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Does lower birth order amplify the association between high socio-economic status and central adiposity in young adult Filipino males?

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Abstract

Objective—To test the hypothesis that lower birth order amplifies the positive association between socioeconomic status and central adiposity in young adult males from a lower-income, developing country context.

Design—The Cebu Longitudinal Health and Nutrition Survey is an ongoing community-based, observational study of a one year birth cohort (1983).

Subjects—970 young adult males, mean age 21.5 y (2005).

Measurements—Central adiposity measured by waist circumference; birth order; perinatal maternal characteristics including height, arm fat area, age, and smoking behavior; socioeconomic status at birth and in young adulthood.

Results—Lower birth order was associated with higher waist circumference and increased odds of high waist circumference, even after adjustment for socioeconomic status in young adulthood, and maternal characteristics that could impact later offspring adiposity. Furthermore, the positive association between socioeconomic status and central adiposity was amplified in individuals characterized by lower birth order.

Conclusions—This research has failed to reject the mismatch hypothesis, which posits that maternal constraint of fetal growth acts to program developing physiology in a manner that increases susceptibility to the obesogenic effects of modern environments.

Keywords

Mismatch; Developmental Origins of Health and Disease; Fetal origins; Maternal constraint; Birth order; Central obesity

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Introduction

Obesity is a serious challenge to global public health (1). Public health interventions have traditionally targeted individual-level behaviors affecting dietary intake and physical activity, but these efforts have had no success at reducing obesity prevalences. Consequently, there is growing interest in how social and physical environments influence obesity risk, and efforts to identify mutable, environmental causes of obesity are underway (2–10). However, it is equally important to explain heterogeneity in adiposity among individuals that share an environment. Some people are clearly more susceptible to obesogenic environments than others, but why? One explanation is genetic variation; that some people posses *thrifty genes* that increase their susceptibility to modern, obesogenic environments (11). However, the importance of thrifty genes and the degree to which they could explain the modern obesity pandemic is still debated (e.g. 12, 13).

Another explanation is Gluckman and Hanson's *mismatch hypothesis* (14–22), which falls under the broader Developmental Origins of Health and Disease (DOHaD) paradigm (23). Briefly, it posits that maternal constraint of fetal growth can signal the developing fetus to prepare for a poor nutritional environment. Highlighted causes of maternal constraint include maternal body size, age, diet, and birth order (14, 24). The hypothesized fetal response to these signals is an integrated set of adjustments in the way energy is handled in the body (19). These adjustments are thought to enhance fitness via improved survival or fecundity during lean times, but could lead to obesity and related metabolic disorders in a nutritionally abundant environment. Ultimately, the mismatch hypothesis posits that prenatal influences can modify how we experience our postnatal environment, and could at least partly explain why some people are more susceptible to obesogenic environments than others.

There is substantial evidence that constraints on fetal growth are associated with offspring obesity later in life. For example, maternal exposure to famine during gestation is associated with increased risk of obesity in the adult offspring (25, 26). Other studies have found an inverse association between birth weight and central adiposity once body mass index (BMI) is accounted for, though these associations tend to be mild, and are not consistently detected (27). Maternal smoking has also been associated with both lower birth size and subsequent obesity later in life (28, 29). However, most previous studies have not explicitly tested the hypothesis that obesity results from an *interaction* between the constraint of fetal growth and later environment. Instead, most studies have estimated direct associations between fetal development and later obesity, irrespective of the postnatal environment. Given the mismatch hypothesis, failure to account for this interaction could lead to underestimation of the relationship between fetal growth and obesity.

Using data from a birth cohort of young adult Filipino males, we tested the hypothesis that lower birth order modified the association between central obesity and socio-economic status (SES), a useful proxy for the obesogenic environment in this context (30, 31). We also examined the influence of birth order on birth size, and explored BMI growth curves (from birth to young adulthood) in groups defined by firstborn status and SES. Although birth order is a hypothesized prenatal influence on later disease (24), and has been associated with

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reduced birth size (24, 29, 32–34) and increased risk of central adiposity (e.g. 35, 36, 37) and diabetes (38–40), it has not been adequately investigated in epidemiological studies.

Methods

Study design and sample

Data are from the Cebu Longitudinal Health and Nutrition Survey (CLHNS), a community-based, one-year birth cohort study in Metropolitan Cebu (pop 1.9 million), Philippines. The region includes 270 administratively defined communities called *barangays* (average area 2.65 km^2) comprising a 720 km^2 contiguous area. A single stage cluster sampling procedure was used to randomly select 33 barangays, and pregnant women residing in these barangays were recruited for the study in 1982 and 1983. Those who gave birth between May 1, 1983, and April 30, 1984, were included in the sample. More than 95% of identified women agreed to participate. A baseline interview was conducted among 3,327 women during their 6th or 7th month of pregnancy. Another survey took place immediately after birth; there were 3,080 non-twin live births which make up the CLHNS birth cohort. Subsequent surveys were conducted bimonthly to age 2, then in 1991, 1994, 1998, 2002, and 2005 (n=1885, 61% retention). For this analysis we used a sample of young adult males still enrolled in the CLHNS in 2005 (mean age 21.5 y) with complete case data (n=970; 98% of the 2005 sample, 59% of the original sample at birth). Males included in this analysis sample did not differ at from the remainder of the cohort at baseline (by t-test; $p \leq 0.05$) in mean 1983 household assets, birth length, ponderal index, maternal height, maternal AFA, maternal age, or birth order. However, the analysis sample did have slightly higher birth weights (difference 0.05 kg; 95% CI 0.01 to 0.09). Because the socio-environmental determinants of obesity are more complex and poorly understood in females (30, 31), we have excluded them from this analysis.

