EARLY ORIGINS OF THE GRADIENT: THE RELATIONSHIP BETWEEN SOCIOECONOMIC STATUS AND INFANT MORTALITY IN THE UNITED STATES*

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Although relationships between social conditions and health have been documented for centuries, the past few decades have witnessed the emergence of socioeconomic gradients in health and mortality in most developed countries. These gradients indicate that health improves, although decreasingly so, at higher levels of socioeconomic status. To minimize problems with reverse causality, I tested competing hypotheses for observed socioeconomic gradients for infant mortality outcomes. I found no support for the income-inequality hypothesis and negligible support for the occupational-grade hypothesis. The results indicate that absolute material conditions are the most important determinants of socioeconomic effects on the risk of infant mortality and that while poverty has the most pronounced effect on risk, income is decreasingly salutary across the majority of the mortality gradient.

The relationship between socioeconomic status (SES) and health has been the focus of much epidemiological and sociological research for the past three decades (Adler et al. 1994). Basic conclusions that can be drawn from these studies are that (1) there is a graded relationship between measures of SES and health outcomes and behaviors in most developed countries (Marmot, Kogevinas, and Elston 1987); (2) health behaviors are one important mediator of this relationship but cannot explain the entire SES-health relationship (Adler and Newman 2002; Adler and Ostrove 1999); (3) the direction of causality also works from health to SES (Deaton 2002; Smith 1999), but empirical evidence from longitudinal data suggests that SES drives much of the observed differences in health (Chandola et al. 2003; House and Williams 2000); and (4) the social environment is related to individual health through effects that are independent of individual characteristics by conditioning and contextualizing individual responses to threats to health (Yen and Syme 1999).

Recent empirical research has begun to redefine the notion of the *gradient* (i.e., the graded relationship between SES and health) by arguing that the shape of the relationship between SES and health is actually curvilinear, such that there are decreasing returns to health at higher levels of SES (Backlund, Sorlie, and Johnson 1996; Ecob and Smith 1999). Although SES is not a theoretically nuanced concept in the social sciences and generally represents whatever it is that one is measuring, it is clear that various measures of SES have a relationship with health and that although the mediating (proximate) causes of differences in health and the actual health outcomes themselves may change over time, the fundamental relationship between measures of SES and health persists (Link and Phelan 1995).

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Although research that has documented the relationships between poverty and compromised birth outcomes has been widespread (for a review, see Kramer et al. 2000), it is less clear whether the mortality gradients that have been observed among adults exist for infants and, further, whether the shapes of these gradients are similar. In general, most studies have addressed adult mortality gradients in which the direction of causality may be difficult to disentangle (Deaton 2002). Only two previous studies have explicitly addressed whether socioeconomic gradients for health exist among adolescents (Brooks-Gunn, Duncan, and Rebello Britto 1999; Goodman 1999), and two recent studies have investigated the presence and nature of these gradients among infants in the United States (Conley and Bennett 2000, 2002).

Considering infants is extraordinarily important for many reasons. First, studying the intergenerational transmission of parental SES to infants' health minimizes selection effects, such that health cannot be said to solely determine SES. Although health problems among children may affect a family's income and the working status of the parents, outcomes among infants may not be subject to these same selection effects. However, because the passing of biologically determined social statuses through deleterious health behaviors may be a possibility (Conley and Bennett 2000, 2002), controlling for potential confounders, such as maternal birth weight, is important.

Second, the health and mortality status of infants has been called a synoptic indicator of the social conditions in a society (Gortmaker and Wise 1997)—the presence of disparities in mortality in the United States indicates that there is considerable improvement to be made. Third, the deaths of infants are sometimes avoidable and always tragic events for families, and eliminating disparities simply means that we can identify some social factors that are associated with increasing probabilities of death to attempt to close the gap between those at the bottom rung of society with those at the top (although ostensibly not by increasing rates among those at the top). For these reasons, the study of the social determinants of infant mortality can be seen as crucial, not only to our understanding of general patterns among adults, but as an object of study in their own right because infants are helpless and entirely subject to the social conditions into which they are born.

RESEARCH QUESTIONS

In this study, I sought to address four general questions: (1) is there a graded relationship between SES and mortality at the beginnings of life? (2) what is the empirical shape of any estimated relationship between SES and infant mortality? (3) what are some of the potential mediators between any SES-infant mortality relationships? (4) which hypotheses that explain the gradient are empirically tenable for infant mortality?

THE SES-HEALTH GRADIENT

Only two studies have explicitly documented whether SES disparities in children's health are similar to those observed among adults. In one study, SES gradients for both education and income were discovered for self-rated health, depression, and obesity, while only income was related to attempted suicide (Goodman 1999). In the other, large gradients were found between income and verbal ability; moderate relationships were found between income and socioeconomic achievement (e.g., completing high school, job status, and earnings) and between income and stunting and fighting behaviors; and no effects were found for a host of other developmental outcomes, such as obesity, anxiety, hyperactivity, and self-reported grades (Brooks-Gunn et al. 1999). One recent article investigated the presence of income gradients for low-birth-weight (LBWT) status in an ongoing panel study (Conley and Bennett 2000); the authors found no income or educational effects on the probability of LBWT among infants, net of parental birth weight (BWT).

Therefore, in the absence of a significant amount of data, it remains possible that differences in birth outcomes exhibit a purely threshold effect and that the high rates of

LBWT and infant mortality that have been observed are peculiar to poverty. The observed gradients among adults could be due to either a social-hierarchy effect that is not manifested until individuals at least partly determine their own health, or accumulations of poverty effects over the life course that begin to exhibit a graded nature in adulthood. However, both the presence of moderate SES-health gradients during adolescence (Chen, Matthews, and Boyce 2002) and data from the National Center for Health Statistics (NCHS) on raw rates of infant mortality by levels of maternal education suggest that gradients may exist even as early as birth (Pamuk et al. 1998). There is also reason to believe that because the health of infants is largely reliant on the health of mothers, the distribution of the risk of infant mortality may mimic mortality distributions among women.

Because differences in the health of adults are not peculiar to poverty, many hypotheses have been offered to explain the presence of SES gradients. An early hypothesis, offered by Wilkinson (1996), was that SES gradients are simply markers for an individual's position in a social hierarchy; this hypothesis would explain why absolute (average) differences in income explained little to none of the mortality gap *among countries*, but the levels of inequality *within a country* were highly predictive of these differences. Although Wilkinson (1999) later recognized that the observed shape of the income-health relationship was probably curvilinear, he still argued that the psychosocial effects of social hierarchies—mediated by stress processes—were the most important determinants of inequalities in health within developed countries (Marmot and Wilkinson 2001).

Kawachi (2000) further elaborated on this argument by noting that levels of *income inequality* within a country are related to health through the following processes: (1) high income inequality may lead to underinvestment in human capital, (2) high income inequality is associated with low social capital investments that may erode the social fabric, and (3) perceived disparities in income may work through psychological pathways to diminish health (e.g., relative deprivation). Although each mechanism may operate at different levels of aggregation (e.g., community, county, or state), most statistically significant effects of income inequality on health have been observed at the state level (Ellison 2002), which may lend credence to the first explanation—that a high level of income disparity in a state may lead to lower investment in human capital. Given that many policy and budgetary decisions are made at the state level, this area of aggregation is appropriate for this hypothesis. This finding is buttressed by the observation that the effects of income inequality are the largest, and frequently extant *only*, among the socioeconomically disadvantaged (Wagstaff and van Doorslaer 2000)—those who would be hurt the most by low levels of human capital investment within a state.

