The Impact of Family Socioeconomic Status and Income Inequality on Stature in the United States

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Abstract: More than two decades has passed since laboratory research first linked smaller height with various stressful environments and associated neuroendocrine functioning, and since income inequality was hypothesized as a possible explanation for the increasing American-European height gap. We employ the 1959-2002 National Health Examination Survey (NHES) and National Health and Nutrition Examination Survey (NHANES), and 1990 Summary Tape File 3 (STF3), data to estimate the influence of family socioeconomic status (SES) and income inequality on height attainment in the United States. Controlling for genes, diet, illness, and access to medical care, we find that parental income positively and metropolitan-level income inequality negatively affected childhood growth. We conclude by discussing the biological plausibility of such effects in a developed nation where it is commonly thought that most residents have reached their genetic potential, and implications for public policy in light of evidence suggesting height is positively associated with adult health, earnings, and overall well-being.

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1. Introduction

Residents of the United States of America have experienced a virtually unnoticed but remarkable macroscopic morphological transformation other than the well-documented obesity epidemic[1-3] since World War II. Once the tallest in the world and despite their relative economic prosperity, "Americans" have been overtaken in stature by the Dutch, Swedes, Norwegians, Danes, British, and Germans.[4,5] Assuming stature is one useful summary metric for biological well-being,[5-8] this international income-height paradox raises important questions about what constitutes the good life.[9,10] Recent empirical evidence, furthermore, suggests that height has an independent positive effect on individual earnings similar to that of gender and race,[11] and is positively correlated with various health outcomes[5] and life satisfaction.

What might explain the observation that height (and health in general) has not kept pace with Western European developments? Although research employing relatively recent data from Sweden points to the adverse impact of economic hardship, family size, familial dissension, and family disunity on child height attainment,[12] evidence that county-level environment mediates the correlation between heights among brothers mustered into the US Civil War,[13] and several leading economic historians studying stature have hypothesized that greater economic inequality, an inferior health care system, or less supportive social safety nets in the United States may be a useful guide,[5,14,15] as far as we know no study to date has investigated any of these possibilities employing recent representative individual-level U.S. data. In this paper we consider only the first hypothesis, originally suggested by Richard Steckel (1983). Specifically, we begin by estimating height trends among children between 1950 and 2000 – a period during which income inequality measured by the Gini coefficient rose from 0.378 to 0.398 – using 1959-2002 National Health Examination Survey (NHES) and National Health and Nutrition Examination Survey (NHANES) data. Controlling for parental heights, we then estimate whether parental socioeconomic status (SES) had any influence on child stature other than that which can be traced through the three usual suspects – nutrition, illness, and access to medical care – using 1988-1994 NHANES III data. And lastly, we estimate whether income inequality by metropolitan area influenced height by matching variables generated from the 1990 Summary Tape File 3 (STF 3) and matching them to the NHANES III data. In general, we find some support that parental SES influenced child height attainment net of parental height and childhood nutrition, illness, and access to medical care. And we find that income inequality stunted height net of all these factors. We conclude by discussing the biologic feasibility of such contextual effects on human growth, and implications for future work in light of recent evidence suggesting height independently influences adult labor, health, marital, and social outcomes.

2. Background

Despite claims that "adult height differentials by social category within the male and female population provide a strong indicator of durable inequality" within the United States and other advanced capitalist economies,[16] most research analyzing the distribution or determinants of height is historically and internationally oriented in an effort to understand secular changes or cross-national variations in the distribution of resources.[6,14,17] The scarcity of research on height attainment within the United States is surprising for three further reasons. First, social scientists have suspected that income poorly reflects general well-being since the development of national income accounting in the 1930s.[10,18-21] Second, research produced by economic[6,22] and medical[23] historians and auxiological epidemiologists[24] intimates that stature aptly summarizes genetic heredity and environmental influences on overall well-being. And third, as noted above, recent evidence links adult height with economic, marital and political outcomes often associated with self-reported quality of life.[6,25-27]

Richard Steckel, for instance, reports that across 30 nations a reduction of the Gini coefficient of 0.2 would have increased average adult height by more than seven centimeters – controlling for per capita income, level of urbanization, income of respondents, educational attainment, sex, ethno-racial characteristics, and age.[14] The Steckel (1983) inequality-height hypothesis aligns neatly with the debate concerning the impact of SES and income inequality on health more generally that has received considerable recent scholarly attention,[28-31] as well as with the clinical and laboratory evidence cited below suggesting the biological plausibility of inequality's unhealthy effects through both material and psychosocial pathways.[32]

More recent research during the past two decades on the physiologic regulation and material-psychosocial environmental determinants of human growth offers a "biocultural" model that demonstrates the joint influence of genes and socioeconomic context, and their interaction, on the endocrine system and human development.[33] Evidence of a positive relationship between SES (e.g., education, income, occupation) and stature is, of course, as old as the written record, and it has generally been acknowledged that the environment is the primary determinant of overall health for more than a century.[16,33-35] At the dawn of modern epidemiology in the early to middle 1800s, for example, Villerme noted that stature and other body proportions were not predetermined and could be affected by government policies in Paris,[8] and Engels in England wrote of "a general enfeeblement of the frame in the working class."[36] But it was not until the mid-twentieth century that systematic observational data on infants and children indicating a positive relationship between quality of childcare environment and height were forthcoming,[37-44] and not until the early 1980's that stressful environments and various associated hormonal disturbances were linked to smaller stature. Although some very recent evidence suggests stress associated with insufficient sleep may augment hunger and weight by altering diet-related hormone (e.g., leptin and ghrelin) levels and thereby growth,[45] for example, most non-human experimental and clinical human studies indicate that emotionally disturbed environments can depress secretion of digestive enzymes and food absorption, resulting in malnutrition even when sufficient food is available,[46] or alter the release of other hormones important for human growth.[47-54] Among children who survive severe emotional abuse or neglect, furthermore, it appears that an overabundance of stress hormones early in life can retard mental and physical development well into adulthood.[55] In short, both material resources and psychosocial hetronomous contextual factors seem to matter for vertical growth.

The so-called "milk hypothesis" and recent work reporting a direct influence of family emotional environment on child cortisol levels and various health outcomes are examples that may stimulate thinking about the possible impact of stress generated by low SES, income inequality, or other upstream[56] forces on stature. The association between milk consumption and taller height has been well known since late 1920's,[57-59] and we now know that hormone cholecalciferol (vitamin D_3 rather than D_2, the form commonly added to milk) is essential for the intestinal absorption of calcium and the mineralization of new bone tissue. What is less well known is that the synthesis of vitamin D_3 that is required before it enters the blood stream and makes its way to the intestine and bones via the liver and kidneys occurs in the deep layers of the skin when people are exposed to ultraviolet light.[60] While distributions of milk (rather than soda or what often erroneously passes for unadulterated fruit juice) consumption and sun exposure may be correlated with parental SES and income inequality, more recent work investigating connections between socioeconomic conditions, psychosocial stress, and health provides a more attractive example.