Measures

The theoretical model that describes the hypothesized relationships among variables included in this analysis is given in figure 1. The primary exposure, birth order, was assessed during the baseline interview (1983). Birth order is represented continuously or as firstborn status (versus all others) in our analyses.

First we estimated the impact of birth order on birth size for gestational age. Birth weights (kg) for infants born at home (62%) were measured by trained birth attendants with Salter hanging scales. The remainder, born at hospitals or clinics, were weighed on clinical scales. Lengths (cm) were measured within 6 days of birth using custom made length boards. Ponderal index is a measure of body mass independent of length (though unfortunately it can not distinguish lean mass from fat mass) and was calculated as weight kg/length m^3. Gestational age was estimated from the mother's self-reported date of her last menstrual period. For cases where this date was unknown, when pregnancy complications occurred, or when the infant was born weighing less than 2.5 kg, gestational age was clinically assessed using the Ballard method (41).

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To isolate the impact of birth order on birth size, we controlled for several possible confounders. Birth order is inversely associated with maternal SES at birth due to reduced fertility in high SES mothers. SES, in turn, is positively associated with maternal age, height, and arm fat area, and inversely associated with smoking, each of which are determinants of birth size. Failure to control for these factors could obscure any relationship between birth order and birth size. We measured maternal SES at baseline using an assets-based index that reflects longer-term wealth and living standards. This SES index was calculated using a principal components analysis of data on ownership of a variety of household assets at baseline (e.g. television, land, etc) (42, 43). Maternal height was measured with a folding stadiometer. AFA was calculated from mid-upper arm circumference and triceps skinfold thickness (44) during the second or third trimester of pregnancy. Maternal smoking was represented dichotomously (yes/no), irrespective of the number of cigarettes smoked. Indicator variables were used to represent younger (<20 years) and older (>35 years) maternal ages, versus a reference age (20–35 years).

Then we estimated the impact of birth order on central adiposity in young adulthood. The primary outcome, waist circumference (WC), was measured by trained interviewers in 2005 at the midpoint between the bottom of the ribs and the top of the iliac crest. High WC was defined using a fairly low cut point of WC>85 cm that may be more appropriate in Asian populations (45). We again controlled for maternal age, height, and AFA, and smoking because each of these variables could have a developmental effect on later central adiposity. We also controlled for offspring SES in 2005, again using the same continuous index derived from a principal components analysis of 2005 household assets ownership (42, 43).

Analytical methods

First we used multivariable linear models to estimate the effect of birth order and firstborn status on birth weight, length, or ponderal index. Nonlinear effects of birth order were tested using quadratic and cubic terms. We then used multivariable linear models to estimate impact of birth order or firstborn status on young adult WC, or log odds of high WC. Again, nonlinear effects of birth order were tested using quadratic terms. We report both the crude estimates and estimates adjusted for potential confounders.

We then added an interaction between birth order (or firstborn status) and 2005 SES (our proxy variable for the post-natal environment) using the appropriate product term. Under the mismatch hypothesis, our expectation was that the positive effects of SES on central adiposity would be amplified in individuals with lower birth orders. Additional interactions were tested between SES and the other prenatal variables in the model that could also moderate the effects of SES under the mismatch hypothesis (maternal age, height, AFA, and smoking). Interactions were considered significant and reported when the corresponding $p < 0.10$.

All linear models included random intercepts to account for potential dependence among observations caused by the cluster-randomized design of the CLHNS that could lead to biased standard errors for estimated regression coefficients. Because we are not otherwise interested in interpreting estimated random effects for this analysis, we do not report them.

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All reported p-values are two-sided. All models were run using Stata, version 10.0 (Stata corp., College Station, Texas).

In an exploratory analysis, we evaluated the mean BMI growth curves from birth to young adulthood of four groups defined by firstborn status (versus not) and high versus low maternal SES measured at birth (defined by the median). This was to help evaluate whether firstborn status coupled with high SES was associated with a postnatal growth trajectory characterized by early catch up growth. Heights and weights were recorded bimonthly from birth to age two, and at ages 8.5y, 11.5y, 16y, 19y, and 21.5y. BMI was calculated as weight kg/height m^2.

Results

Sample characteristics are reported in table 1. The sample in 2005 had a mean age of 21.5y. They were characterized by low mean WC (72.2 cm; *sd* 7.6) and body mass index (21.0; *sd* 3.1). Only 6% were classified as having high WC. The median birth order was 3, and ranged from 1–15. 22% were classified as firstborn. A higher proportion of firstborns compared to higher order births were preterm (21.6% versus 15.2%, chi2 p=0.027) or small for gestational age (36.6% versus 23.5%, chi2 p<0.000).

Firstborn status was associated with reduced birth weight, length, and ponderal index, adjusted for gestational age (table 2). The relationship between continuously measured birth order and birth weight, length, or ponderal index, was best described by a third order polynomial model (table 2, figure 2), though the impact of birth order on length after adjustment was not significant at p<0.05. Higher order births were increasingly larger up to the sixth born. Birth weight then decreased as birth order increased. The final upward trend was likely spurious, due to the small sample sizes at that end of the birth order distribution. The model was not altered by the exclusion of two outliers with recorded birth weights under 1 kg. Young maternal age, height and AFA were important determinants of birth length but not ponderal index; and maternal smoking, like birth order, was an important determinant of ponderal index but not length.

