Socio-economic variations

The effect of maternal socio-economic status throughout the lifespan on infant birthweight

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Summary

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The objective of this study was to investigate whether maternal socio-economic status during childhood and at the time of pregnancy each have unique associations with infant birthweight when biological determinants of birthweight are controlled. The data are from a three-generation study which contains information on the mothers and grandmothers of 987 singleton infants, collected over a period of 25 years. We used simple and multivariable regression to assess the association between indicators of a woman's socio-economic status and her offspring's birthweight.

Women who grew up in poor households had smaller babies than those who did not, and a unit increase in the income/needs ratio (analogous to the poverty index), in non-poor households only, was associated with a 185 g [95% CI 70, 200] increase in infant birthweight. Maternal age at the index infant's birth had a positive association with birthweight that diminished as women reached their mid-twenties. Among mothers with low education, high grandmaternal education was associated with a 181 g [95% CI 71, 292] increase in infant birthweight, while high grandmaternal education had no effect among infants whose mothers were relatively well-educated. This interaction between grandmaternal and maternal education is consistent with claims that cumulative stress is an important mechanism connecting maternal socio-economic status and infant health.

Keywords: *maternal childhood socio-economic status*, *maternal socio-economic status*, *maternal education*, *grandmaternal education*, *maternal age*, *birthweight*.

Introduction

Increased birthweight is associated with better infant health and lower infant mortality. Most biomedical scholars regard this association as causal, a fact that is disputed by some economists.[1] Moreover, some argue that birthweight is associated with positive outcomes beyond infancy.[2–4] Because of this, there has been a great deal of research on antecedents of birthweight. It is widely recognised that there are differentials in birthweight by maternal socio-economic status (SES), although there is still much we do not understand about the role of maternal SES as an antecedent of birthweight.

The dimensions of maternal SES that have received the most attention are income and education at the time of pregnancy.[4–6] Scholars hypothesise that educated women are more likely to seek out, understand and comply with medical advice on optimal behaviour during pregnancy. In addition, women with more income are more likely to have the resources to seek such advice, as well as to have the resources to comply.

Recently, Conley and Bennett have identified a fixed-effects model examining the effects of income at the time of pregnancy on birthweight within infant sibling groups, taking advantage of the fact that a mother's household income is often different at the time she gives birth to each of her children.[4] Their results indicate there is no effect of income on birthweight, and lead those authors to question the importance of SES – or at least income – as a determinant of infant birthweight. Conley and Bennett's fixed-effect approach, however, precludes the examination of maternal SES during childhood, because this cannot vary across a woman's children. Of course, this is not a problem if maternal SES in childhood has no effect on infant birthweight above and beyond its correlation with maternal SES at the time of pregnancy.

Whether or not socio-economic well-being in childhood has lasting effects on health across the lifespan is a question with wide currency among epidemiologists.[7] Preston and colleagues[8] have delineated four hypotheses about low SES in childhood and adult health. The first is the 'scarring' hypothesis, whose proponents argue that children with low SES are more likely to experience adverse health events with long-lasting consequences that directly and negatively affect health in adulthood. The second hypothesis, called 'acquired immunity', posits that adults who were low SES in childhood are more likely to have been exposed to infectious disease in childhood, and therefore predicts that they will exhibit higher levels of resilience to infectious disease in adulthood. The third hypothesis is called 'selection' and calls attention to how adults who survive a low-SES childhood might be 'positively selected' for good health. The difference between the selection hypothesis and the acquired immunity hypothesis is that the former emphasises underlying resilience rather than immunity to specific infectious diseases. Finally, the fourth hypothesis is called 'correlated environments'. Proponents of this last hypothesis argue that any association between childhood SES and poor health outcomes is spurious and merely the consequence of the fact that children from low SES backgrounds are more likely to be low-SES adults. According to this view, it is the latter (low SES in adulthood) that actually affects health.

In more recent models of socio-economic health disparities in reproductive and perinatal health, an additional hypothesis has been advanced regarding cumulative stress. [9–11] If the cumulative stress hypothesis is correct, then some people – those whose adult SES is higher than that of childhood – may 'outgrow' the health risks of low childhood SES when their environments improve in adulthood. Others, who enjoyed relatively favourable circumstances in childhood, are less vulnerable to the health risks of low adult SES if it occurs, because they have not experienced these environmental risks for their entire lives. The cumulative stress model suggests that there may be interactions between childhood and adult SES effects on health.