Also using Wilkinson as a point of reference, Marmot (2002) contended that income gradients are actually proxies for occupational gradients and that controlling for occupational grade may reduce income-health relationships to statistical nonsignificance. Marmot argued that these occupationally driven gradient effects are the result of the degree of social participation and the ability to "control one's destiny" that also exist along a continuum. Thus, Marmot recognized that while there may be diminishing health returns to the level of material comfort (i.e., income), social participation and control may "show no such threshold" (p. 33).

The hypotheses set forth by Kawachi (2000), Marmot (2002), and Wilkinson (1996) rely heavily on psychosocial stress processes as the cause of SES and health relationships, and simultaneously recognize the important—but secondary—role of absolute material standards in maintaining health. Log-linear income gradients are generally the empirical marker for these hypothesized relationships, such that increasing SES *always* guarantees better health, even at the extremes of the distribution. The prior assumption of the use of this functional form for income (i.e., the log transformation) always yields monotonic income-health relationships. These results, if they accurately reflect reality, are puzzling

because it is not intuitive that individuals with high incomes are still less healthy than those who are directly above them on the income distribution.

More recently, some authors have offered materialist arguments by empirically demonstrating that not only are there diminishing health returns to income but these returns flatten out toward the far right of the income distribution. Proponents of the material-conditions hypothesis have argued that income is an important indicator of health in and of itself, not simply because it is a proxy for other hierarchies; the presence of a declining or zero relationship at higher incomes indicates that status hierarchies may play an important role at the lower end and middle of the income distribution,[1] but are secondary to material conditions across the distribution, particularly at the poorer end of the distribution (Ecob and Smith 1999; Lynch and Kaplan 2000; Smith 1997).

LITERATURE REVIEW: CORRELATES OF INFANTS' HEALTH

Several risk factors for compromised birth outcomes and the subsequent risk of infants' deaths have been documented in the literature (Gortmaker and Wise 1997; Kramer 1987). Although all the factors that mediate these relationships are not known, several risk factors tend to covary with social status and may subsequently help to elaborate these relationships for birth outcomes. These factors are considered next.

At the most distal level, income and education are related to infant mortality and the risk of LBWT (Cramer 1995; Din-Dzietham and Hertz-Picciotto 1998), although the actual nature of this relationship has been virtually untested (cf. Conley and Bennett 2000). Race/ethnicity is also related to health outcomes, such that black infants die at more than two times the rate of white infants and have nearly three times the occurrence of LBWT than do white infants (Frisbie, Forbes, and Pullum 1996; Pamuk et al. 1998). On the other hand, in spite of poor socioeconomic conditions, Hispanic infants have similar rates of both LBWT and infant mortality as do white infants (Hummer et al. 1999; Markides and Coreil 1986). However, income and education do not sufficiently explain racial differences in mortality (Hummer 1993), in large part because they do not fully account for differences in social position.

Additional risk factors include access to medical care and social services. Infants of women who self-pay for their births, or are uninsured, appear to suffer a higher risk of death (Carrasquillo et al. 1999; Moss and Carver 1998). Two programs that help protect the health of pregnant women and infants—WIC (Special Supplemental Nutrition Program for Women, Infants and Children) and Medicaid—have had mixed results, with the use of WIC associated with lower odds of infant mortality, but Medicaid associated with higher odds (Moss and Carver 1998).

With regard to sociodemographic variables, mothers who are married have nearly half the infant mortality rate of nonmarried women, but this advantage is probably confounded with SES, access to health care, and social support (MacDorman and Atkinson 1999). The relationship between maternal age and infant mortality is curvilinear, with higher rates of infant mortality among the babies of teenage and older mothers. However, this relationship varies by race/ethnicity, such that risks tend to accumulate with increasing age among black women (Geronimus 1992).

Behavioral characteristics can be conceptualized as part of a much larger set of intervening factors that mediate the relationship between social factors and the risk of infant mortality. Socioeconomic variables shape the population distributions of these

1. Given that materialist arguments are often grounded in Marxist frameworks, although they are not necessarily Marxist, there is clearly room for psychosocial effects on health that are independent of material effects. See, for example, Marx's (1844/1978) explanation of alienation processes in the labor market. For Marx, alienation is rooted in class structures and therefore has a material basis; nonetheless, it is possible that the psychosocial effects of alienation, for example, may not be completely captured by the level of wage remuneration (i.e., income) an individual receives.

proximal risk factors by determining access to resources that can be used to avoid risks or to minimize the consequences of risky behaviors. Cigarette smoking is the most important known modifiable risk factor for LBWT and infant mortality, given its high prevalence rates in comparison with other types of substance use (Chomitz, Cheung, and Lieberman 1995). Smoking during pregnancy restricts the flow of oxygen to the fetus and can result in a multitude of poor birth outcomes (e.g., respiratory problems and intrauterine-growth retardation).

Besides smoking, little is known about the net effect of the broader range of substance-use variables during pregnancy at the population level. Alcohol is the second-most-used substance during pregnancy, and its effects may have profound consequences that may be manifested in fetal alcohol syndrome. The effects of alcohol use are dependent on the age of the mother, as well as the amount of alcohol she consumed during pregnancy, with older women who are heavier drinkers more likely to give birth to infants who suffer from the effects of fetal alcohol syndrome (Jacobson et al. 1998). The relationship between the use of illicit drugs and infant mortality (Wise 1993) is plagued by methodological problems, such as small nonrepresentative samples and unreliability of self-reports. It is also difficult to separate the effects of drug use from the generally poorer health of addicted women. Substance use during pregnancy may have direct effects on fetal development and health, yet may also indicate social disorganization and/or abuse in the home that may ultimately affect the risk of infant mortality long after the neonatal period.

One of the key behavioral characteristics that has been cited as being responsible for the largely favorable birth outcomes of Hispanic infants is a healthy maternal diet (Guendelman and Abrams 1995). Maternal weight gain during pregnancy is an indirect measure of the intake of nutrients. Weight gain is influenced by maternal dietary intake, prepregnancy weight and height, the length of gestation, and the size of the fetus (Chomitz et al. 1995); low or inadequate weight gain may thus reflect poor nutritional status. In addition, appropriate exercise during pregnancy and the use of vitamins are also seen as positive lifestyle practices that lower the risk of poor birth outcomes. Prenatal nutrition, exercise, and health may also be highly correlated with postnatal nutrition, health, and exercise and even with nutrition patterns for infants (especially if the mothers are breast-feeding); therefore, the effects of prenatal behaviors may also extend beyond the neonatal period as proxy indicators for postnatal behaviors.

Another factor that is thought to influence maternal health and its effect on the risk of infant mortality is the use of prenatal care. Although the empirical evidence on the relationship between prenatal care and adverse pregnancy outcomes is mixed, most researchers have agreed that its efficacy extends beyond birth outcomes into overall maternal and child health (Fiscella 1995; Goldenberg, Patterson, and Freese 1992) and may even reflect future access to—and use of—public health and health care services.

Finally, maternal health during pregnancy is a key intervening factor for the risk of infant mortality and LBWT. Previous loss of a fetus during pregnancy (hereafter pregnancy loss) is indicative of a higher risk of infant mortality because a woman who previously lost an infant is at a greater risk of having a compromised birth or of her infant dying (Eberstein et al. 1990). Attempts to prevent an early delivery indicate maternal health problems that may increase the risk of adverse birth outcomes. Furthermore, body mass is indicative of a woman's prepregnancy body weight, as well as past and present nutritional and genetic factors. Recent studies have also highlighted that biological inheritances may be passed on through parental BWT and its simultaneous effect on SES and poor health behaviors during pregnancy (Conley and Bennett 2000, 2002).