Consistent evidence from data collected on the Caribbean island of Dominica suggests, for example, that the degree of genetic dissimilarity between caretakers and children is positively related to cortisol levels and several adverse health outcomes. Specifically, mean cortisol levels are highest when subjects reside with a step-father and half-siblings or when they reside with distant relatives, and the next highest levels of cortisol are detected among children residing in single-mother households.[54] Cortisol and certain other hormones which are released slowly (rather than rapidly for fight or flight purposes) in response to external stress shut down body functions which are nonessential in the short term but may be important for growth.[55] While neither the milk nor the caretaker studies provide direct evidence that childhood psychosocial environmental factors influence stature, results of relatively recent research are compatible with the historic record and establish that in the United States and Western Europe persons of lower SES are shorter, have more fat mass, and have less muscle and skeletal mass than higher SES persons.[61]

A substantial amount of work has investigated the determinants of various components of primate stature that have different growth curves (e.g., leg, trunk, head), and a post-neonatal growth curve distinguishing between *infancy* (2-36 months), *childhood* (3-7 years), *juvenile* (7-10 years for girls, 7-12 for boys), *adolescence* (10-18 for girls, 12-20 for boys), and *mature* (19 or more years for girls, 20 or more years for boys) stages of height velocity is generally regarded as representative of the pattern of overall human growth.[33,62] Pre-maturity factors influencing growth may usefully be separated into (1) genetic/endocrine regulation, (2) nutrition, (3) disease/illness, (4) care/rest/stress environment, and (5) household socioeconomic status (SES) analytical categories.

A charitable estimate of the proportion of height variation by birth cohort since the second World War and group socioeconomic status that is explained by genetic factors (category 1), according to a recent review of the literature[63] and consistent with evidence from the United States,[64] is 80 percent. Of the remaining 20 percent, the main contextual factors typically thought to influence stature are childhood, rather than prenatal, nutrition and disease (categories 2 and 3). For instance, although some clinical research has shown caloric intake among pregnant women to have a positive effect on birth weight and height after five years,[65,66] others examining the impact of intrauterine nutritional deprivation during war and famine report no such effect.[67,68] The role of postnatal nutrition and disease on attained stature is more certain, but believed to be weaker in economically developed nations. An underlying assumption of this belief is that a higher average standard of living has a similar effect across economically diverse population subgroups, with relatively equal access to protein, vitamin D, calcium, phosphorus, magnesium, zinc, and iron,[57,58,69,70] but there has been some recent debate regarding evidence generated in the 1970s suggesting that height is adversely influenced by childhood asthma[71-74] and diabetes.[75-80] Some studies also suggest that females are protected more than males against stress caused by nutritional, climatic, or psychosocial deprivation.[55,81-83] Consequently, although such "buffering" mechanisms are poorly understood,[84] it appears that gene-environment interactions influence attained height also.

The limited scholarly attention given to the contextual determinants of height (categories 4 and 5, as well as extra-household factors) in the United States today emanates not only from the supposition that genes explain most of the variation in height in a developed economy, but also because many of the effects of household SES on child development are mediated by nutrition, illness, and childcare environment. Any other impact of SES is thought to be slight, and extra-individual or extra-household social or spatial factors such as income inequality are thought to be non-existent, or are at least left out of the models of which we are aware.[54]

Although we are able to control for parental height in the following analysis, one should not interpret this effect as solely genetic. Indeed, early work on the body composition and stature of international migrants dating back to the early 1900s suggested that cultural and nutritional context may be more important then genes in determining child height attainment.[85] And more recently those emphasizing a cohabitational[86] or transgenerational[61] perspective have argued that studies attributing significant influence to parental height often ignore current or past familial SES differences.

3. Data and Methods

Four nationally representative samples containing height and other information of individuals, in addition the 1990 Summary Tape File 3 (STF 3), data are analyzed below. We first employ 1959-1962 National Health Examination Survey (NHES), 1999-2002 National Health and Nutrition Examination Survey (NHANES), data to estimate trends in height attainment in the United States among men and women aged 20-69 years who were born between 1890 and 1980 by race, and whether trends in stature were associated with family SES and income inequality during the past half century. The NHES was and NHANES is administered by the U.S. Center for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS). The NHES Cycle I data were collected by administering a household survey in which respondents were asked to provide demographic and health information about themselves and other household members, by matching this information with that available from health records, and by a direct medical examination during which physical and physiological measurement were obtained. Although the adult probability sample consisted of information for 7,710 non-institutionalized U.S. residents, only 6,672 of these were examined for height, and after deleting those without information for race and other variables of interest, our final sample included 5,887 observations.[87,88] Unfortunately the NHES does not include a place-of-birth variable, so our trend estimates cannot exclude the foreign-born. The 1999-2002 NHANES is a continuation of the NHES, and the most recent phase of the NHANES, which has been conducted periodically since 1971. Like NHES, the target population is the non-institutionalized U.S. civilian population, and of approximately 7,000 individuals of all ages being interviewed in their homes, about 5,000 complete the health examination component of the survey. Thus, once dropping those individuals who were less than 20 or more than 69 years old, and for whom we lack height or other desired information for purposes of this study, 7,690 observations remain. Although we have sufficient data from the 1959-1962 NHES and 1999-2002 NHANES to estimate how height has changed for all residents of the United States across nine birth cohorts (1890s-1970s), for instance, we cannot compare Latino with non-Latino black and white heights before the 1930 birth cohort because the NHES has too few observations for Latinos.

While the NHES and 1999-2002 NHANES data permit one to observe height trends during the previous century in the United States, and for non-Latino blacks and whites separately by gender, educational attainment, and household income – they do not include information that allows us to estimate the relative contributions of genetic, family environmental, and area-level socioeconomic characteristics to height attainment. The youth file of the seventh of the NCHS health examination surveys, NHANES III (1988-1994), however, does. Specifically, building on recent work which analyzed the relationship between adult height and adult SES (i.e., income, educational attainment),[5] we link the NHANES III demographic youth data file with its health examination, vitamins and medicine, and individual food (IFF) and combination food (CFF) file counterparts to estimate the relative contribution of biological inheritance, nutrition, illness, access to medical care, and family SES environment on child height attainment. The youth file contained responses for 13,944 children aged 2 months to 17 years about whom information was collected, and 13,149 of these received a medical examination either in a mobile examination center or in their home. Although no observations are lost when merging the supplemental vitamins and medicine file, our sample is reduced to 11,656 after linking to the food combination file. And after deleting those observations not having data needed for our model, we are left with 7,815 (2,634 infants, 3,626 children, and 1,555 adolescents).