In this paper, we explore two questions. First, are both maternal SES in childhood and maternal SES at the time of pregnancy independently associated with infant birthweight? Second, are these associations additive, or are they multiplicative as the cumulative stress model would suggest?

A strength of our study is that we are able to control for important confounders of maternal SES, such as the mother's own birthweight, grandmaternal health, the mother's height and the mother's health status. While the factors we control for are not exhaustive by any means, most studies of maternal SES and birthweight have only rudimentary controls for biological confounders of SES.

The data for our study come from a three-generation cohort study of urban residents. Eighty per cent of the population was African American, and the 20% who were European American were highly disadvantaged. The persistent disparity in birthweight between African Americans and European Americans is well-known as is the fact that this disparity is narrower or even non-existent among low-income Americans compared with middle- or high-income Americans.[12,13] The inability of scholars to 'account for' this disparity by introducing controls for SES, and its relative narrowness or non-existence when low-income populations are examined, suggests that the determinants of birthweight, both biological and environmental, might operate differently among African Americans and low-income European Americans, a possibility that makes a focus on this population of high interest.

Methods

Study population

The database from which our study sample derives has been described in detail elsewhere.[14] It is from the Pathways to Adulthood Study (PAS), which comprises three groups of participants: (1) a group of women who presented for prenatal care at Johns Hopkins Hospital

from 1960 to 1964 (hereafter called G-1s for first generation); (2) the children whose births prompted their mothers' entry into the study (G-2s); and the children of the G-2 children (G-3s). The PAS is a follow-up of the Johns Hopkins Collaborative Perinatal Study (JHCPS), which was part of a larger research project called the National Collaborative Perinatal Project. Data for the PAS were collected in the early 1990s when the G-2s were between 27 and 33 years of age, but the database also includes the data collected at the G-2s' birth and the follow-up.

The PAS investigators determined that 2694 G-2s and 2306 G-1s from the JHCPS were eligible to participate in the 1990s round of data collection that constituted the PAS. Of those eligible, 2220 G-2s and 2014 G-1s were located. Of those located, 1758 G-2s and 1552 G-1s completed a full interview in the 1990s round of data collection. The response rate for the G-2s was 65.3% [(1758/2694) × 100]. The bulk of those not interviewed were never located (17.6%, $n = 474$). Other categories of G-2s who were not interviewed include those known to have died (3.2%, $n = 88$), those known to be alive for whom we obtained some critical pieces of information from a relative (2.6%, $n = 71$), and those not interviewed for other miscellaneous reasons (6.2%, $n = 168$). Only 5.0% of G-2s who were found refused to participate ($n = 135$). The response rate for the G-1s was 67.3% [(1552/2306) × 100]; as was the case with the G-2s, most of those not interviewed were never located (12.7%, $n = 292$). Of course, due to their older age, a larger percentage of G-1s were known to be deceased (10.2%, $n = 236$). Only 6.5% of G-1s who were found refused to participate ($n = 151$).

Study sample

The unit of analysis for this study was the G-3. We made a number of selections to arrive at our study sample.

We began with the 2694 G-2s eligible for inclusion in the PAS. We excluded G-2s without complete G-2 and G-1 interviews ($n = 1212$), male G-2s ($n = 674$), female G-2s who had no children ($n = 218$), female G-2s whose G-1 interview was not carried out with their biological mothers ($n = 29$); and G-2s whose mothers reported being neither white nor black at entry into the JHCPS ($n = 4$). The resulting sample of G-2s was 554.

These 554 G-2s reported bearing a total of 1131 G-3s. We excluded G-3s whose birthweights were not recorded ($n = 30$), and those G-3s who were reported by the G-2 as not being normal at birth ($n = 60$). Also excluded were G-3s who were twins ($n = 19$) and an infant whose reported birthweight was <500 g ($n = 1$). Finally, we excluded those whose mothers reported having diabetes ($n = 13$).