DATA AND METHODS

The data set chosen for these analyses was the National Maternal and Infant Health Survey (NMIHS), conducted in the United States in 1988 (NCHS 1991). This data set contains a

unique combination of information on vital records (e.g., birth certificates) and survey data that are combined as a sample of nationally representative births among women in 1988 (Sanderson, Placek, and Keppel 1991). The probability of the selection of live births was 1 of every 354 and for infant deaths, 1 of every 6 (Sanderson et al. 1991). The NMIHS oversampled black infants and low and very low birth-weight infants.

Although this data set is dated, it represents the richest national data set drawn from vital records for investigating the social determinants of birth outcomes. The anticipated arrival of the Early Childhood Longitudinal Study—Birth Cohort 2000 should help to supplement the NMIHS. In the meantime, aside from the NMIHS, only annual natality files (i.e., "linked birth-death files"), with limited sociodemographic characteristics, or data from the Pregnancy Risk Assessment Monitoring System, with limited regional samples, are tenable for analyzing disparities in LBWT and infant mortality.

Dependent Variables: All-Cause, Endogenous, and Exogenous Infant Mortality

The key dependent variables included (1) an overall category of all-cause infant mortality (unweighted deaths = 3,795), (2) deaths that were due to endogenous causes (unweighted n = 2,732), and (3) deaths that were due to exogenous causes (unweighted n = 1,063). I used the same cause-of-death coding scheme for endogenous and exogenous causes as that used by Hummer (1993). Furthermore, I specified separate models to capture separate death processes; in short, "exogenous deaths are those related more to environmental and external causes (such as infections), while endogenous deaths occur due to the genetic makeup of the infant, the circumstances of life in utero, and the conditions of labor" (Hummer 1993:535). These distinctions group common causes of death into similar categories to allow a more detailed analysis of the disparate correlates of each type of death.

Regression Models

Three separate logistic regression models were specified for each of the three outcomes just discussed. It is possible to estimate predictors of the cause-of-death groupings (survived versus death that was due to endogenous causes and survived versus death that was due to exogenous causes) with a multinomial logistic regression that would be more efficient than would separate binary logits[2] (Long 1997). However, doing so would prevent the use of distinct functional forms for each of the key predictor variables. Given that these functional forms were crucial to the research agenda, I specified binary logits for each cause-of-death grouping, with survival past the first year of life representing the reference group. In short, a nominal level of efficiency was traded for a better model fit through the specification of distinct functional forms of SES for both endogenous and exogenous deaths.

Furthermore, the NMIHS was conducted using a complex sampling design that oversampled for LBWT and black infants; therefore, to correct standard errors, the use of design-effects adjusted models was appropriate. All models specified in these analyses used the *svylogit* regression command in Stata 7.0 (StataCorp 2001) to apply appropriate weights and to adjust for the sampling effects to obtain an accurate estimation of standard errors. Log-likelihood estimates were obtained from models using conventional estimation procedures (i.e., the *logit* command in Stata) because the *svy* estimators in Stata are not reliant on "true likelihoods" owing to clustering and the dependence of observations within sampling strata (StataCorp 2001). The following cases were excluded from the

2. Estimates were obtained using both binary logit regression and multinomial logit regression, and other than differences in model fit (possibly because of specification errors in the functional forms of the key predictors in the multinomial models), the key results and conclusions were virtually indistinguishable statistically and substantively.

analyses for overall infant mortality: women who were not white, black, or Hispanic; had multiple births; extraordinarily LBWT infants (less than 500 grams); very high BWT infants (more than 8,165 grams); infants who were born at fewer than 22 weeks of gestation; and infants whose gestational ages were unknown.[3] The final sample sizes included 12,814 infants in the all-cause analyses, 11,751 infants in the endogenous-death analyses, and 10,082 infants in the exogenous-death analyses.

Predictor Variables

The first key independent variable was a measure of total household income that was adjusted for nonincome payments, such as Supplemental Security Income, food stamps, and Aid to Families with Dependent Children. The original metric of the variable contained income categories, so category midpoints were taken to represent raw household income. This variable was measured in the year before a woman gave birth and thus (hypothetically) included the three months before pregnancy, as well as the roughly nine months of gestation. In addition to including nonwage income, this measure was adjusted for household size by dividing household income by a size elasticity raised to .38 (Rogers, Hummer, and Nam 2000). Therefore, income in this study was measured as Income / (Household Size)$^{.38}$; this method assumes diminishing costs for additional family members. However, there were no measures of household debt or wealth, and the failure to control for wealth and debt could have biased the estimation of the income effects.

To determine the best functional form, I used a fractional polynomial regression[4] for each of the three outcome variables (Becketti 1995). These techniques rely solely on the data points of the NMIHS data set; however, because of the lack of theoretically meaningful functional forms that could be chosen a priori, a purely empirical justification was needed.[5] Using the full set of covariates and weighted data, I used the following sets of polynomial expressions for the adjusted measure of household income: (1) total infant mortality, income$^{-.5}$, and income; (2) endogenous Infant mortality, income$^{-.5}$, and income^{3}; (3) exogenous infant mortality, income, and income^{2}. The gross (unadjusted) relationships between each of these income specifications and the predicted probabilities for each of the three dependent variables are plotted in Figure 1. Plotting the predicted values for each of the outcomes between the 10th and 90th percentiles of the income distribution yielded gradients that flatten out to the far right of the distribution.

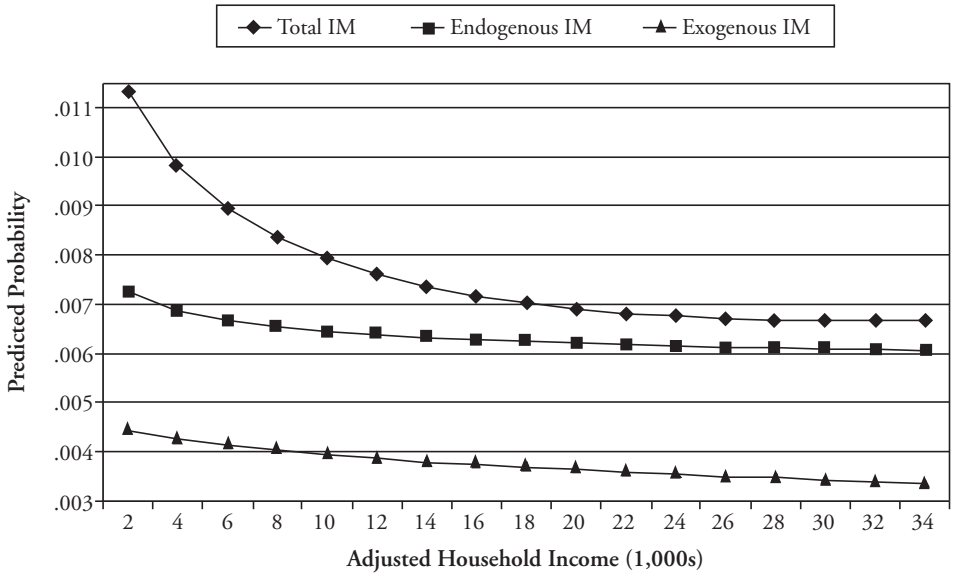
The second key socioeconomic variable, *occupational grade*, was proxied by the Nakao and Treas (1990, 1994) occupational prestige score (NTOP). Rather than being an imputed value (i.e., from the educational and income level of occupational incumbents), this measure uses actual assessments of occupational prestige (OP) for over 500 occupations using data from respondents to the 1989 General Social Survey. Because data are

3. Additional exclusions for models of endogenous infant mortality included those just listed, as well as those who died from exogenous causes. Similarly, models of exogenous infant mortality excluded those who died from endogenous causes.