The health examination file gives us the data to compute our dependent variable – recumbent height for those less than two years old and standing height for children aged 2-17 years. Specifically, we normalize height in centimeters by age for each person in our sample. The demographic file provides us with age, gender, ethno-racial group identification, and parental height. Age enters our models as a continuous variable, we distinguish between Latinos and non-Latino white, black, and other populations, and parental height is employed as mid-parent height.[89,90] The vitamins and medicine file includes data on whether a person was taking any form of medication or supplemental vitamins, and how much, but none of these variables turned out to be useful and are therefore excluded from our models below. The CCF and IFF; however, contain information about the intake of beverage and food items that fall into 15 different single- or multi-component food combination types (e.g., beverage, sandwich, frozen meal, mixed dish) according to 24-hour dietary recall,[91] rather than according to the more desirable food frequency survey method.[92] Nonetheless, in addition to total energy (nutrient) intake, we are able to compute the proportion that each of the six essential nutrient categories for growth (i.e., carbohydrate, fat/lipid, protein, mineral, vitamins, and water) represent in each persons diet.[33] The data also permit us to control for past disease and illness, access to medical care, and household environment; and we only report the influence of SES and metropolitan socioeconomic context variables after doing so below (see Table 1 for variable definitions).

4. Results

Figures 1 and 2 reveal that standing mean height among male and female adults aged 20-69 years born between 1890 and 1980 generally rose until the 1950s, and then during the next three decades remained fairly constant or began to decline. Stature among Latinos and other Americans not classified as non-Latino black or white (a population which we shall term "Latino" throughout the remainder of this paper); however, continued to rise.

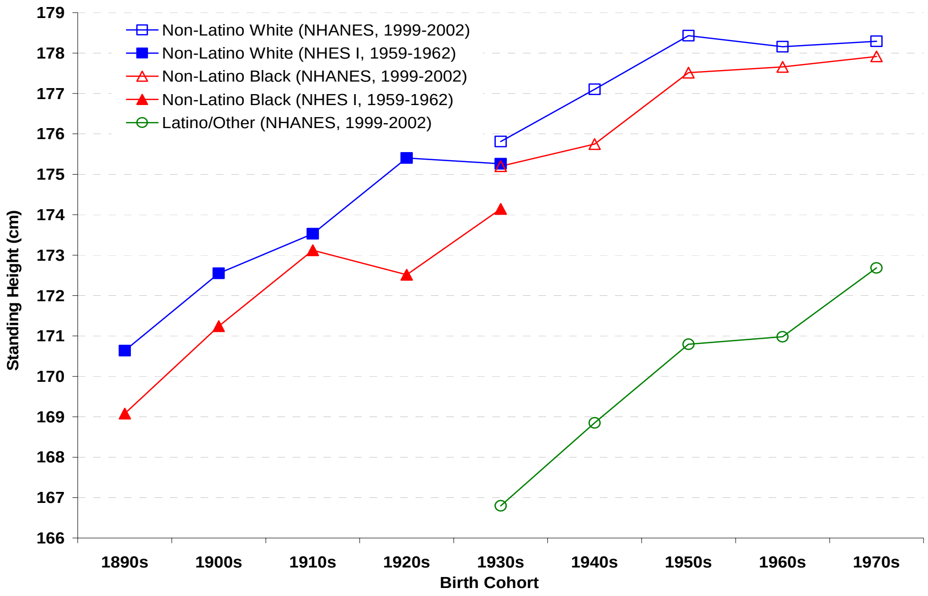

Figure 1: Height (cm) among Men Aged 20-69 Years Old and Residing in the United States by Ethno-Racial Group and Birth Cohort, 1959-2002

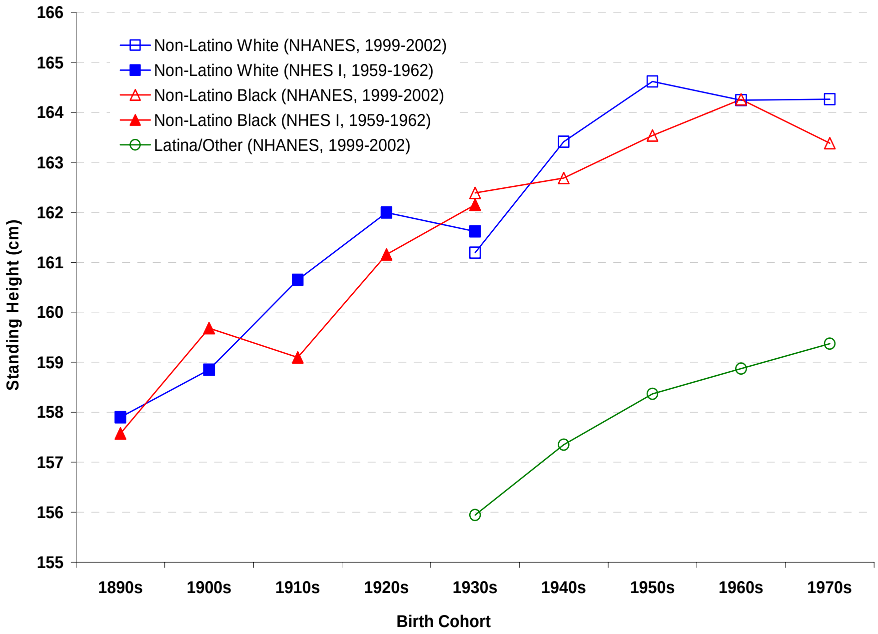

Figure 2: Height (cm) among Women Aged 20-69 Years Old and Residing in the United States by Ethno-Racial Group and Birth Cohort, 1959-2002

Although the stature of first-generation international migrants to the United States partly explains the lower heights of Latinos in general, Figures 3 and 4 intimate that immigration alone is insufficient for explaining the flattening of the U.S. height curve and Americans' relative decline compared to northwestern Europeans during the past generation. Indeed, it appears that heights among Latinos, regardless of country of birth, have continued to rise, unlike those of the rest of the U.S. population. Heights of non-Latino white women who were born in the United States during the 1970s decreased more than three centimeters compared to those born in the 1950s, for example.