Our data on births are clustered at two levels. Within our study sample of 1008 G-3s, there are both siblings (all the children of a single G-2), as well as first cousins (all of the grandchildren of a single G-1 with multiple G-2s in the PAS). Dealing with this multiple clustering is not entirely straightforward using standard software. Therefore, we made one further selection to derive the sample upon which these analyses are based. In this sample, we selected the offspring of all G-2s who were the only female child of a G-1 in the JHCPS, and the oldest female G-2 in the cases where a G-1 had multiple female G-2s in the JHCPS. The result was a final analysis sample of 987 infants.

Data sources

The protocols for the JHCPS included collecting prospective data on: (1) the health of the G-1 during G-2's pregnancy; (2) the birthweights of all the G-1's previous children; (3) the socio-economic attainment of the G-1; and (4) the cognitive, physical, social and emotional development of the G-2 at regular intervals from birth to age 8. The protocols for the PAS consisted of a number of types of data collection from both the G-1 and the G-2, including: (1) a complete birth/parental history on the G-2; (2) the birthweights of all G-3s; (3) data on the health of the G-2; and (4) a life history calendar from which it is possible to get dates of all the G-2's co-residential sexual partnerships (including marriages), educational milestones (such as high school graduation or college attendance), and spells of welfare (AFDC) receipt.

Outcome

The primary outcome is infant birthweight in grams. In our study sample, birthweight of G-3s ranged from 680 to 5358 g with a mean of 3175, median of 3232, and standard deviation of 637. Infant birthweight was reported by the G-2s. While the length of the recall period varied from 18 years to <1 year, studies have shown that mothers' recall of infant birthweight is reasonably accurate.[15,16]

Covariates

We have five indicators of maternal SES during childhood. The first is an indicator of G-1 household income. PAS investigators constructed an index similar to the US poverty level based on the G-1's report of household income at the time of enrolment in the study. Just as in the case of the poverty level, a value of 1 indicates that the household income reported by the G-1 is equal to the year-specific poverty level for a household of her size. The second is G-1 education (less than high school, high school graduate or higher). The third is family structure (whether or not the G-2 lived with both parents from birth to 16 years of age). The fourth is G-2's number of siblings, and the fifth is programme participation (whether or not G-2's household received public assistance at either birth or age 7). All indicators of G-2 SES in childhood were reported by the G-1 in face-to-face interviews that took place during G-1's pregnancy with G-2 and again when the G-2 was 7 years of age.

We have four indicators of maternal SES at the time of her pregnancy with the index infant: G-2 education; family structure (whether the G-2 was married or in a co-residential union at the time of her pregnancy); maternal activity status and programme participation (whether the G-2 was: neither working nor getting public assistance; working only; working and getting public assistance; or not working and getting public assistance); and G-2's age at the time of the focal birth. All these measures were reported by the G-2 in the life history calendar or the face-to-face interview. The life history data were then linked to the date of the focal infant's birth provided in the birth history.

In the analyses presented below, we control for a number of potential confounders. These include: G-3 sex, G-2's adult height, G-1 pre-pregnancy body mass index, median birthweight of the G-1's children born before the G-2, the deviation of G-2 birthweight from the G-1's median and G-2 multipara. In addition to these sex and stature variables, we control for four factors measuring the health of the G-3's mother and grandmother, which might have affected infant birthweight. These are: G-1 had a sexually transmitted disease (STD) before or during pregnancy with G-2, G-2 hospitalised at least once between birth and age 8, whether or not G-1 smoked during pregnancy with G-2, and whether or not the G-2 smoked during pregnancy with infant. We did examine maternal race (African American or European American), but it had no effect on birthweight, so we dropped it.

Analytical approach

This report is concerned with associations between maternal SES and infant birthweight. The analyses proceeded as outlined below.