4. I used this approach (*fracpoly* in Stata 7) to take advantage of the flexibility of atypical polynomial expressions that capture smoother relationships between variables. As the supporting documentation for these models suggests, "linear and quadratic functions are severely limited in their range or curve shapes, whereas cubic and higher order curves often produce undesirable artifacts such as 'edge effects' and 'waves'" (StataCorp 2001:544). Linear splines were another option considered, but given that splines require linear relationships *between* the prespecified "knots" and that there may be no a priori reason for discontinuities in the relationship between income and mortality, I relied on the fractional polynomial approach. See Royston and Ambler (1999a, 1999b) and Becketti (1995) for a more thorough description of this approach.

5. Previous analyses indicated that some form of curvilinear relationship between income and mortality is appropriate (Ecob and Smith 1999), but the polynomials obtained from the fractional polynomial regression consistently outperformed (e.g., provided equal or better fits than did logged income) conventional transformations of income (i.e., linear, log-linear, and quadratic).

Figure 1. Predicted Probabilities for Infant Mortality (IM) by Cause of Death: Unadjusted Income Relationships (Model 1)



available from both the mother and father of an infant, several different operationalizations of OP were considered, including the mother's only, the father's only, the higher of the two (unless the mother was unmarried or did not work), and an average of the two scores. *The highest of the two scores* was chosen for the primary analyses, although each operationalization was considered, especially as it related to hypotheses regarding causes of the gradient. Although the scores between the mother and the father are correlated, they generally are not highly correlated enough to cause multicollinearity problems when they are used in the same model; however, much like income, OP may best be defined as a resource that the whole family can draw on, and using a combined score yielded the best results.[6] On the basis of a series of analyses of gross relationships and model fit, the best functional form was a log-linear transformation of the raw score to capture diminishing risk returns across the distribution of scores.[7]

The third key socioeconomic variable was a measure of income inequality at the state level. Although it would seem more desirable to use a more disaggregate level of analysis, confidentiality issues prohibited me from determining anything other than the state of birth of infants. The measure of income inequality was the Gini coefficient, which ranged from a score of perfect income *equality* (0) to a score of perfect income *inequality* (100).

6. Measuring occupational grade by other means, such as the Duncan Socioeconomic Index, for example, yielded less-promising results and did not change the substantive conclusions of this study. Therefore, although alternative operationalizations were considered, the NTOP outperformed all the other measures.

7. I did use a fractional polynomial regression approach to the OP measures (and the income-inequality measure), but tolerance levels were extraordinarily low when more than one order polynomial was included in the model. Owing to the already tenuous relationship with infant mortality, the most parsimonious and best-fitting form for OP was determined to be a natural log transformation.

Internationally, Gini coefficients range from 25 in Denmark, Austria, and Belarus to more than 60 in Brazil and Sierra Leone (Ellison 2002). However, within the United States (mean ≈ 45) the range is attenuated to scores of 38.5–49.2. A state-level Gini coefficient was appended to each mother/infant file in this study. In addition, because average income is strongly and negatively correlated with income inequality, a measure of median state household income was included to ensure that any observed effects are truly inequality effects, rather than state-level average income effects. The best functional form—although it was difficult to make assessments, given that virtually no raw relationships existed between income inequality and infant mortality—was also a log-linear transformation.

Because education is a precursor to both income levels and occupational attainment, these variables were treated as controls and appear in the model simultaneously with the SES measures discussed earlier.[8] Years of mother's education completed at the time of childbirth and the number of years of education completed by the father were both included. These variables are linearly related to the raw probabilities of infant mortality and were therefore left in their original metric, years. Although correlated, tolerance levels indicate that the inclusion of both variables in the same model will not inflate variance estimates substantially.

Sociodemographic characteristics, including race/ethnicity, parity/age, marital status, and insurance status, were controlled for. Race/ethnicity was a dummy variable representing the race/ethnicity of the mother as non-Hispanic black, Hispanic, or non-Hispanic white. Parity, operationalized using the Kleinman and Kessel index (1987), took into account the interactions between birth order and maternal age (first birth, low parity, and high parity).[9] Marital status was a single dummy variable indicating whether a woman was married at the time of birth, and insurance status was represented by three dummy variables (self-pay, Medicaid, and private insurance).

A set of behavioral variables included participation in WIC, weight gain during pregnancy, adequacy of prenatal care, and key health behaviors (smoking, drinking, exercise, and use of vitamins). Participation in WIC was a dummy variable in the models; short-term WIC participation was any term less than six months, and long-term WIC participation was six months or more. Weight gain during pregnancy calculated the difference between weight in pounds at the time of pregnancy and weight in pounds at the time of childbirth; this variable included weight gains of 0–15 pounds (low), 16–40 pounds (normal), and 41 or more pounds (high). Prenatal care was measured using Kotelchuck's (1994) Adequacy of Prenatal Care Utilization Index, a four-category measure that distinguishes the "adequate plus" group of women who record a higher level of care than that recommended by the American College of Obstetrics and Gynecologists. It included the following categories of prenatal care: (1) missing/no care, (2) inadequate care, (3) intermediate care, (4) adequate care, and (5) adequate-plus care. Dummy variables were added to the equations to indicate whether mothers engaged (or failed to engage) in any of the following health behaviors: (1) smoking (any amount), (2) drinking alcohol (an average of two times a week or more), (3) no vitamin use, and (4) no exercise.

8. Using such a broad set of measures of SES may lead to collinearity problems, artificially inflate variance estimates, and lead to the wrong conclusions regarding the tests of the hypotheses. Therefore, tolerance statistics for each set of SES variables were computed, although the results are not presented. In short, problems that were due to collinearity were observed largely for the use of several polynomial instances of the income measure (all-cause infant mortality and exogenous infant mortality, in particular). However, given the strength of the association between income and infant mortality, variance inflation does not affect the tests of the hypothesis. There are high-enough tolerances with respect to the other SES variables to support a simultaneous analysis of multiple measures of SES in the same model.

9. Parity is defined as high in third or higher-numbered births to women who are younger than age 25 and fourth or higher-numbered births to women aged 25–29. All other births are considered low parity (Kleinman and Kessel 1987:750).

Table 1. Descriptive Statistics

Variable	Mean	IMR	$\rho_{x,im}$
Total Sample		7.5	
Mother's Education (years)	12.66		–.015**
Father's Education (years)	12.83		–.014**
Income (1,000s)	17.82		–.019**
NTOP[a]	40.45		–.014**
Income Inequality (Gini)	44.10		.001
Median State Household Income (1,000s)	29.62		–.007**
Race/Ethnicity (non-Hispanic white)		6.4	
Non-Hispanic black	0.16	13.3	
Hispanic	0.13	6.4	
Parity (1st birth)		7.3	
Low	0.44	6.5	
High	0.14	11.0	
Marital Status (married)		6.6	
Unwed	0.26	10.2	
Payer for Birth (insurance)		6.1	
Medicaid	0.27	10.6	
Self-pay	0.13	7.9	
WIC Participation (no)		7.3	
Short term (< 6 months)	0.17	8.1	
Long term (6+ months)	0.13	8.1	

(continued)

Two additional variables that were added to the models were whether or not early delivery was prevented during the pregnancy and the mother's body-mass index—operationalized as low (9.0–19.8), medium (19.9–26), and high (greater than 26). A control for mother's LBWT status was added that represents a mixture of potentially biological, genetic, and social factors that may have an independent effect on birth outcomes and may be correlated with the income measure. Finally, a control for infants' BWT was added to determine whether there are independent effects of SES or whether these effects work largely through BWT status. Measures of continuous BWT (in grams) and BWT squared were added to the models to capture the reverse j-shaped relationship between BWT and the risk of infant mortality. That is, whereas increasing BWTs are associated with declines in the risk of infant mortality across nearly the entire distribution, excessive BWTs are associated with an increase in the probability of death (Frank, Frisbie, and Pullum 2000).[10] Descriptive characteristics for all these variables are presented in Table 1.