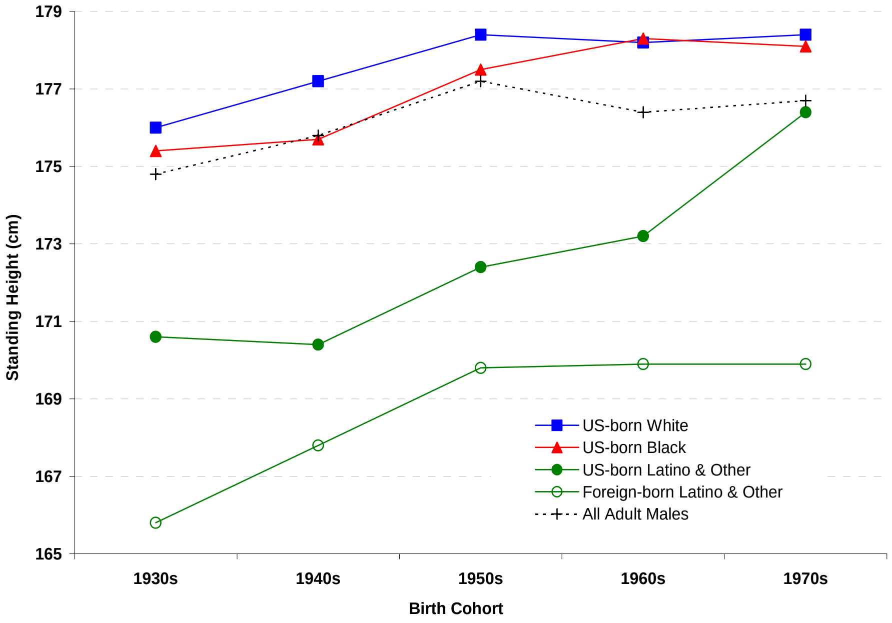

Figure 3: Height (cm) among Men Aged 20-69 Years Old and Residing in the United States by Ethno-Racial-Nativity Group and Birth Cohort, 1999-2002

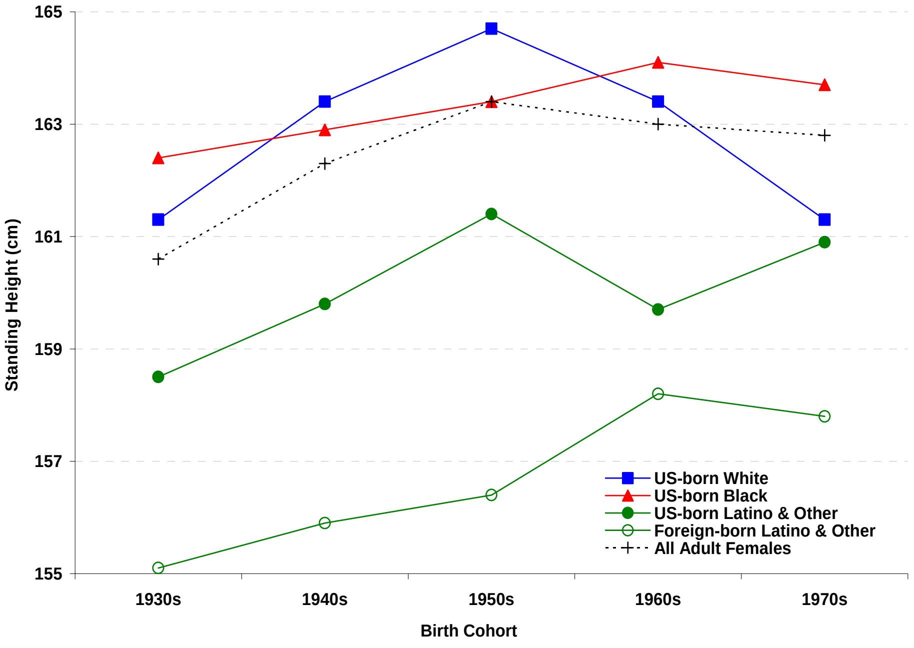

Figure 4: **Height (cm) among Women Aged 20-69 Years Old and Residing in the United States by Ethno-Racial-Nativity Group and Birth Cohort, 1999-2002**

Apparently, as measured by height, the health of neither men nor women during childhood benefited from the tremendous economic growth following World War II and subsiding circa 1973,[93] but women faired worse. Despite persistent wage[94] and potential health disparities between men and women, descriptively height appears to be positively related to family income. Figure 5 illustrates that for both men and women born during the 1890s-1920s or the 1930s-1970s, those residing in families earning less than $20,000 in 2002 dollars were shorter than members of families earning between $20,000 and less than $55,000, and adults residing in families earning at least $55,000 were the tallest. Not only did male and female heights rise over the century, but the slopes of the female and male earnings-height gradients are quite similar. We cannot conclude; however, that the female decline in stature is attributable to family earnings differentials or trends because this graph is comparing adult heights with the current family earnings, not with parents' earnings. Below we will estimate this relationship directly employing different data, however.

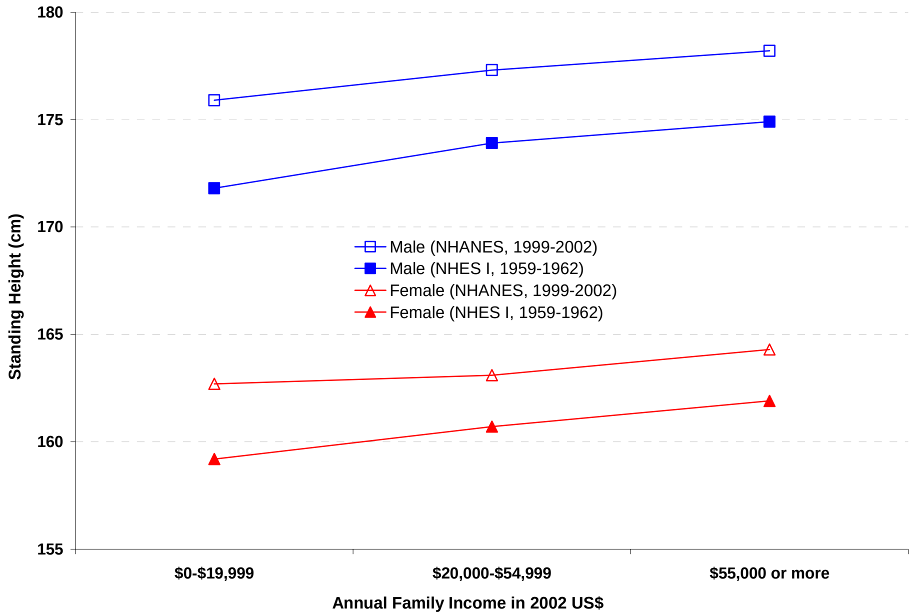

Figure 5: Height (cm) among Adults Aged 20-69 Years Old and Residing in the United States by Income and Gender, 1959-2002

Figure 6 shows that among men born during the 1930s-1970s the height gap between those in higher income families and those in lower and middle income families converged and became smaller than that among women, which remained fairly constant. This suggests that the earnings-height gradient may have become flatter over the past half century, but again, we are here comparing current family earnings with adult height, not the income of one's family during childhood.