Firstborn status and lower birth order were associated with WC and log odds of high WC (table 3). The association between birth order and WC (or log odds of high WC) was linear (versus the nonlinear association we observed between birth order and birth size). After adjustment for potential confounders, this relationship was attenuated. Maternal height and AFA were consistent, positive predictors of central obesity. We failed to detect non-linearities in these variables using quadratic terms that tested the hypothesis that there was risk at both ends of their respective distributions. Maternal smoking and age were not meaningful predictors of WC. Model results were not appreciably altered when the natural log of WC was used, nor when preterm birth (*yes/no*), small for gestational age (*yes/no*), or birth size variables were included as covariates (models not shown).

We detected a meaningful interaction between 2005 SES and birth order that was consistent with the mismatch hypothesis (table 4). For example, based on the estimated coefficients, a one SD increase in SES (2.9; observed range −3.4 to 16.0) would be associated with a 149%

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increase in odds of high WC (OR 2.49; 95% CI 1.65 to 3.76). However, the same increase in SES coupled with an increase in birth order would be associated with a 114% increase in odds of high WC (OR 2.14; 95% CI 1.58 to 2.89). The interaction could be viewed as a reduction in the positive impact of SES among individuals with higher birth orders (see figure 3). We found no evidence for a similar interaction between SES and the other prenatal variables (maternal age, height, AFA, smoking). We repeated our models, replacing 2005 SES with the same measure from 1983 to ascertain the relative importance of early versus later SES (model not shown). The estimated impact of early SES on central obesity was weaker than that of current SES, and there was no interaction between birth order and early SES in any model.

Mean BMI growth curves of four groups based on firstborn status and SES are displayed in figure 4. The high SES, firstborn group was characterized by low BMI at birth and rapid early postnatal gains in BMI. They had the largest mean BMI by six months, though at two years they were not distinguishable from the other high SES group. However, they had the largest increase in BMI across childhood and adolescence, resulting in the highest mean BMI in young adulthood. While BMI growth curves among high SES individuals were differentiated by firstborn status, there was no apparent impact of firstborn among lower SES individuals.

Discussion

The DOHaD paradigm broadly posits that environmental influences on prenatal and early postnatal development can alter physiology and/or behavior in a manner that increases risk of metabolic diseases, including obesity, in adulthood (23). Interest in the developmental origins of disease intensified after David Barker's observation that the geographical distribution of neonatal mortality in England and Wales in 1911–15 closely corresponded to CVD mortality from the same areas in 1968–78 (46). Because most neonatal deaths at that time were attributed to low birth weight, Barker hypothesized that poor fetal nutrition was acting to program the body's physiology in ways that adapted the offspring for a life of food insecurity while increasing "susceptibility to the effects of an affluent diet." While the DOHaD paradigm was initially met with a great deal of skepticism (47), it now finds a great deal of support, and DOHaD research has intensified over the past 20 years. This support is largely based on a large body of research illustrating that birth size is associated with later disease in a variety of human cohorts (23). However, it is now well understood that for the DOHaD paradigm to move forward, researchers must move beyond investigating birth size (48–51) and start testing specific hypotheses focused on upstream determinants of the fetal environment.

Our goal was to test the mismatch hypothesis, which posits that maternal constraint of fetal growth increases susceptibility to the obesogenic effects of modern environments. Maternal constraint refers to the set of normal, non-pathological factors through which the mother limits fetal growth (24). Maternal constraint of fetal growth is important to ensure that the developing fetus does not outgrow the pelvic canal of its mother (22). The impact of maternal constraint was illustrated most famously by Walton and Hammond (52) who found that upon cross breeding large Shire horses and smaller Shetland ponies, the size of the foal

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at birth was primarily dependent on the size of the mare. Gluckman and Hanson's focus on maternal constraint is notable because it is a normal process that operates in all pregnancies to some degree (24). This contrasts with the idea that the influences on fetal development which lead to later disease are caused by nutritional *insults* (53). The importance of nutritional insults has been well illustrated in studies which found that maternal exposure to the Dutch Hunger Winter (1944–45) during early pregnancy was associated with increased rates of obesity in young adult male offspring (25), and increased BMI and WC among 50 year old female offspring (26). Associations between maternal smoking and later offspring obesity have also been reported (28), further illustrating the potential impact of environmental insults during fetal development on later obesity. However, the idea that maternal constraint, a normal, non-pathological influence on the fetal environment, can impact offspring obesity has not been well investigated in humans (24). There is indirect evidence in the form of studies reporting an inverse association between birth size and later central adiposity (reflected by WC or skinfold ratios) (27); however birth size is a non-specific indicator of the fetal environment that reflects both normal and abnormal influences.

Among the forms of maternal constraint, we chose to focus on birth order and its impact on later obesity for several reasons. First, lower birth order is associated with reduced size at birth (32). We estimated associations between both firstborn status and continuously measured birth order with birth weight, length, and ponderal index. The results from our subsample of CLHNS males were consistent with earlier findings from an analysis of a larger subset of the CLHNS birth cohort: firstborns were both shorter and thinner at birth than higher order births (54). Other cohort studies have also confirmed that firstborns are both shorter and thinner at birth (e.g. 29). Our model results also indicated that the relationship between birth weight and birth order was best described by a third order polynomial relationship. While we posited that the increase in weights at the high end of the birth order distribution was likely spurious (due to small sample sizes (e.g. only 4% of males in the analysis sample were of birth order nine or above), it is interesting that a study of sheep found a similarly unexpected increase in birth weight at a parity of nine (55).