1 The coding of the variables was based on univariable analyses of the variable in question and the outcome to discover non-linearities. On the basis of these analyses, we divided G-1s into two educational groups: those with less than high school, and those with a high school diploma or more. We divided G-2s into two educational groups as well: those with some college or more, and those with a high school diploma or less. Our preliminary analysis indicated that spline specification was the best way to code the income/needs ratio (a continuous variable).[17,18] This specification permits the separate assessment of the effect of income for the poor and near poor (i.e. those with income needs ratios ≤ 1.5) and those who are not poor (i.e. those with income/needs ratios >1.5).
2 We used ordinary least squares regression to estimate the association of maternal SES in childhood and SES at the time of pregnancy with G-3 birthweight while controlling for potential confounders. Because our data are clustered at the family level (i.e. there are infants in our sample who are siblings), we adjusted for this clustering by using Generalised Estimating Equations (GEE) with robust standard errors.
3 Our model-building strategy consisted of entering variables in groups (e.g. G-2 health factors) to a model that included the infant sex, G-2 multipara and stature measures (median birthweight of the G-1's children, deviation of G-2's birthweight from this median and G-2 adult height). For the potential confounders, we only included those whose effects on birthweight were significant at the 0.10 level in preliminary multivariable analyses and which, when included, caused a 10% or more change in our estimates of the SES/birthweight association.
4 We checked for interactions between maternal SES in childhood and in adulthood.

Results

In Panel A of Table 1, we present an attrition analysis that suggests that our sample is unbiased in terms of SES. Among the 2694 G-2s eligible for inclusion in PAS, the percentage of African American was 81.7; the comparable percentage in our study sample of 554 was 82.5.

Table 1. Attrition analysis

A. SES differences between those who were included vs. not included	Eligible ($n = 2694$)	Study sample ($n = 554$)
Per cent G-2s African American	81.7	82.5
Per cent G-1s with high school education or more	25.0	25.2

B. Birthweight differences between G-3s excluded from sample and G-3s not excluded	G-3 median birthweight (g)
G-3 'normal' at birth	
Normal	3232
Not normal	2325
G-2 is diabetic	
Diabetic	3345
Not diabetic	3203

Note: G-1 denotes women enrolled in the Johns Hopkins Collaborative Perinatal Study in 1960–64. G-2 denotes children whose birth prompted G-1s' entry into the study. G-3 denotes the index children born to the G-2s.

Just over a quarter (25.2%) of the 2694 eligible G-2s had mothers with a high school diploma or more at their birth, while exactly 25% of the study sample of 554 were in this category.

In Panel B of Table 1, we present evidence regarding two of our G-3 exclusion criteria. We show that the median birthweight of 'normal' births was 3232 g, whereas the median birthweight for 'not normal' G-3s was 2325 g. It is usual in studies of birthweight to exclude infants with congenital anomalies, as many of these anomalies are known to cause fetal growth restriction and would therefore dilute analyses seeking to identify risk factors outside of the congenital anomaly pathway. As expected, G-3s born to diabetic mothers were heavier than others (a median of 3345 g, compared with 3203 g for non-diabetic mothers). Diabetes is known to be associated with higher birthweights but not necessarily healthy perinatal outcomes. Inclusion of births to women with diabetes may therefore dilute effects.

Table 2 contains the distribution of all variables in the analysis, as well as the mean and standard deviation of birthweight for each category of each variable. The test of significance refers to the results of ANOVA on birthweight using all the predictors in Table 1.

The results of the additive multivariable model are in Table 3. The use of GEE allowed us to estimate the correlation of birthweight among the siblings in our analysis that is not accounted for by the variables in our model: it was 0.23.

The G-1 health factors we considered and rejected for our final model included complications in the pregnancy with the G-2 and prior stillbirths or miscarriages before the G-2's birth. We also looked at a number of maternal anthropometry factors, such as G-2 head and chest circumference and placental weight. G-2 prior bad outcomes of pregnancy (stillbirths and miscarriages) did not affect infant birthweight and is not included in the model. Finally, we examined the effect of specific medical conditions of both the G-1 and the G-2 on G-3 birthweight, and only one (G-1 STD before or during pregnancy with G-2) was statistically significant. All the indicators of SES, however, are included in our final model as they are the variables of interest.