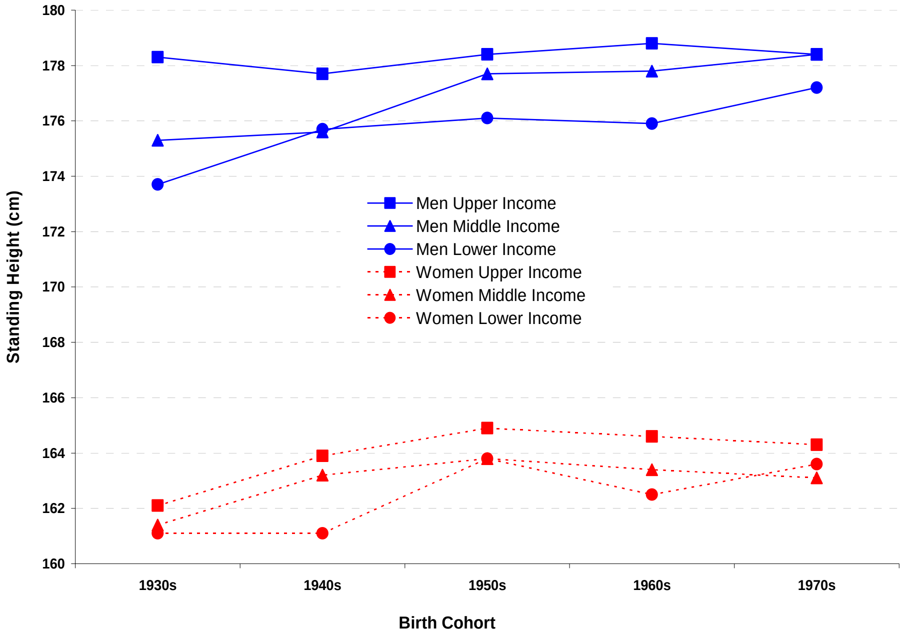

Figure 6: **Height (cm) among Adults Aged 20-69 Years Old and Residing in the United States by Income, Gender and Birth Cohort, 1999-2002**

A similar story is revealed when one considers the relationship between adult educational attainment and height. The education-height gradient (Figure 7) appears to have remained fairly the same, and the education-height gap (Figure) is estimated to have narrowed slightly, during the past half century.

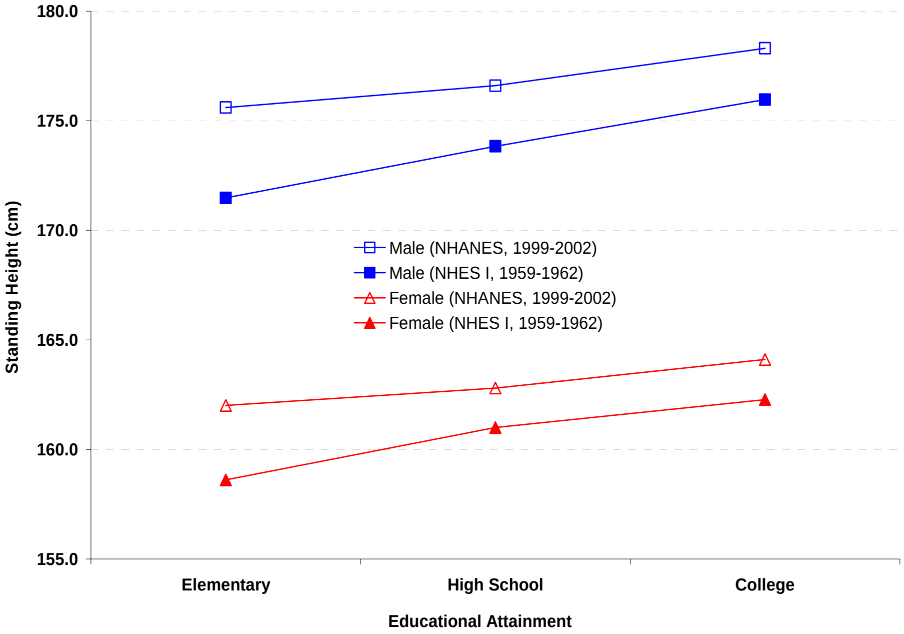

Figure 7: Height (cm) among Adults Aged 20-69 Years Old and Residing in the United States by Educational Attainment and Gender, 1959-2002

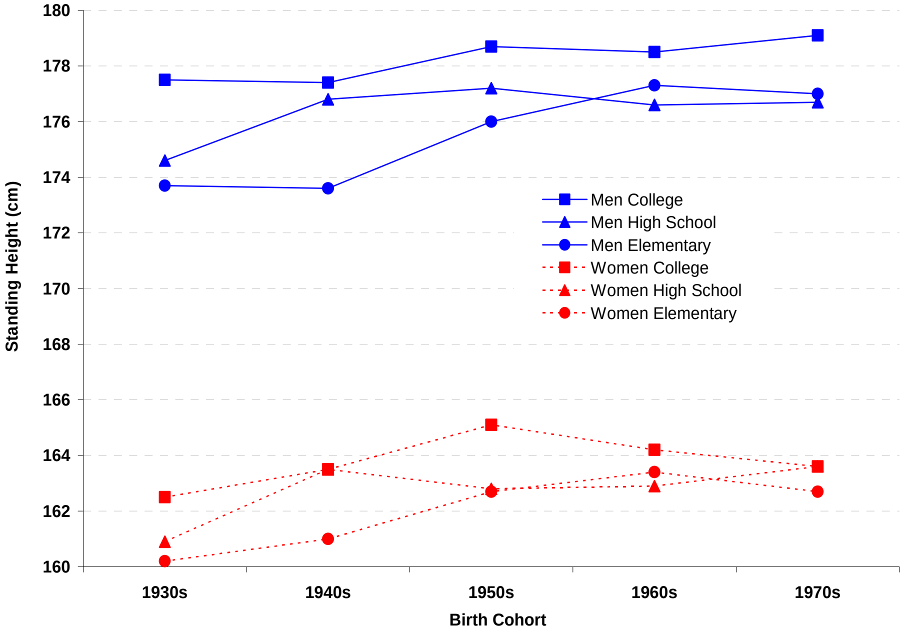

Figure 8: Height (cm) among Adults Aged 20-69 Years Old and Residing in the United States by Educational Attainment, Gender, and Birth Cohort, 1959-2002