Previous studies have also reported associations between firstborn status and later obesity in adulthood. For example, firstborn status was associated with a four fold increase in odds of adiposity (skinfold thickness>85th percentile) in a cohort of young adult African Americans after adjustment for other perinatal measures including maternal BMI, education, and household size (35). Ravelli and Belmont, using data from a cohort of 19 year old Dutch males, found that being an only child was associated with obesity (56), though there was no apparent association between lower birth order and obesity in larger families. We found that firstborn status, and lower birth order in general, were associated with increased risk of central adiposity reflected by WC in young adult males.

However, under the mismatch hypothesis, the effects of birth order should be more apparent if the individual has experienced a nutritionally abundant postnatal environment. In high income countries, where nutritional energy abundance seems to be the norm, this interaction is less important (i.e. most people are experiencing the environment required to express the deleterious effect of reduced fetal growth). However, in a lower-income country such as the Philippines, where there is greater variation in nutritional environments and the prevalence

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of underweight is still considerable, the nature of this interaction is critical. By exploiting this environmental variation, we were able to test the mismatch hypothesis.

To test for this interaction, we used a measure of SES based on household assets in young adulthood as a crude but useful proxy measure for the obesogenic environment for males in our study. In previous analyses, we used a spatial clustering method to investigate obesogenic environments in this sample (57). We found that SES reflected in 2005 household assets was an important predictor of central adiposity in males that largely explained the spatial clustering (57) in high WC that we observed. Consequently, we concluded that the 2005 SES was a crude but useful measure to identify obesogenic environments, and took advantage of this to test the mismatch hypothesis in the CLHNS males. However, we found that the socio-environmental determinants of obesity were much more complex in the females, and at this point have not developed a useful way to estimate their exposure to obesogenic environments that would facilitate their inclusion in this analysis.

We confirmed our prediction under the mismatch hypothesis in the males: low birth order amplified the impact of SES on young adult central adiposity. While the idea that disease arises from a discordance between the pre- and postnatal nutritional environment is not a new one for DOHaD researchers, to our knowledge, only one previous study has explicitly tested for an interaction between prenatal variables and environment measured in young adulthood; Barker et al. found that the risk of coronary heart disease associated with low social class were amplified in men who were born thin at birth (58).

Rapid postnatal growth is also a hypothesized determinant of later obesity (29, 59). Because birth order is associated with higher maternal SES (due to reduced fertility among high SES women), our expectation was that firstborns would be more likely to experience an early postnatal environment that would promote rapid catch-up growth. This contrasts to other maternal constraints on fetal growth such as young age, or small body size, which tend to be associated with lower SES and thus a poorer postnatal environment that instead promotes growth faltering. We explored mean BMI growth curves in subgroups based on firstborn status and SES at birth, finding that being firstborn and having a high SES was associated with lower birth BMI followed by a rapid increase in BMI through early infancy. Furthermore, this group also had the largest mean BMI in young adulthood. The early trajectory was similar to that found among firstborns in the ALSPAC study who were thin at birth and also experienced rapid catch-up growth (29). However, due to the exploratory nature of this analysis, we were not able to determine whether the rapid postnatal growth we observed mediates the impact of birth order and SES on later young adult BMI, or whether rapid postnatal growth is a downstream indicator of aspects of fetal growth and development that impact later obesity risk independently of the postnatal growth pattern. We did try to test the hypothesis that early rather than later environment was a more important modifier of birth order by replacing SES in young adulthood with SES measured at birth in our interaction models. We found that estimated impact of early SES on later central obesity was weaker than that of current SES, and there was no interaction between birth order and early SES on young adult WC in any model. However, this is an admittedly crude attempt at elucidating the nature of this timing and more research is clearly needed.

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While our analysis failed to reject the mismatch hypothesis, we must interpret our results cautiously. Lower birth order is a complex exposure variable that is likely associated with a variety of pre- and postnatal factors that we were not able to account for. Birth order is linked to household size which may influence body size in adulthood. However, household size likely reflects different exposures at various points across the life course and thus its inclusion in our linear models is somewhat problematic. We tried to crudely account for this influence by including a measure of household size averaged across the study period (results not reported). While this inclusion attenuated the estimated effects of birth order on WC, it did not affect the interaction between birth order and SES. In a lower income country like the Philippines where underweight is still prevalent, it is also possible that higher WC associated with lower birth order is due to improvement in overall nutrition found in higher SES individuals from smaller families. We tried to account for this by estimating the impact of birth order on both continuously measured WC and on the upper end of the WC distribution. We also looked at the joint impact of birth order and SES on young adult height (results not reported), finding that continuously measured birth order and SES affected height similarly to WC, though the joint impact of firstborn status and SES seemed to be confined to WC. Another competing hypothesis that we did not explicitly test was the possibility that lower order births, particularly firstborns, are allocated more food during their childhood development (60, 61). However, this mechanism predicts a strong association between lower birth order and relatively better lifecourse nutrition (usually reflected by height) in large, poorer families whose overall nutritional environment is limited. Thus it seems unlikely to account for the increased risk of central obesity that our results suggest.