Mother's family structure in childhood, as indicated by co-residence with both biological/adoptive parents until age 16 and number of siblings, has no association with infant birthweight. When the income/needs ratio of the mother's household is held constant, infants whose mothers grew up in homes that received public assistance were, on average, 97 g [95% CI 3, 192] heavier than those who did not. Among the poor or near poor (those whose households were identified as being $\leq$1.5 of what they needed), there is no linear association between the income/needs ratio and birthweight. Among those living in households of adequate income, which we designate as having an income/needs ratio of >1.5, we see a significant linear association between the income needs ratio and birthweight; the coefficient indicates, for example, that mothers whose households at birth had income needs ratios of 2.5 delivered infants who were, on average, 185 g [95% CI 70, 300] heavier than those whose households at birth were 1.5. This additive model indicates that

Table 2. Birthweight by variables in the analysis

Variable	*n* (%)	Infant birthweight Mean (SD)	*P*-value
G-2 lived with both G-1s until age 16			
No	638 (64.5)	3175 (643)	
Yes	351 (35.5)	3175 (625)	0.545
G-2 number of siblings[a]			
1 or 2	149 (15.1)	3313 (565)	
3 or 4	382 (38.6)	3131 (565)	
5 or 6	283 (28.6)	3171 (644)	
7+	175 (17.7)	3158 (608)	0.028
G-2's household received public assistance in childhood			
No	573 (57.9)	3176 (664)	
Yes	416 (42.1)	3174 (597)	0.960
G-1 household income/needs ratio at G-2 birth[a]			
≤1.5 of poverty level	798 (80.7)	3163 (637)	
>1.5 of poverty level	191 (19.3)	3224 (637)	0.237
G-1 education			
Less than high school diploma	736 (74.4)	3129 (641)	
High school diploma or more	253 (25.6)	3309 (604)	<0.001
G-2 activity status at G-3 pregnancy			
Neither working nor welfare	246 (24.9)	3109 (653)	
Working only	318 (32.2)	3228 (586)	
Working and welfare	143 (14.5)	3129 (642)	
Welfare only	282 (28.5)	3193 (671)	0.124
G-2 marital status			
Single	458 (46.3)	3170 (701)	
Cohabiting	194 (19.6)	3129 (606)	
Married	337 (34.1)	3207 (557)	0.394
G-2 age at G-3 birth[a]			
≤17 years	119 (12.0)	3066 (732)	
18–24 years	545 (55.1)	3191 (596)	
≥25 years	325 (32.9)	3188 (663)	0.141
G-2 education at G-3 pregnancy			
High school diploma or less	678 (68.6)	3138 (645)	
Some college or more	311 (31.5)	3255 (610)	<0.001
G-3 sex			
Female	491 (49.6)	3119 (590)	
Male	498 (50.4)	3230 (676)	0.006
G-1 smoked during G-2 pregnancy			
No	581 (58.7)	3153 (607)	
Yes	408 (41.3)	3205 (676)	0.206
G-1 had sexually transmitted disease before or during G-2 pregnancy			
No	900 (91.0)	3187 (641)	
Yes	89 (9.0)	3056 (581)	0.065
G-2 hospitalised between birth and age 8			
No	723 (73.1)	3211 (626)	
Yes	266 (26.9)	3075 (655)	0.003
G-2 smoked during G-3 pregnancy			
No	417 (42.2)	3251 (581)	
Yes	572 (38.8)	3119 (669)	0.001
G-2 multipara			
No	498 (50.3)	3182 (647)	
Yes	491 (49.7)	3168 (626)	0.727
G-1 body mass index before pregnancy with G-2[a]			
Underweight	68 (6.9)	3069 (752)	
Normal	564 (58.1)	3177 (618)	
Overweight	242 (24.9)	3186 (613)	
Obese	96 (9.9)	3312 (605)	0.094
Median birthweight of G-2's older siblings[a]			
<2500 g	140 (14.2)	2878 (766)	
2500+ g	849 (85.8)	3224 (599)	<0.001
Deviation of G-2 birthweight from siblings[a]			
Smaller	128 (12.9)	3026 (707)	
About the same	757 (76.5)	3180 (631)	
Bigger	104 (10.5)	3321 (544)	0.002
G-2 adult height[a]			
≤5′1″	159 (16.1)	2960 (712)	
5′2″ or 5′3″	264 (26.7)	3126 (615)	
5′4″ or 5′5″	266 (26.9)	3273 (564)	
5′6″+	300 (30.3)	3243 (645)	<0.001

Note: G-1 denotes women enrolled in the Johns Hopkins Collaborative Perinatal Study in 1960–64. G-2 denotes children whose birth prompted G-1s' entry into the study. G-3 denotes the index children born to the G-2s.

