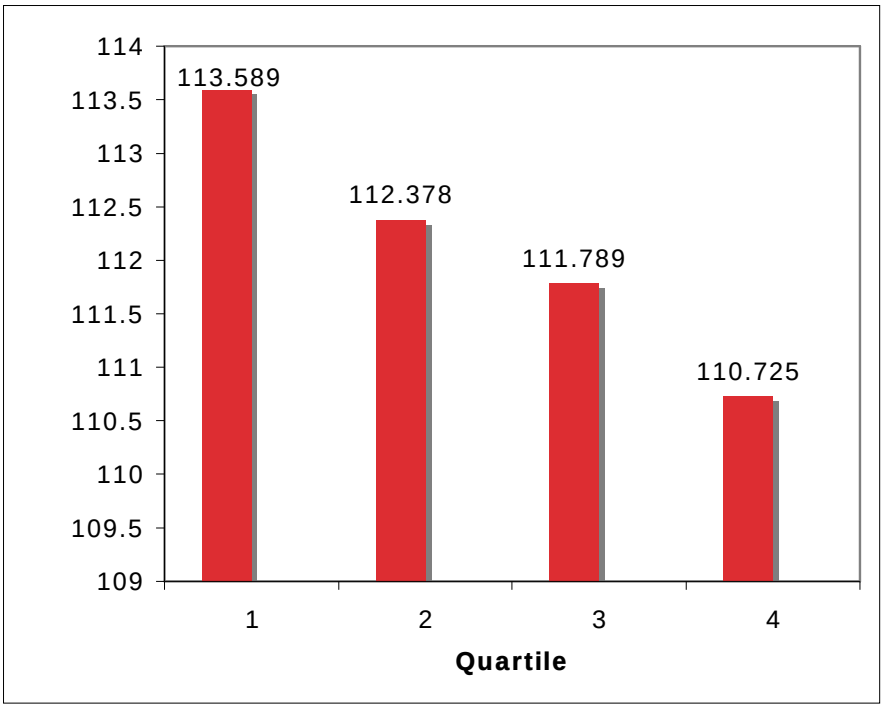

Figure 3. SBP vs. Quartiles of Birth Weight in Adult Males

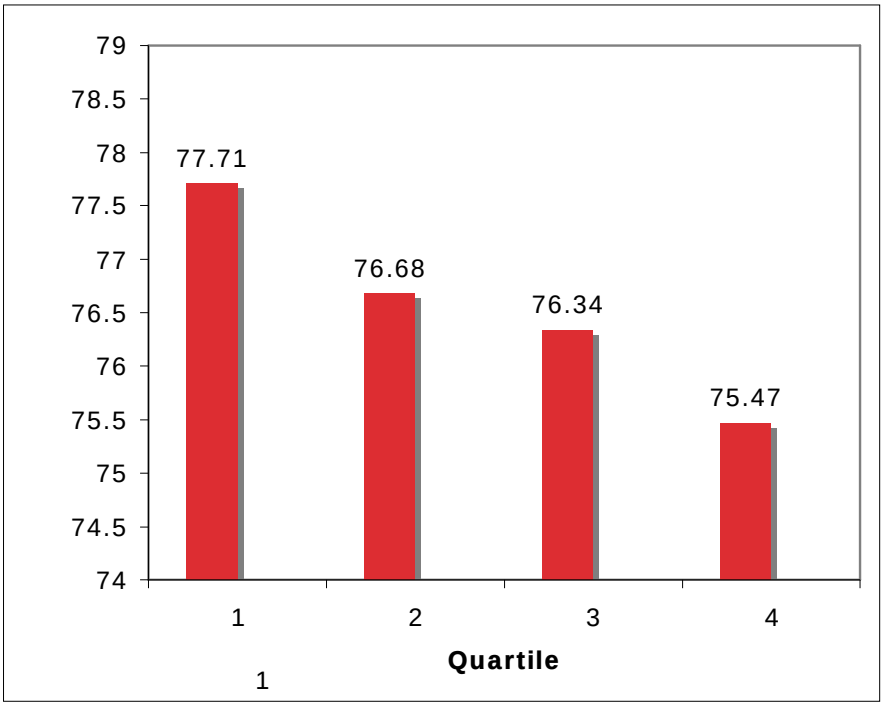

Figure 4. DBP vs. Quartiles of Birth Weight in Adult Males