More research on the underlying mechanisms explaining the relationship between birth order, fetal growth, and later disease are clearly needed. If low birth order is truly related to increased disease risk in adulthood in the manner described by the mismatch hypothesis, then a better understanding of the biological mechanisms connecting birth order and fetal growth will likely provide important insights for intervention efforts. More research is also need to evaluate whether rapid postnatal growth is a mediator of the hypothesized impact of birth order on later obesity. This is especially important as birth order is clearly not a target for public health intervention. The interaction between pre- and post-natal nutritional environments requires more explicit testing in human populations, ideally using data from prospective longitudinal birth cohort studies. The global public health impact of the mismatch hypothesis with respect to birth order could be critical, as obesogenic environments become more common, and the proportion of lower order pregnancies among humans increases.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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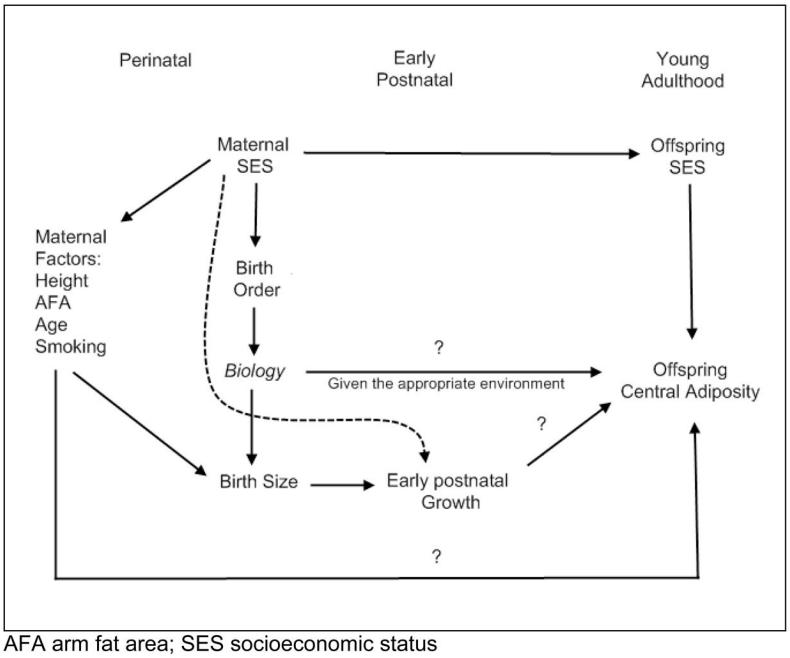

AFA arm fat area; SES socioeconomic status

Figure 1.
Theoretical model

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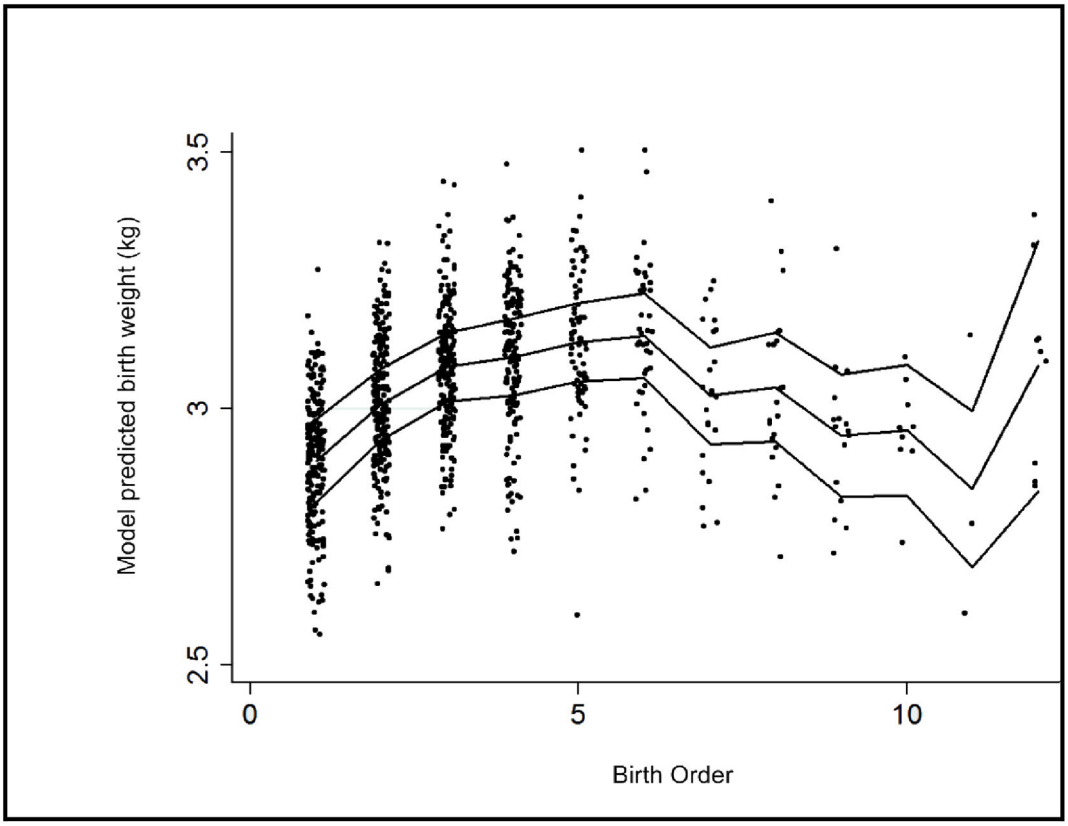

Figure 2.
Cubic relationship between birth order and model predicted birth weight (adjusted for maternal age, height, arm fat area, smoking and socioeconomic status).