[a]Denotes variables were entered into the model in continuous form.

Table 3. Adjusted[a] effects on index G-3 birthweight for G-2's childhood socio-economic status, and socio-economic status at pregnancy with the index G-3 infant

	Mean difference in birthweight (g) [95% CI]
G-2's childhood socio-economic status	
Childhood family structure	
Mother lived with both parents until age 16	–47.0 [–144.7, 48.7]
Mother did not live with both parents until age 16	0.0 Reference
Siblings	
Number of siblings	3.7 [–16.0, 23.4]
Household received public assistance	
Received public assistance	97.2 [2.7, 191.6]
Did not receive public assistance	0.0 Reference
Income/needs ratio of childhood household	
≤1.5	54.0 [–87.3, 195.3]
>1.5	84.0 [7.1, 160.8]
G-1's education at pregnancy with G-2	
High school diploma or more	117.3 [18.6, 216.1]
Less than high school diploma	0.0 Reference
G-2's socio-economic status when pregnant with index G-3	
Work status and public assistance receipt status	
Neither working nor receiving public assistance	–60.0 [–150.2, 30.4]
Both working and receiving public assistance	–38.8 [–157.8, 80.3]
Only receiving public assistance	54.5 [–43.1, 152.2]
Only working	0.0 Reference
Marital status	
Married	41.8 [–44.5, 128.2]
Consensual union	–30.2 [–123.0, 62.5]
Single	0.0 Reference
Mother's age at G-3's birth	
Linear	137.3 [32.7, 241.9]
Quadratic	–3.0 [–5.2, –0.7]
Mother's education when pregnant with G-3	
Some college or more at pregnancy	81.7 [3.7, 159.6]
High school diploma or less at pregnancy	0.0 Reference

Note: G-1 denotes women enrolled in the Johns Hopkins Collaborative Perinatal Study in 1960–64. G-2 denotes children whose birth prompted G-1s' entry into the study. G-3 denotes the index children born to the G-2s.

[a]Adjusted for all others reported in the table as well as infant sex, G-1's pre-pregnancy body mass index, the median birthweight of G-1's children, G-1 smoking during pregnancy with G-2, G-1 had a STD during or before pregnancy with G-2, G-2's deviation from the median birthweight of G-1's children, G-2's adult height, G-2 hospitalised before age 8, G-2's smoking during pregnancy with infant, and parity.

infants whose grandmothers had a high school diploma or more were, on average, 117 g [95% CI 19, 216] heavier than those whose grandmothers had less than a high school degree.

With respect to maternal SES at the time of the pregnancy, neither activity status (work and welfare receipt) nor marital status had an association with infant birthweight. Maternal age at pregnancy does have a non-linear association. At young maternal age, infants are heavier as age increases, but the association tapers off substantially by the time women reach their twenties (the addition of the quadratic term significantly improved the fit of the model: $\chi^2 = 7.85$, 1df). Mothers who had some college or more at the time of the infant's birth delivered infants 82 g [95% CI 4, 160] heavier than those infants whose mothers had a high school diploma or less.

We found one significant interaction between grandmaternal and maternal education. We included this interaction in our final model because its addition improved the overall fit of the model ($\chi^2 = 7.04$, 1df). Table 4 shows that, among infants whose mothers had relatively low levels of education, relatively high grandmaternal education had a significant positive association with birthweight: 181 g [95% CI 71, 292]. Relatively high grandmaternal education, however, has no association with birthweight at all among infants whose own mothers are relatively well-educated. We also checked for interactions between maternal SES and infant sex, and maternal SES and maternal birthweight, but none were found.

Discussion

We find that, in this urban, largely African American population, maternal SES exhibits associations with infant birthweight that are both statistically and clinically significant (i.e. approximately equivalent to the effect of smoking during pregnancy). These associations persist even after a set of biological factors such as average familial stature, grandmaternal health and maternal health are controlled. Many previous studies focusing on socio-economic effects on birthweight have not been able to control for these biological confounders.