10. Some of the relationship between SES and infant mortality is expected to work through its effects on BWT; however, it is also expected that SES may have independent effects on infant mortality, given its relationship to levels of stress, coping abilities, sociointeractional processes, and ability to gain access to material and social resources. Furthermore, given that not all LBWT infants die, for example, it is possible that BWT moderates the relationship between SES and infant mortality. That is, LBWT may represent a significant risk factor that can be effectively "managed" by infant care. This hypothesis may be more salient, however, for instances of exogenous deaths because many endogenous deaths are due to congenital malformations that may be difficult to "overcome," regardless of the level and quality of care. Nonetheless, the effects of SES on infant mortality, net of BWT and contingent on BWT (interaction effects) were estimated.

(Table 1, continued)

Variable	Mean	IMR	$\rho_{x,im}$
Pregnancy Weight Gain (16–40 pounds)		6.2	
0–15 pounds	0.13	18.9	
40+ pounds	0.23	5.1	
Prenatal Care (adequate)		4.8	
Missing (no care and missing)	0.04	23.5	
Inadequate	0.10	10.5	
Intermediate	0.17	5.0	
Adequate plus	0.32	9.3	
Smoke During Pregnancy (no)		6.8	
Yes	0.23	10.0	
Alcohol Use (no)		7.5	
Yes	0.03	10.8	
Vitamin Use (no)		9.8	
Yes	0.10	7.3	
Exercise (no)		8.5	
Yes	0.57	6.3	
Prevent Early Delivery (no)		6.2	
Yes	0.26	11.4	
Previous Loss (no)		7.3	
Yes	0.22	8.5	
Body Mass Index (medium)		7.0	
Low	0.24	7.8	
High	0.18	9.0	
Mother's Birth Weight (normal)		7.4	
Mother's Low Birth Weight	0.09	8.6	
Mother's Birth Weight (missing)	0.27	8.7	
Infant's Birth Weight (grams)	3,337.46		–.168**

Notes: Proportions and all-cause infant mortality rates (deaths per 1,000) are presented for categorical variables; means and correlations with all-cause infant mortality status are presented for continuous variables. Weighted data with sampling design-adjusted estimates were used for the descriptive statistics, given the oversampling strategies.

[a]NTOP = the Nakao and Treas occupational prestige score.

*$p < .05$; **$p < .01$

Statistical models were built hierarchically, specifying the effects of income (Model 1), income inequality (Model 2), and OP (Model 3)—net of controls for parental education. After all the SES variables were estimated simultaneously (Model 4), blocks of variables were added as sociodemographic variables (Model 5), behavioral variables (Model 6), and pregnancy-related variables (Model 7). Infants' BWT was controlled for in the next model (Model 8), and interactions between OP and BWT were considered (Model 9) directly before interactions between income inequality and BWT to determine whether any noneffects of income inequality in the whole sample could be observed only among those with relatively low BWTs (Model 10). Finally, interactions between income and BWT were specified in the final model (Model 11).

RESULTS

All-Cause Infant Mortality

Income and all-cause infant mortality. Income is significantly related to all-cause infant mortality in a curvilinear relationship that indicates that additional income above a given threshold no longer has salutary effects (see Figure 1). Virtually none of this relationship is accounted for by parental education, but a nominal portion of this relationship is accounted for by sociodemographic characteristics (Model 5, Table 2). Although the exclusion of race/ethnicity actually suppresses the income-mortality relationship, insurance status and parity-age interactions account for a great deal of this relationship. Failure to control for behavioral characteristics (participation in WIC, in particular) suppresses this relationship (Model 6, Table 2), while the health-related variables have a nominal mediating effect, as seen in Model 7 (Table 2).

Some of the income-mortality relationship is mediated by infants' BWT (Model 8, Table 2); in addition, the interaction term between income and BWT (Model 11, Table 2) demonstrates diminishing returns to income among those who were born at LBWTs, although this general pattern appears to hold across BWTs. Income has an effect on the probability of being born at LBWT, but has an independent effect on survival, and this effect is further conditioned by BWT.

Occupational grade, income inequality and all-cause infant mortality. Occupational grade is negatively related to all-cause infant mortality (Model 3, Table 2), although much of the benefit of advantaged occupational status is due to the better income it provides (Model 4, Table 2). However, the remainder of this benefit is mediated by sociodemographic characteristics, particularly race/ethnicity (Model 5, Table 2). Although this relationship is accounted for by the risk factors that are associated with race/ethnicity, the effects are actually significant, and negligibly larger, for infants who were born at moderate and optimal BWTs[11] (Model 9, Table 2). On the other hand, income inequality is unrelated to the probability of dying during infancy (Model 2, Table 2).

Endogenous Infant Mortality

Income and endogenous infant mortality. Income has a significant and curvilinear relationship with endogenous infant mortality as well (see Figure 2[12] and Model 1, Table 3), although a nominal proportion of this relationship is actually due to one's social status, as measured by OP and education (Model 4, Table 3). A large proportion of this relationship is mediated by sociodemographic characteristics (Model 5, Table 3), although the relationship is actually stronger net of measured behavioral characteristics (Model 6, Table 3); health-related variables account for another small proportion of the total income effects (Model 7, Table 3).

Plotting the predicted values for the income–endogenous mortality relationship (see Figure 2) indicates that the largest disparities in risk are those between the poor and the nonpoor, and there is some indication that additional income ceases to be salutary just past the median of the distribution. Regression diagnostics confirmed that the slight upturn in the curve to the far right of the distribution is not driven by influential cases or highly variable data.

11. "Optimal" BWT is the weight that is associated with the smallest risk of infant mortality.

12. All probabilities for mortality presented in the figures were plotted between the 10th and 90th percentiles of the income distribution so as to minimize the effect of extreme values and not to extrapolate beyond the boundaries of the data. Furthermore, predicted probabilities were computed, holding all covariates at their means, from models that estimated (1) the gross (unadjusted) effect of income on mortality, (2) adjustments for other SES variables, (3) adjustments for the former plus sociodemographic characteristics, and (4) adjustments for the entire set of covariates, including infants' BWT.

A moderate portion of the income–endogenous infant mortality relationship is mediated by income-BWT relationships (Model 8, Table 3). Nonetheless, the income–endogenous infant mortality relationship persists net of controls even for infants' BWT, although a significant product term (Model 11, Table 3) demonstrates that while effects on income are present (and largely linear) among those who were born at healthy BWTs, the effects of income on endogenous mortality are larger among those who were born at LBWTs (figure not shown). Although this relationship is also curvilinear and exists across the gradient, the effects are the most pronounced between the poor and the nonpoor, a pattern that holds for most of the income–infant mortality relationships, but is the most prominent for endogenous infant mortality.

Occupational grade, income inequality, and endogenous infant mortality. Occupational grade is negatively related to endogenous infant mortality (Model 3, Table 3), although, similar to all-cause infant mortality, much of these effects work through income (Model 4, Table 3). Again, the remainder of this benefit is due to racial/ethnic differences in the risk of endogenous mortality (Model 5, Table 3). Furthermore, this risk does not emerge at various BWTs, as the nonsignificant product term demonstrates (Model 9, Table 3). However, income inequality is unrelated to the probability of dying from endogenous causes (Model 2, Table 2) and is not moderated by BWT (Model 10, Table 3).

Exogenous Infant Mortality

Income and exogenous infant mortality. Income is related to exogenous infant mortality across a gradient, and although the effects are most pronounced between the poor and the nonpoor, there seems to be a much steeper gradient across the continuum. That is, there are increasing returns to income across virtually the entire income gradient (see Figure 3). Similar to the patterns found for all-cause infant mortality and endogenous mortality, a negligible portion of the estimated income effects is accounted for by other SES measures (Model 4, Table 4), while much of this relationship is accounted for by sociodemographic characteristics, such as age/parity and insurance status (Model 5, Table 4). Controlling for behavioral factors (Model 6, Table 4) accounts for a nominal proportion of this original relationship, while controls for the full set of variables, including infants' BWT, account for more of the income–exogenous mortality relationship.