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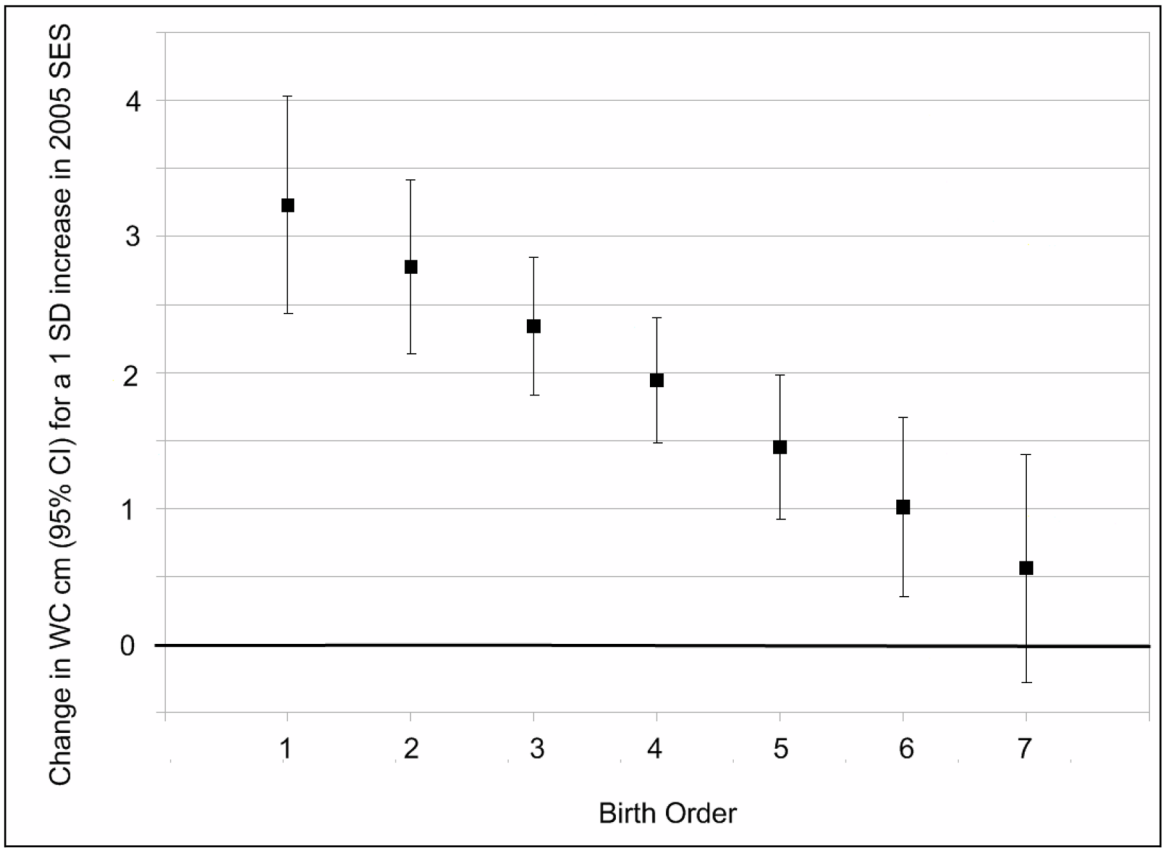

CI confidence interval; SD standard deviation (=2.9; observed range -3.4 to 16.0)

Figure 3.

The impact of young adult socioeconomic status on waist circumference is amplified for lower birth orders.

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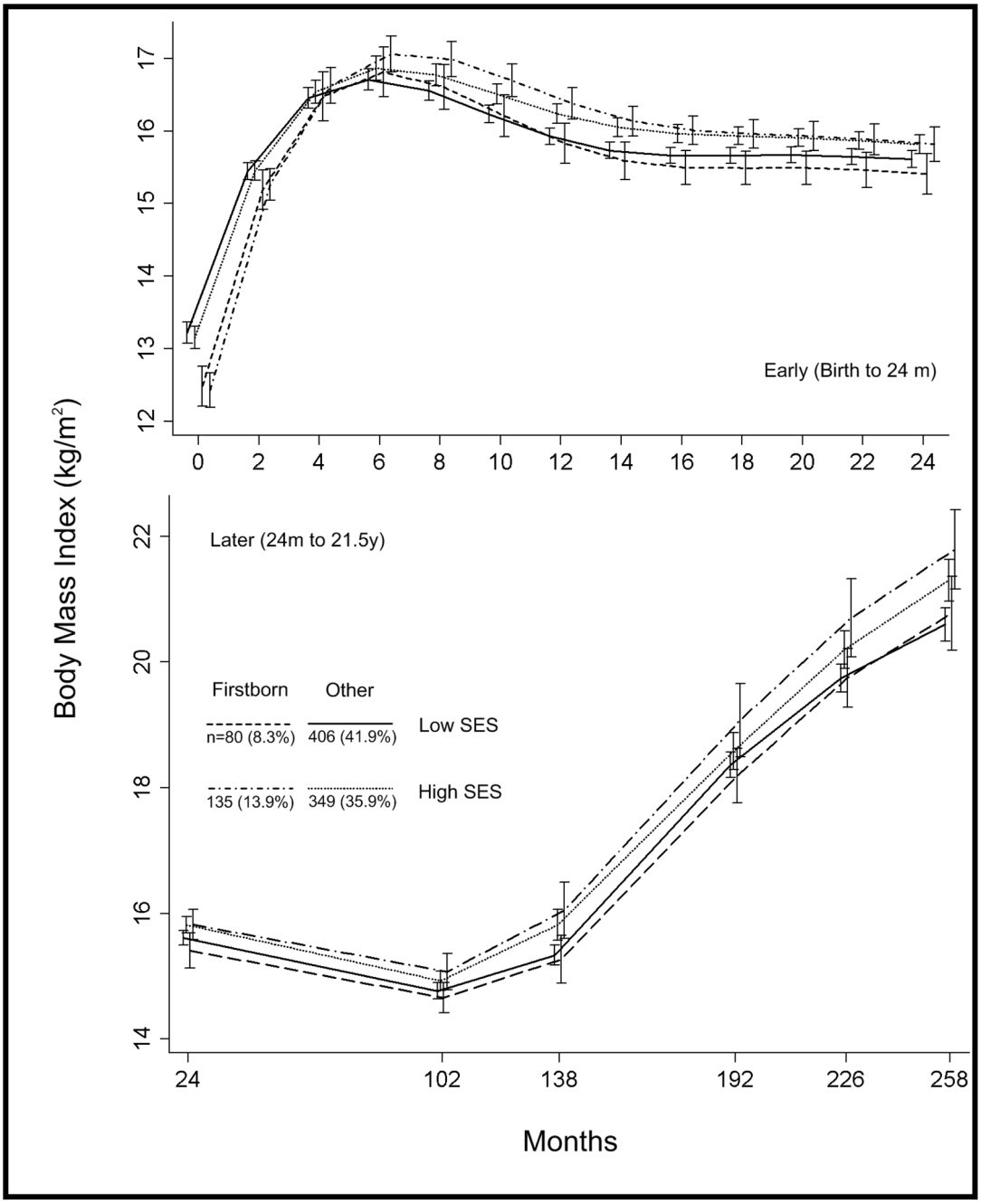