When one considers the relationship between height and the distribution rather than the level of income, the Steckel (1983) income inequality-height, like the SES-height gradient, hypothesis is given some support. Figure 9 links the 1959-1962 NHES and 1999-2002 NHANES height data for those born between 1890 and 1980 with U.S. Bureau of the Census estimates of income inequality (as measured by the Gini Coefficient) between 1947 and 1980 and interpolated estimates between 1913 and 1946[95] to assess whether inequality and height were related. Specifically, we plot the mean Gini coefficient for each decadal birth cohort, and find that, in general, between the 1910s and 1970s inequality fell and mean height rose. In addition to the diverging overall trends, for instance, cross-decade periods that saw declining inequality also experienced increasing height (e.g., 1910s-1920s, 1930s-1940s) and those that saw rising or relatively stable inequality experienced declining or constant height trends (e.g., 1920s-1930s, 1940s-1970s).

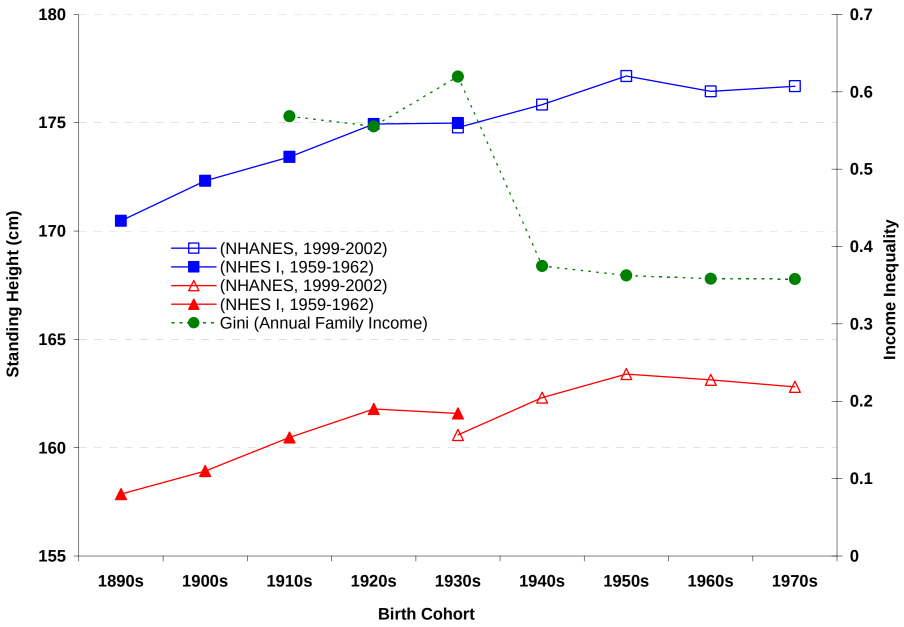

Figure 9: **Height (cm) among Adults Aged 20-69 Years Old and Residing in the United States by Income Inequality, Gender, and Birth Cohort, 1959-2002**

Even if height and income inequality were not related temporally it may be the case that they are geographically. In Figure 11 we compare the mean height of adult females according to the pooled 1993-2002 Behavioral Risk Factor Surveillance Survey (BRFSS) data and income inequality as measured by the Gini using the 1990 Summary Tape File 3 (STF3) data. The correlation between average male adult height and the Gini is -0.032, and between average female adult height and the Gini it is -0.240. The female results are plotted below as further evidence that income inequality may help explain why American heights have not kept pace with those of North Western Europeans.

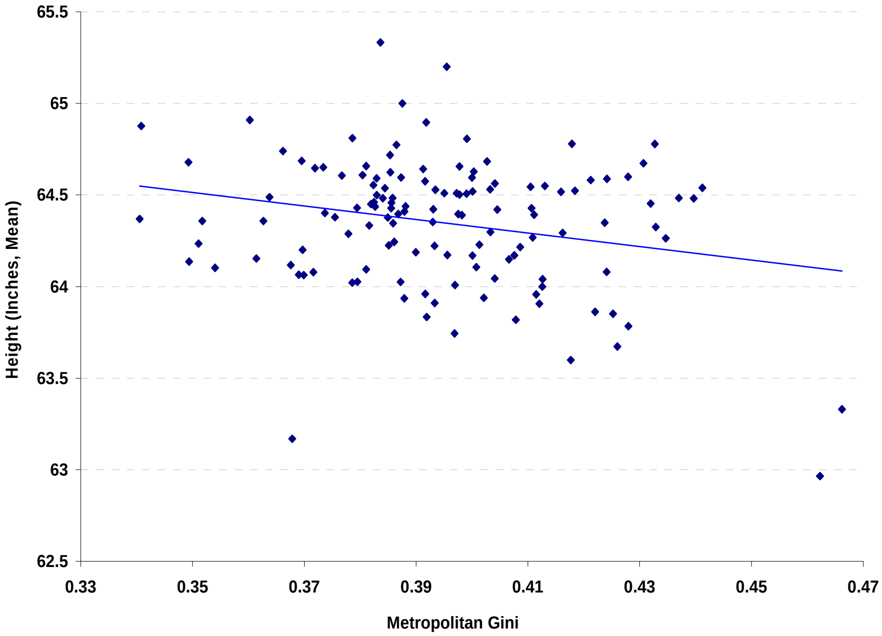

Figure 10: **Height (cm) among Female Adults and Income Inequality by Metropolitan Area, United States (1990 STF3 and 1993-2002 BRFSS)**

To assess whether there is any merit to the idea that SES or income inequality has an independent effect on height attainment in the United States, we first define six categories of explanatory variables and present the means and standard deviations for each variable by three childhood growth periods (Table 1). In the "genetic" category, non-Latino whites are the control group, and in the nutrition category the percent of total nutrient intake accounted for by water is excluded. Finally, statistics for the metropolitan-level variables were computed using fewer observations as a result of merging metropolitan-level (STF3) and county-level (NHANES III) data

*** TABLE 1 HERE ***

Results of regressing age-normalized height on these explanatory variables are shown in Table 2, and several things are worth highlighting. First, even after controlling for genetic, nutritional, disease\illness, and medical care\home environment, there is some evidence that per capita family income augmented child height attainment across the entire age distribution and income inequality reduced it. Second, although family SES also seems to have had a positive impact on the growth of infants, parental educational attainment and income inequality are inversely related to infant height attainment. These negative effects, as well as the apparent positive influence of residing with a smoker, require further investigation which we do not attempt here. But third and lastly, the take-home message is robust and clear. Even when we account for genes, diet, disease, and access to medical care, familial SES positively and metropolitan-level income inequality negatively influenced the vertical growth of U.S. children.