In addition, infants' BWT moderates the income–exogenous mortality relationship slightly (Model 11, Table 4), although the largest effects appear to be an offset of the intercept that is overwhelmed by the primacy of the effects of BWT (figure not shown). Again, while income works through BWT, it has an independent effect on exogenous infant mortality, net of BWT.

OP, income inequality, and exogenous infant mortality. The salutary effects of OP on exogenous infant mortality (Model 3, Table 4) are accounted for largely by the estimated income effects (Model 4, Table 4). However, a marginally significant (Model 9, Table 4) moderating relationship between OP and BWT is estimated for exogenous mortality. This relationship indicates steeper OP gradients at lower BWTs, with negligible effects at average and optimal BWTs (figure not shown). Finally, there are no independent, or gross, effects of state-level income inequality on the probability of all-cause or cause-specific infant mortality (see Model 2 in each of the tables), and these effects were not suppressed by the potentially moderating effects of BWT (see Model 10 in each of the tables).

DISCUSSION AND CONCLUSIONS

Relationships between income and health have been documented for several years, but only recently has it been argued that these relationships are not peculiar to poverty but, rather, exist along a continuum of material advantage. The gradient was originally conceptualized as a linear function between SES and health, and the competing hypotheses

Table 2. Logistic Regression of All-Cause Infant Mortality on SES: Controlling for Sociodemographics, Health Behaviors, Health-Related Variables, and Mother's/Infant's Birthweight

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
Income^{-5} (1,000s)	–.3730**			–.3777**	–.2215**	–.2443**	–.2216**	–.2127**	–.2411**	–.2385**	–.0199
Income (1,000s)	.0338**			.0363**	.0256**	.0274**	.0247**	.0262**	.0280**	.0274**	.0283**
Mother's Education (yrs.)	–.0247**	–.0460**	–.0387**	–.0248†	–.0270†	–.0074	–.0041	–.0028	–.0169	–.0159	–.0182
Father's Education (yrs.)	–.0164	–.0320**	–.0213†	–.0141	–.0243*	–.0131	–.0170	–.0115	–.0142	–.0158	–.0115
Occupational Grade (logged)			–.3045**	–.1604†	–.0757	–.1133	–.1117	–.1031	.4467†	–.1245	–.0959
Income Inequality (logged)		–.6274		–.8657	–1.3994	–1.7504	–1.7666	–1.5619	–1.5308	–2.6536	–1.4676
Median State Household Income		–.0001		–.0001	–.0001	–.0001	–.0001	–.0001	–.0001	–.0001	–.0001
Race/Ethnicity (Non-Hispanic white)											
Non-Hispanic black					.5443**	.4420**	.4204*	–.0680	–.0228	–.0284	–.0163
Hispanic					–.1906	–.1879	–.1642	–.2597†	–.2146	–.2122	–.2196
Parity (1st birth)											
Low					–.1019	–.2357**	–.2322**	.1135†	.1211†	.1169†	.1285†
High					.1990*	.0192	.0316	.3755**	.4137**	.4146**	.4136**
Marital Status (married)											
Not married					–.0270	–.0608	–.0650	–.2309*	–.1601	–.1652	–.1462
Payer (insurance)											
Medicaid					.1567*	.2374**	.2261**	.1152			
Self-Pay					.1084	.1096	.1174	.1313			
WIC Participation (no)											
Short term						–.3728**	–.4009**	–.0110			
Long term						–.3724**	–.4054**	.0607			
Weight Gain (normal)											
Low						.9075**	.9187**	.0821			
High						–.2548**	–.2819**	.0906			
Prenatal Care (adequate)											
Inadequate						.4409**	.4366**	.3854**			
Intermediate						–.0394	–.0398	.0673			

Adequate plus						.5845**	.5231**	.0756			
Missing/No care						1.343**	1.3172**	.5446**			
Smoke While Pregnant (no)											
Yes						.2754**	.2452**	.0657			
Alcohol Use (no)											
Yes						.0769	.1128	–.1486			
Vitamin Use (yes)											
No						.0394	.0765	–.0256			
Exercise (yes)											
No						.3230**	.3231**	.1013†			
Prevent Delivery (no)											
Yes							.5457**	–.0067			
Previous Loss (no)											
Yes							.1055	–.1541*			
Body Mass Index (medium)											
Low							.1033†	–.0722			
High							–.0369	.0729			
Mother's Low Birth Weight							.0264	–.1451†			
Mother's Birth Weight Missing							.1024†	.0378			
Infant's Birth Weight								–.0034**	–.0026**	–.0053	–.0031**
Infant's Birth Weight^2								.0001**	.0001**	.0001**	.0001**
Occupational Grade × Birth Weight									–.0002**		
Income Inequality × Birth Weight										.0005	
Income^5 × Birth Weight											–.0001**
Constant	–3.5414	–0.9779	–3.0212	0.6408	1.8811	2.5949	2.3557	8.5155	6.9751	13.2899	7.8952
Log-Likelihood	–7,750.0903	–7,742.3391	–7,757.8228	–7,734.5419	–7,713.0253	–7,438.5811	–7,419.9782	–6,760.8541	–6,813.9275	–6,816.5335	–6,801.5219
Sample Size	12,814	12,814	12,814	12,814	12,814	12,814	12,814	12,814	12,814	12,814	12,814

Notes: Survived is the reference category. Estimates of model log-likelihoods came from standard logit estimates; standard errors were obtained from a survey-specific procedure.

†$p < .10$; *$p < .05$; **$p < .01$

Table 3. Logistic Regression of Endogenous Infant Mortality on SES: Controlling for Sociodemographics, Health Behaviors, Health-Related Variables, and Mother's/Infant's Birth Weight