Figure 4.

Mean body mass index growth curves (with 95% confidence intervals) for groups of males defined by firstborn status and socioeconomic status at birth.

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Table 1

Sample characteristics for 970 young adult Filipino males enrolled in the Cebu Longitudinal Health and Nutrition Survey

Variables		Values	
Birth order	Median (Range)	3	(1 to 15)
Firstborn	%	22.2	
Waist circumference (cm)	$\text{Mean}_{(SD)}$ (Range)	$72.2_{(7.6)}$	(56.5 to 112)
High waist circumference (>85cm)	%	6.1	
Birth weight (kg)	$\text{Mean}_{(SD)}$ (Range)	$3.0_{(0.43)}$	(0.9 to 4.2)
Birth length (cm)	$\text{Mean}_{(SD)}$ (Range)	$49.3_{(2.0)}$	(39.74 to 55.5)
Ponderal index (kg/m^3)	$\text{Mean}_{(SD)}$ (Range)	$25.2_{(3.0)}$	(13.6 to 40.6)
Gestational age (wks)	$\text{Mean}_{(SD)}$ (Range)	$38.7_{(2.1)}$	(30 to 44)
Maternal age at birth (yrs)	$\text{Mean}_{(SD)}$ (Range)	$26.7_{(6.0)}$	(14.9 to 45.6)
Maternal height (cm)	$\text{Mean}_{(SD)}$ (Range)	$150.7_{(5.0)}$	(136.1 to 166.1)
Maternal AFA (cm2)	$\text{Mean}_{(SD)}$ (Range)	$14.8_{(5.5)}$	(3.8 to 50.6)
Smokes	%	12.4	
SES (1983)	$\text{Mean}_{(SD)}$ (Range)	$0_{(2.1)}$	(−2.0 to 8.0)
SES (2005)	$\text{Mean}_{(SD)}$ (Range)	$0_{(2.9)}$	(−3.4 to 16.0)

AFA arm fat area; SES socioeconomic status

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Table 2

Mutilivariable linear models estimating the impact of birth order or firstborn status on birth weight, length, and ponderal index.

	Dependent Variable					
	Birth weight (g)		**Birth length (cm)**		**Ponderal index* (kg/m^3)**	
Birth order models						
Birth order	157.27	(76.82 to 237.68)	0.14	(−0.25 to 0.52)	11.31	(5.49 to 17.10)
Birth ordersquared	−23.12	(−38.95 to −7.66)	−0.02	(−0.09 to −0.06)	−1.72	(−2.85 to −0.61)
Birth order cubed	1.02	(0.20 to 1.84)	0.0003	(−0.004 to 0.004)	0.08	(0.02 to 0.14)
Gestational age (wks)	44.67	(32.50 to 56.82)	0.22	(0.16 to 0.28)	0.77	(−0.11 to 1.65)
Maternal age <20 yrs	−47.15	(−131.47 to 37.17)	−0.51	(−0.91 to −0.11)	3.48	(−2.64 to 9.60)
Maternal age 20–35 yrs	REF		REF		REF	
Maternal age >35 yrs	−55.41	(−157.47 to 46.65)	−0.28	(−0.77 to 0.20)	−1.60	(−9.00 to 5.78)
Maternal height (cm)	11.70	(6.51 to 16.89)	0.07	(0.04 to 0.09)	−0.02	(−0.39 to 0.36)
Maternal AFA (cm^2)	7.12	(2.28 to 11.96)	0.04	(0.02 to 0.06)	−0.10	(−0.45 to 0.26)
Smokes	78.78	(−158.61 to 1.05)	0.13	(−0.25 to 0.51)	−8.81	(−14.59 to −3.02)
SES (1983)	6.60	(−6.29 to 19.50)	0.05	(−0.01 to 0.11)	−0.36	(−1.31 to 0.58)
Firstborn models						
Firstborn	−183.98	(−252.92 to −115.04)	−0.25	(−0.58 to −0.75)	−11.71	(−16.71 to −6.71)
Gestational age (wks)	44.74	(32.65 to 56.82)	0.22	(0.16 to 0.28)	0.80	(−0.09 to 1.68)
Maternal age <20 yrs	−45.61	(−128.69 to 37.46)	−0.46	(−0.86 to −0.07)	0.29	(−3.13 to 8.96)
Maternal age 20–35 yrs	REF	-	REF		REF	
Maternal age >35 yrs	−38.66	(−124.05 to 46.74)	−0.39	(−0.80 to 0.01)	0.19	(−4.26 to 8.14)
Maternal height (cm)	11.69	(6.52 to 16.86)	0.07	(0.04 to 0.09)	−0.03	(−0.41 to 0.34)
Maternal AFA (cm^2)	7.63	(2.81 to 12.44)	0.04	(0.02 to 0.06)	−0.05	(−0.41 to 0.30)
Smokes	−70.77	(−149.28 to 7.75)	0.10	(−0.27 to 0.48)	−7.54	(−13.26 to −1.82)
SES (1983)	7.16	(−5.58 to 19.90)	0.05	(−0.01 to 0.11)	−0.36	(−1.30 to 0.58)

*Coefficients reported as a change in independent variable associated with a tenth of a point change in ponderal index.