*** TABLE 2 HERE ***

5. Discussion

Some have argued that less sophisticated anthropometric metrics such as height, weight, blood pressure, and respiratory functioning provide important supplementary information about health not captured by more conventional morbidity or mortality rates and self-reported health status[7].

Whereas body weight is simply the cumulative sum of net energy over the life course; however, vertical growth is a negative function of the proportion of energy intake employed in maintenance (basal metabolism), repair (the restoration of cells, tissues, organs, or organ systems following illness), and physical workload.[33] The distributive use of energy intake, furthermore, will affect overall height attainment differently by age because human growth occurs at varying velocities. In other words, the potential usefulness and simplicity of height as a proxy for childhood environment does not necessarily imply that its variation is neatly modeled. The growth (velocity) curve requires at least two (three) mathematical functions for accurate description, for instance.[96,97] We are less ambitious in this exploratory paper and investigate instead how various family and metropolitan socioeconomic factors influenced deviation from mean height-for-age among children in the United States.

Our results intimate that both family SES and metro-level income inequality affected growth, controlling for a host of other known growth determinants. These are consistent with the Steckel (1983) inequality-height hypothesis, and they are generally consistent with others' work suggesting that attained height embodies parental height (a proxy for both genetic and intergenerational SES effects), childhood gross or net nutrition,[98,99] and health habits and SES,[100] in addition to childhood health and illness.[101-103] But they also complement research by demographers, economists, and epidemiologists showing that the infrastructural and socioeconomic characteristics of where one resides may affect health.[104-108] It has been estimated, for example, that the correlation between heights of brothers in the Union Army during the US Civil War and county-of-origin population size (a proxy for economic context) was negative, and that variation in heights rose with county population.[13] Such evidence exists in other nations and at other times,[109] and suggests a larger role of environment in determining heights in places that are poorer. Anthropologists, for instance, have provided evidence spanning almost a century that cultural and socioeconomic context is more important than genes in determining body shape and size by comparing children of Italians, Jewish, and Guatemalan immigrants residing in the United States to their parents as well as children remaining in the sending nations.[85,110] The attribution of contextual effects on height to poorer areas or nations, if our findings are correct however, may be misleading.

The import of considering socio-spatial determinants of height attainment during childhood and public health interventions likely to impact these is heightened if adult height has an independent effect (beyond adult health) on labor market and other socioeconomic outcomes often correlated with human well-being.[26,111] Indeed, on recent study finds that height has a similar impact on earnings differentials as gender and race.[11]

Although auxological epidemiology emerged in France, Belgium and England almost two centuries ago and height has been used a summary measure for physical and subjective well-being (e.g., overall health status) since the 1970s by nutritionists and development economists, no study to date, as far as we know, has investigated how individual characteristics and socioeconomic environment jointly are currently influencing height in general or across demographic groups in the United States. In light of recent scholarly work,[6,15,25] and popular press coverage[4] suggesting a strong direct relationship between height (or other anthropometric measures of human growth) and being married, securing higher earnings and employment promotions, it is surprising that there has been virtually no attention to trying to understand the determinants of height in the United States.

Recent research indicating that stressful environments, particularly proximate socioeconomic conditions such as family environment and income inequality, harm neuroendocrine functioning that in turn may stunt growth and have long-term harmful health, labor, and other social effects, it seems reasonable that more attention be given to the determinants of height even within economically advanced nations.

	Definition	Infant		Child		Adolescent		All	
Dependent Variable									
Height	Recumbent length (for those aged 2-36 months) or standing height (for those aged 3-17 years) (cm)	78.524	(10.674)	117.589	(16.137)	159.286	(11.131)	112.719	(31.946)
Age-normalized height	= Height (cm) as percent of mean height by age group (cm)	0.997	(0.135)	0.997	(0.134)	1.000	(0.064)	0.998	(0.124)
"Genetic"									
Age	Age (years since birth)	1.414	(0.853)	6.362	(2.398)	13.702	(1.934)	6.155	(4.753)
Male	= 1 if gender is male	0.509	(0.500)	0.536	(0.499)	0.361	(0.481)	0.492	(0.500)
Latino	=1 if ethno-racial group is Mexican-American or other Hispanic	0.256	(0.436)	0.354	(0.478)	0.400	(0.490)	0.330	(0.470)
Non-Latino Black	=1 if ethno-racial group is non-Latino black	0.015	(0.122)	0.021	(0.144)	0.019	(0.138)	0.019	(0.136)
Non-Latino Other	=1 if ethno-racial group is non-Latino other (excluding white)	0.017	(0.130)	0.013	(0.114)	0.008	(0.091)	0.014	(0.116)
Mid-parent Height	Mean height of parents (cm)	169.411	(6.396)	168.782	(6.481)	168.859	(6.221)	169.009	(6.407)
Nutrition									
Total Nutrient Intake	Total nutrient intake according to 24-hour diatary recall (gm)	1298.154	(462.514)	1521.566	(573.424)	1756.062	(810.529)	1492.925	(618.241)
Protein	Protein intake according to 24-hour dietary recall (percent)	0.031	(0.015)	0.043	(0.014)	0.045	(0.020)	0.039	(0.017)
Fat	Fat intake according to 24-hour dietary recall (percent)	0.036	(0.016)	0.047	(0.019)	0.052	(0.025)	0.044	(0.020)
Carbohydrates	Carobohydrate intake according to 24-hour dietary recall (percent)	0.116	(0.039)	0.157	(0.039)	0.161	(0.048)	0.144	(0.045)
Vitamins & Minerals	Vitamin & Mineral intake according to 24-hour dietary recall (percent)	0.004	(0.001)	0.005	(0.001)	0.005	(0.002)	0.005	(0.001)
Disease & Illness									
Past Disease or illness	=1 if child had asthma or one of 11 other diseases or illnesses at any time	0.179	(0.383)	0.253	(0.435)	0.275	(0.446)	0.232	(0.422)
Medical Care & Home Environment									
Doctor	=1 if doctor or other health care professional was seen for health reason durnig last two years	0.995	(0.067)	0.939	(0.239)	0.845	(0.362)	0.939	(0.239)
Smoker	=1 if there is anyone who smokes in the home	1.582	(0.493)	1.585	(0.493)	1.567	(0.496)	1.580	(0.494)
Family Socioeconomic Status									
Family Income	Per capita annual family income ($1,000s)	4.955	(3.586)	4.768	(3.383)	5.087	(3.611)	4.894	(3.500)
Education	Highest grade completed by household reference person	11.331	(3.091)	11.038	(3.246)	11.039	(3.221)	11.137	(3.192)
Metropolitan Socioeconomic Context[a]									
Income Inequality	Gini coefficient computed from 1989 annual houehold income	0.403	(0.020)	0.404	(0.019)	0.404	(0.018)	0.404	(0.019)
Average Income	Mean 1989 annual household income	34.452	(5.479)	34.120	(5.187)	33.740	(5.279)	34.158	(5.305)
Percent non-Latino black	Percent of population who claimed to be non-Latino black	0.126	(0.065)	0.128	(0.066)	0.127	(0.067)	0.127	(0.066)
Sample Size (N)		2,634		3,626		1,555		7,815	