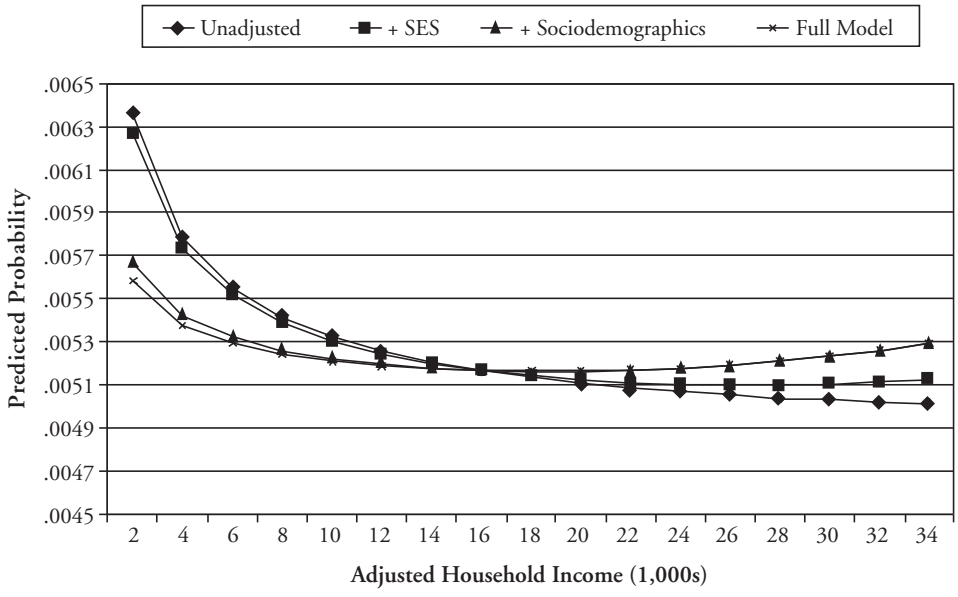
Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
Income$^{-.5}$(1,000s)	.4581**			.4336**	.2138*	.2629**	.2279*	.1799†	.1719†	.1724†	–.1747
Income^{3} (1,000s)	.0001			.0001	.0001	.0001	.0001	.0001	.0001	.0001	.0001
Mother's Education (yrs.)	–.0121	–.0224	–.0167	–.0098	–.0131	.0037	.0078	.0099	.0029	.0035	.0029
Father's Education (yrs.)	–.0158	–.0229†	–.0137	–.0103	–.0140	–.0036	–.0080	.0036	.0032	.0021	.0028
Occupational Grade (logged)			–.2607*	–.1988†	–.0728	–.1394	–.1395	–.1174	.1292	–.1092	–.1053
Income Inequality (logged)		.1370		–.0197	–.7579	–1.3479	–1.2891	–.8886	–.7839	–2.7801	–.7620
Median State Household Income		–.0001		–.0001	–.0001	–.0001	–.0001	–.0001	–.0001	–.0001	–.0001
Race/Ethnicity (Non-Hispanic white)											
Non-Hispanic black					.6400**	.4617**	.4240*	–.18881	–.0978	–.0988	–.1044
Hispanic					–.0645	–.1390	–.1092	–.2245	–.0993	–.0981	–.1023
Parity (1st birth)											
Low					–.2239**	–.3824**	–.3865**	.0583	.0451	.0436	.0484
High					.0179	–.1795†	–.1716†	.2395*	.2423**	.2452	.2399**
Marital Status (married)											
Not married					–.0682	–.0704	–.0774	–.2998**	–.2871**	–.2899**	–.2883**
Payer (insurance)											
Medicaid					.0543	.2274**	.2134*	.0622			
Self-pay					.0717	.0971	.1105	.1373			
WIC Participation (no)											
Short term						–.4720**	–.5158**	–.0277			
Long term						–.6168**	–.6667**	–.0911			
Weight Gain (normal)											
Low						1.1415**	1.1524**	.1059			
High						–.3105**	–.3428**	.1308			
Prenatal Care (adequate)											
Inadequate						.3774**	.3771**	.3158**			
Intermediate						–.0487	–.0519	.1111			

Adequate plus						.7538**	.6620**	.0923			
Missing/No care						1.5521**	1.5198**	.5382**			
Smoke While Pregnant (no)											
Yes						.0841	.0474	–.1764*			
Alcohol Use (no)											
Yes						.1068	.1398	–.2143			
Vitamin Use (yes)											
No						.0414	.0912	–.0136			
Exercise (yes)											
No						.0434**	.4235**	.1336*			
Prevent Delivery (no)											
Yes							.7205**	.0129			
Previous Loss (no)											
Yes							.1991**	–.1384†			
Body Mass Index (medium)											
Low							.0868	–.1065			
High							–.0597	.0486			
Mother's Low Birth Weight							.0582	–.1341			
Mother's Birth Weight Missing							.1512*	.0694			
Infant's Birth Weight								–.0036**	–.0033**	–.0074*	–.0038**
Infant's Birth Weight^2								.0001**	.0001**	.0001**	.0001**
Occupational Grade × Birth Weight									–.0001		
Income Inequality × Birth Weight										.0009	
Income$^{.5}$ × Birth Weight											.0002*
Constant	–5.0279	–4.8062	–3.8728	–4.0846	–1.6999	–.0889	–.6302	5.4517	4.5185	12.9427	5.432251
Log-Likelihood	–6,366.3391	–6,361.6822	–6,367.9293	–6,359.9666	–6,347.7201	–5,988.0218	–5,949.2895	–5,027.2267	–5,064.4483	–5,063.4156	–5,063.9737
Sample Size	11,751	11,751	11,751	11,751	11,751	11,751	11,751	11,751	11,751	11,751	11,751

Notes: Survived is the reference category. Estimates of model log-likelihoods came from standard logit estimates; standard errors were obtained from a survey-specific procedure.

†$p < .10$; *$p < .05$; **$p < .01$

Figure 2. Predicted Probabilities of Endogenous Infant Mortality Across Levels of Income



emerged with this understanding. Under this assumption, the notion of the gradient becomes problematic because it is difficult to hypothesize why a wealthy person would still be less healthy than a very wealthy person. However, recent empirical analyses have demonstrated that there are diminishing health returns to increasing SES; these analyses have confirmed that although the relationship between income and mortality is curvilinear and exists along a gradient, there are generally no or few added benefits past a particular point in the distribution. Furthermore, this relationship cannot be wholly accounted for by controlling for (albeit limited) sociodemographic, behavioral, and pregnancy/health-related variables.

Similar to adult mortality, infant mortality (all-cause, endogenous, and exogenous) exists along a graded continuum, and elevated risks are not peculiar to poverty. Although the income curves for this analysis are entirely data driven, the results are not implausible when one considers the effects of income on health. In Figure 1, it can be seen that for all-cause and endogenous infant mortality, the largest income effects are observed to the left of the distribution (poorer respondents), and this effect flattens out such that there are no longer any advantages past a given level of income—although this level is to the far right of the distribution.

The use of fractional polynomials also aids in this process because it can minimize the "waviness" of certain specifications. Given that endogenous infant mortality is reliant on pregnancy processes and can be caused by material deprivations like poor nutrition, these results seem consistent with our understanding of the etiological processes involved. On the other hand, although the effects are also more pronounced for those who are living in poverty, the returns to income for exogenous infant mortality persist across the whole of the distribution in the unadjusted models (see Figure 3). Given that exogenous infant

mortality is more reliant on social conditions than is endogenous infant mortality, it is likely that both material conditions and psychosocial effects play a role, given that those at the far right of the distribution hardly suffer from any material deprivation, although *relative deprivation* may play a role. The (marginally significant) moderating effect that OP plays on the relationship between BWT and exogenous infant mortality further reinforces this possibility. Nonetheless, although the empirical shapes of these gradients may provide clues to the culpability of various social and material factors for the risk of infant mortality, a more direct test of the competing hypotheses was undertaken, and these results are discussed next.

The results demonstrate that income inequality is unrelated to infant mortality. Although occupational grade is marginally related to all-cause and exogenous infant mortality (as moderated by BWT effects), it does not "explain away" the observed income effects on postneonatal mortality. It may be that prestige per se is not an adequate proxy for occupational grade, given that it is largely determined by the educational achievement, and income (to a lesser extent) of incumbents to the occupations (Hauser and Warren 1997). Nevertheless, OP is highly correlated with factors that determine social participation and the ability to control one's destiny (Garbin and Bates 1961),[13] and occupations such as professor are consistently ranked higher than many of those with much larger salaries.

Thus, the material-conditions hypothesis may be the most viable explanation for the relationship between SES and infant mortality. This result is supported by the observation that the most severe disparities occur between those who are in poverty and those who are not (i.e., the curve is still the steepest at the lowest levels of income) and that, in general, there is a threshold at which additional income is no longer salutary.

Nonetheless, it is worth considering that this threshold occurs past the median of the distribution for endogenous infant mortality and to the far right of the distribution for exogenous infant mortality, where poverty is no longer an issue. Although occupational grade and income inequality do not provide suitable explanations for this gradient, there are clearly other factors involved, since individuals in the upper-income percentiles, for example, are not likely to be materially deprived. Therefore, owing to the existence of occupational-grade effects for exogenous infant mortality—largely among those who were born at LBWTs—and to the fact that the threshold for the gradient occurs far into the distribution, there is still room for hypothesizing about the psychosocial conditions that may contribute to this observed effect. As it stands, however, income inequality is generally not one of the viable candidates (see, e.g., Ellison 2002; Wagstaff and van Doorslaer 2000). In short, although material conditions during pregnancy appear primary to the risk of infant mortality, these effects do not preclude the additional role of status hierarchies and other psychosocial explanations for disparities in birth outcomes, particularly effects that may have accumulated over the life course and may not be observable in most survey-based data.