AFA arm fat area; REF reference category; SES socioeconomic status

Point estimates and 95% confidence intervals reported as Estimate (Lower Limit to Upper Limit)

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Table 3

Unadjusted and Mutilivariable linear models estimating the impact of birth order or firstborn status on waist circumference or log odds of high waist circumference.

	Dependent Variable			
	WC (cm)		OR High WC (>85cm)	
Unadjusted models				
Birth order	−0.36	(−0.57 to −0.15)	0.76	(0.64 to 0.91)
Firstborn	1.21	(0.08 to 2.34)	1.88	(1.06 to 3.34)
Adjusted birth order models				
Birth order	−0.51	(−0.76 to −0.27)	0.73	(0.59 to 0.90)
SES (2005)	0.66	(0.50 to 0.82)	1.22	(1.13 to 1.32)
Maternal age <20 yrs	−1.21	(−2.58 to 0.16)	1.19	(0.52 to 2.72)
Maternal age 20–35 yrs	REF	-	REF	-
Maternal age >35 yrs	1.52	(0.18 to 3.21)	1.68	(0.55 to 5.14)
Maternal height (cm)	0.23	(0.14 to 0.32)	1.11	(1.04 to 1.18)
Maternal AFA (cm^2)	0.19	(0.11 to 0.28)	1.07	(1.03 to 1.12)
Maternal Smoking	0.79	(−0.58 to 2.16)	1.55	(0.55 to 4.33)
Adjusted firstborn models				
Firstborn	1.51	(0.32 to 2.70)	1.71	(0.85 to 3.42)
SES (2005)	0.67	(0.52 to 0.83)	1.22	(1.13 to 1.32)
Maternal age <20 yrs	−1.11	(−2.56 to 0.35)	1.41	(0.59 to 3.35)
Maternal age 20–35 yrs	REF	-	REF	-
Maternal age >35 yrs	−0.11	(−1.58 to 1.36)	0.85	(0.31 to 2.34)
Maternal height (cm)	0.23	(0.14 to 0.32)	1.10	(1.04 to 1.17)
Maternal AFA (10 cm^2)	0.19	(0.11 to 0.27)	1.07	(1.02 to 1.12)
Maternal smoking	0.51	(−0.85 to 1.88)	1.28	(0.47 to 3.52)

AFA arm fat area; OR odds ratio; REF reference category; SES socioeconomic status; WC waist circumference

Point estimates and 95% confidence intervals reported as: Estimate (LowerLimit to Upper Limit)

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Table 4

Mutilivariable linear models testing whether associations between socioeconomic status and waist circumference, or log odds of high waist circumference, are modified by birth order or firstborn status.

	Dependent Variable			
	WC (cm)		OR High WC	
Birth order models				
Birth order	−0.56	(−0.80 to −0.32)	0.77	(0.63 to 0.95)
SES (2005)	1.10	(0.83 to 1.37)	1.37	(1.19 to 1.57)
Maternal age <20 yrs	−1.14	(−2.50 to 0.22)	1.23	(0.53 to 2.86)
Maternal age 20–35 yrs	REF	-	REF	-
Maternal age >35 yrs	1.52	(−0.17 to 3.20)	1.65	(0.54 to 5.08)
Maternal height (cm)	0.23	(0.14 to 0.32)	1.11	(1.04 to 1.18)
Maternal AFA (cm^2)	0.20	(0.12 to 0.28)	1.08	(1.03 to 1.13)
Maternal smoking	0.73	(−0.63 to 2.09)	1.42	(0.51 to 3.96)
Birth order X SES interaction	−0.15	(−0.23 to −0.08)	0.95	(0.90 to 1.00)
Firstborn models				
Firstborn	1.38	(0.19 to 2.57)	0.97	(0.40 to 2.36)
SES (2005)	0.55	(0.37 to 0.74)	1.14	(1.03 to 1.26)
Maternal age <20 yrs	−0.90	(−2.35 to 0.56)	1.76	(0.71 to 4.38)
Maternal age 20–35 yrs	REF	-	REF	-
Maternal age >35 yrs	−0.07	(−1.53 to 1.40)	0.86	(0.31 to 2.38)
Maternal height (cm)	0.23	(0.14 to 0.32)	1.10	(1.04 to 1.17)
Maternal AFA (cm^2)	0.19	(0.11 to 0.28)	1.07	(1.03 to 1.12)
Maternal smoking	0.41	(−0.95 to 1.78)	1.16	(0.41 to 3.14)
Firstborn X SES interaction	0.44	(0.10 to 0.79)	1.24	(1.04 to 1.48)

AFA arm fat area; OR odds ratio; REF reference category; SES socioeconomic status; WC waist circumference

Point estimates and 95% confidence intervals reported as (Lower Limit to Upper Limit)