We also find evidence that maternal SES in childhood and adulthood are each independently associated with infant birthweight. This finding does not support Preston *et al.*'s hypothesis of correlated environments.[8] The fact that the association of maternal

Table 4. Unadjusted and adjusted[a] effects of G-1's education by G-2's education on mean G-3 birthweight

	n	Mean G-3 birthweight (g)	Unadjusted mean difference [95% CI]	Adjusted mean difference [95% CI]
G-2 high school (HS) diploma or less				
G-1 <HS diploma	521	3077	0.0 Reference	0.0 Reference
G-1 HS diploma or more	157	3338	261 [224, 298]	181 [71, 292]
G-2 some college or more				
G-1 <HS diploma	215	3253	176 [151, 201]	131 [42, 221]
G-1 HS diploma or more	96	3261	185 [113, 257]	136 [–7, 279]

Note: G-1 denotes women enrolled in the Johns Hopkins Collaborative Perinatal Study in 1960–64. G-2 denotes children whose birth prompted G-1s' entry into the study. G-3 denotes the index children born to the G-2s.

[a]Effects reported adjusted for G-2's family structure in childhood, G-2's number of siblings, G-1 received public assistance during G-2's childhood, income/needs ratio of G-2's household in childhood, G-2's activity status at pregnancy with index G-3, G-2's marital status at pregnancy with index G-3, G-2's age at first birth, G-3 infant sex, G-1's pre-pregnancy body mass index, the median birthweight of G-1's children, G-1 smoking during pregnancy with G-2, G-1 had a sexually transmitted disease during or before pregnancy with G-2, G-2's deviation from the median birthweight of G-1's children, G-2's adult height, G-2 hospitalised before age 8, G-2's smoking during pregnancy with G-3 infant and parity.

childhood SES and infant birthweight is positive does not support either the acquired immunity or the selection hypotheses, as both these hypotheses predict a negative association between maternal childhood SES and birthweight.

The findings from our additive model offer some support for Preston *et al.*'s scarring hypothesis, whose proponents argue that low childhood SES results in permanent detriments to health that persist regardless of adult SES. The findings from our multiplicative model, however, are more supportive of a cumulative stress model. In this model, the health detriments associated with low SES are particularly pernicious when they are unrelenting across the lifespan.

Of course, our results are subject to some limitations. The most important is that we do not have very detailed information about the infants' health at birth (e.g. gestational age) such as one might obtain from a birth certificate. The 20-year lag between the most recent data collection and the penultimate one caused some attrition. Although all the measures of SES at the time of pregnancy that we include are precisely dated, we do not have data on income at the time of pregnancy as income is not well reported retrospectively.

With these limitations in mind, we call attention to two implications of our results for future research. First, studies that examine the effects of SES across the lifespan on health in adulthood – both intergenerational and intragenerational studies – should specifically examine whether or not the positive effects of growing up in a family that is relatively well-off are particularly important among those adults who have not themselves achieved high SES. Second, our findings call into question the utility of assessing the effects of maternal SES on infant birthweight by means of sibling comparisons that take advantage of intra-individual changes in maternal SES at the time of pregnancy with the index infant. Maternal SES in childhood, which cannot by definition vary across siblings, is held constant in such models, and they cannot tell us how it affects birthweight.

Among mothers who were born in households that were poor or near poor (an income/needs ratio of ≤ 1.5), there does not appear to be a gradient by income in birthweight, although these women do have smaller babies, on average, than women who were born into adequacy or affluence. Among the latter group (those mothers born into households with an income/needs ratio of >1.5), there is such a gradient. This finding underlines the idea that socio-economic health disparities are not merely the result of poverty. The fact that income exerted a more powerful effect among the non-poor than the poor has implications for public policy. It suggests that small increases in household income for those near the poverty level (such as those that might be accomplished by moving from depending on welfare to work in the low-wage sector) may not be important for intergenerational outcomes, such as birthweight of the next generation. In contrast, public policy that increases mothers' human capital such that

women can move from poverty or near poverty to adequacy or affluence, may have long-lasting consequences for those women's grandchildren.