Note: (a) The samples sizes for these three variables are smaller -- 804 infants, 1,182 children, and 459 adolescents (total=2,445) -- resulting from merging metropolitan STF3 data to county-level identifiers in NHANES III.

Table 1: Definitions, Means, and Standard Deviations of Variables Employed in Regression Analysis of Height in the United States

	Infants		Children		Adolescents		All	
"Genetic"								
Age	0.133 (0.002) ***	0.137 (0.004) ***	0.053 (0.001) ***	0.053 (0.001) ***	0.023 (0.001) ***	0.025 (0.002) ***	0.005 (0.000) ***	0.005 (0.001) ***
Male	-0.006 (0.003) **	-0.014 (0.004) ***	-0.058 (0.003) ***	-0.060 (0.004) ***	-0.023 (0.004) ***	-0.016 (0.009)	-0.011 (0.004) ***	-0.014 (0.008)
Latino	0.003 (0.010)	-0.001 (0.008)	0.017 (0.006) ***	0.007 (0.008)	0.005 (0.008)	0.003 (0.019)	-0.025 (0.013) *	-0.040 (0.016) **
Non-Latino Black	0.002 (0.003)	0.000 (0.006)	0.016 (0.003) ***	0.015 (0.008) *	0.002 (0.004)	0.004 (0.007)	0.003 (0.003)	0.006 (0.008)
Non-Latino Other	0.010 (0.012)	-0.007 (0.015)	0.000 (0.009)	0.004 (0.009)	0.003 (0.016)	0.008 (0.015)	0.000 (0.028)	0.002 (0.016)
Mid-parent Height	0.002 (0.000) ***	0.002 (0.000) ***	0.004 (0.000) ***	0.003 (0.000) ***	0.004 (0.000) ***	0.004 (0.000) ***	0.004 (0.000) ***	0.003 (0.001) ***
Nutrition								
Total Nutrient Intake	0.000 (0.000) ***	0.000 (0.000) ***	0.000 (0.000) ***	0.000 (0.000)	0.000 (0.000) ***	0.000 (0.000) *	0.000 (0.000) ***	0.000 (0.000) *
Protein	0.271 (0.211)	0.218 (0.298)	0.172 (0.134)	0.091 (0.171)	0.369 (0.146) **	0.332 (0.282)	0.852 (0.207) ***	0.835 (0.439) *
Fat	-0.591 (0.122) ***	-0.505 (0.184) ***	-0.096 (0.104)	-0.062 (0.154)	-0.050 (0.113)	0.024 (0.167)	-0.609 (0.163) ***	-0.519 (0.293) *
Carbohydrates	0.078 (0.046) *	-0.066 (0.088)	0.037 (0.038)	-0.017 (0.051)	0.014 (0.040)	0.092 (0.051) *	0.236 (0.053) ***	0.233 (0.095) **
Vitamins & Minerals	10.054 (2.475) ***	12.605 (3.521) ***	-1.009 (1.262)	2.951 (2.319)	-2.900 (1.646) *	-6.953 (3.417) *	5.462 (2.316) **	4.814 (3.453)
Disease & Illness								
Past Disease or illness	-0.005 (0.004)	-0.007 (0.007)	0.006 (0.004) *	0.000 (0.006)	-0.006 (0.003) *	-0.012 (0.005) **	0.002 (0.004)	0.002 (0.008)
Medical Care & Home Environment								
Doctor	-0.004 (0.010)	-0.008 (0.009)	0.000 (0.007)	-0.001 (0.018)	0.007 (0.005)	0.005 (0.012)	0.002 (0.009)	-0.011 (0.017)
Smoker	0.008 (0.002) ***	0.013 (0.004) ***	0.003 (0.004)	0.004 (0.006)	0.004 (0.004)	0.000 (0.007)	0.000 (0.004)	0.003 (0.008)
Family Socioeconomic Status								
Family Income	0.001 (0.000) **	0.002 (0.001) **	0.001 (0.001)	0.000 (0.001)	0.000 (0.001)	-0.001 (0.001)	0.001 (0.001) **	0.000 (0.001)
Education	-0.001 (0.000) ***	0.000 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	0.000 (0.001)	0.000 (0.001)	-0.001 (0.001)
Metropolitan Socioeconomic Context[a]								
Income Inequality		0.301 (0.144) **		0.129 (0.157)		-0.127 (0.200)		-0.414 (0.225) *
Average Income		0.000 (0.000)		0.001 (0.000) *		-0.001 (0.001)		-0.001 (0.001)
Percent non-Latino black		-0.049 (0.029) *		0.031 (0.061)		0.030 (0.055)		0.038 (0.081)
Intercept	0.439 (0.030) ***	0.278 (0.092) ***	0.035 (0.083)	0.092 (0.114)	0.061 (0.083)	-0.010 (0.101)	0.295 (0.056) ***	0.534 (0.159) ***
R-Squared	0.8822	0.9059	0.8876	0.8810	0.5523	0.5912	0.1294	0.1407
Prob > F	0.0000	0.0000	0.0000	0.0004	0.0000	0.0240	0.0000	0.0040

Note: Regressions are weighted and corrected for survey clustering and stratifcaton by stratum and primary sample unit (PSU) using STATA 8.2

Table 2: Determinants of Age-normalized Height among Children under 18 Years of Age in the United States

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