In sum, our results advance our understanding of the association between maternal SES throughout the lifespan on infant birthweight by demonstrating: (1) that maternal SES effects persist when important confounders are controlled; (2) that maternal SES across the life course, as well as at the time of pregnancy, is associated with infant birthweight; and (3) that childhood SES may exert its strongest effects on infant birthweight among women whose SES at the time of pregnancy is low.

These findings are very encouraging in some ways. By achieving high levels of education themselves, women appear to be able to erase any disadvantage in their children's birthweight associated with growing up in a household with a poorly educated mother. Moreover, among women who fail, for whatever reason, to translate the resources in their childhood home that derive from a highly educated mother into high levels of education for themselves, their childhood advantage still protects their infants. Despite this good news, our findings underline the vulnerability of infants born into families that experience persistent, intergenerational disadvantage.

References

1 Almond D, Chay K, Lee D. The costs of low birth weight. *Quarterly Journal of Economics* 2005; **120**:1031–1083.

2 Leon DA, Koupilova I, Lithell HO, Berglund L, Mohsen R, Vagero D, *et al.* Failure to realise growth potential in utero and adult obesity in relation to blood pressure in 50 year old Swedish men. *British Medical Journal* 1996; **312**: 401–406.

3 Kramer MS. Invited commentary: association between restricted fetal growth and adult chronic disease: is it causal? Is it important? *American Journal of Epidemiology* 2000; **152**:605–608.

4 Conley D, Bennett N. Is biology destiny? Birth weight and life changes. *American Sociological Review* 2000; **65**:458–467.

5 Conley D, Bennett NG. Race and the inheritance of low birth weight. *Social Biology* 2000; **47**:77–93.

6 Cramer JC. Racial and ethnic differences in birthweight: the role of income and financial assistance. *Demography* 1995; **32**:231–247.

7 Kuh D, Ben-Schlomo Y. *A Life Course Approach to Chronic Disease Epidemiology*. Oxford: Oxford University Press, 1997.

8 Preston SH, Hill ME, Drevenstedt GL. Childhood conditions that predict survival to advanced ages among African Americans. *Social Science and Medicine* 1998; **47**:1231–1246.

9 Hogue CJR, Bremmer JD. Stress model for research into preterm delivery among black women. *Obstetrics and Gynecology* 2005; **192**(5 Suppl.):S47–S55.

10 Rich-Edwards JW, Grizzard TA. Psychosocial stress and neuroendocrine mechanisms in preterm delivery. *Obstetrics and Gynecology* 2005; **192**(5 Suppl.):S30–S35.

11 Geronimus AT. The weathering hypothesis and the health of African American women and infants: implications for reproductive strategies and policy analysis. In: *Power and Decision: The Social Control of Reproduction*. Editors: Sen G, Snow RC. Cambridge, MA: Harvard University Press, 1994; pp. 77–100.

12 CDC. Infant mortality and low birth weight among black and white infants – United States 1980–2000. *Morbidity and Mortality Weekly Report* 2002; **51**:589–592.

13 Starfield B, Shapiro S, Weiss J, Liang KY, Paige D, Wang X. Race, family income and low birth weight. *American Journal of Epidemiology* 1991; **134**:1167–1174.

14 Hardy JB, Shapiro S, Mellits ED, Skinner EA, Astone NM, Ensminger M, *et al.* Self-sufficiency at ages 27–33 years: factors present between birth and 18 years that predict educational attainment among children born to inner-city families. *Pediatrics* 1997; **99**:80–87.

15 Gofin R, Neumark I, Adler B. Birthweight recall by mothers of Israeli children. *Public Health* 2000; **114**:161–163.

16 Walton K, Murray L, Gallagher A, Cran G, Savage M, Boreham C. Parental recall of birthweight: a good proxy for recorded birthweight? *European Journal of Epidemiology* 2000; **16**:793–796.

17 Durrelman S, Simon R. Flexible regression models with cubic splines. *Statistics in Medicine* 1989; **8**:551–561.

18 Smith P. Splines as a useful and convenient statistical test. *The American Statistician* 1979; **33**:57–62.