Traditional risk factors for poor birth outcomes are also related to SES and mediate the effect of income on infant mortality. The largest mediators of the income-infant mortality relationship are parity and insurance status. While first births and high-parity births represent risk factors for endogenous infant mortality (relative to low-parity births), both

13. Selected subjective correlates of OP (and Pearson correlations) were reported as follows: interesting/challenging work, .90; intelligence required, .90; scarcity of personnel, .90; originality and initiative, .87; influencing others, .86; desirable to associate with, .84; training required, .84; education required, .83; supervisory responsibility, .79; security, .79; income, .78; honorable/morally good work, .75; advancement opportunities, .71; service to humanity, .59; being one's own boss, .57; clean work, .51; dealing with people, not things, .49; flexible hours, .44; safe work, .35; free time on the job, .15. Although Garbina and Bates's study is relatively old, it is worth noting that OP ratings have been one of the most stable social ratings over the past five decades, and the rankings have changed *little* from decade to decade (Nakao and Treas 1994).

Table 4. Logistic Regression of Exogenous Infant Mortality on SES: Controlling for Sociodemographics, Health Behaviors, Health-Related Variables, and Mother's/Infant's Birth Weight

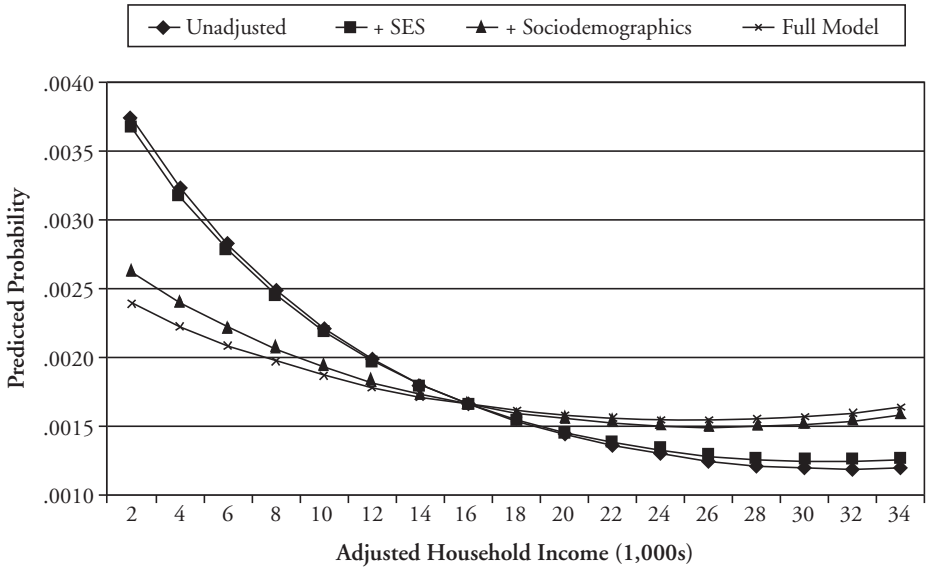
Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
Income (1,000s)	–.0808**			–.0803**	–.0497**	–.0454**	–.0449**	–.0408**	–.0567**	–.0570**	–.0311*
Income^2 (1,000s)	.0012**			.0013**	.0009**	.0009**	.0009**	.0008**	.0010**	.0010**	.0010**
Mother's Education (yrs.)	–.0603**	–.1035**	–.1087**	–.0658**	–.0621**	–.0419†	–.0401†	–.0365	–.0588**	–.0590**	–.0591**
Father's Education (yrs.)	–.0228	–.0539**	–.0384*	–.0257	–.0484**	–.0381*	–.0388*	–.0379*	–.0464**	–.0478**	–.0456**
Occupational Grade (logged)			–.4586**	–.1147	–.0701	–.0334	–.0399	–.0383	.7236	–.1024	–.0856
Income Inequality (logged)		–2.7093		–3.0615	–3.1276	–2.8969	–2.9388	–2.9288	–3.0503	–.6083	–3.0204
Median State Household Income		–.0001		–.0001	–.0001	–.0001	–.0001	–.0001	–.0001	–.0001	–.0001
Race/Ethnicity (non-Hispanic white)											
Non-Hispanic black					.3051	.4175†	.4276†	.2439	.1326	.1264	.1359
Hispanic					–.5471*	–.3223	–.3180	–.3558	–.5266*	–.5258*	–.5259*
Parity (1st birth)											
Low					.2504*	.1616	.1847	.2571*	.3153**	.3114**	.3165**
High					.6239**	.4599**	.4909**	.5768**	.7043**	.6989**	.7063**
Marital Status (married)											
Not married					.0732	–.0314	–.0371	–.0736	.0973	.0966	.1026
Payer (insurance)											
Medicaid					.3682**	.2768*	.2752*	.2294†			
Self-pay					.1909	.1334	.1285	.1303			
WIC Participation (no)											
Short term						–.1386	–.1350	–.0378			
Long term						.0644	.0678	.1758			
Weight Gain (normal)											
Low						.2273*	.2412*	.0226			
High						–.1520	–.1494	–.0067			
Prenatal Care (adequate)											
Inadequate						.5114**	.5077**	.4619**			
Intermediate						–.0414	–.0406	–.0293			

Adequate plus						.1186	.1289	.0091			
Missing/No care						.7452**	.7349**	.5288**			
Smoke While Pregnant (no)											
Yes						.6648**	.6638**	.5346**			
Alcohol Use (no)											
Yes						.0826	.0895	.0195			
Vitamin Use (yes)											
No						.0164	.0181	–.0323			
Exercise (yes)											
No						.0711	.0810	.0373			
Prevent Delivery (no)											
Yes							.0391	–.1012			
Previous Loss (no)											
Yes							–.1437	–.1885†			
Body Mass Index (medium)											
Low							.1519†	.0582			
High							.0032	.0810			
Mother's Low Birth Weight							–.0699	–.1416			
Mother's Birth Weight Missing							–.0213	–.0350			
Infant's Birth Weight								–.0016**	–.0005	.0016	–.0014**
Infant's Birth Weight^2								.0001**	.0001**	.0001**	.0001**
Occupational Grade × Birth Weight									–.0003†		
Income Inequality × Birth Weight										–.0008	
Income^5 × Birth Weight											–.0001*
Constant	–4.3995	7.1668	–2.9302	8.4714	7.8368	6.1228	6.1981	9.5709	8.1839	1.8909	10.67563
Log-Likelihood	–3,313.5455	–3,319.8431	–3,338.3156	–3,294.9431	3,248.2407	3,201.6479	3,196.7961	3,192.3185	–3,249.861	–3,248.9844	–3,249.4349
Sample Size	10,082	10,082	10,082	10,082	10,082	10,082	10,082	10,082	10,082	10,082	10,082

Notes: Survived is the reference category. Estimates of model log-likelihoods came from standard logit estimates; standard errors were obtained from a survey-specific procedure.

†$p < .10$; *$p < .05$; **$p < .01$

Figure 3. Predicted Probabilities of Exogenous Infant Mortality Across Levels of Income



low and high parity were associated with the increased risk of exogenous infant mortality (relative to first births). These variables capture both the birth number and age of mothers, variables that are highly correlated with SES and maternal education. On the other hand, having Medicaid as the primary source of medical insurance (and the primary payer for childbirth) was a significant risk factor for both endogenous and exogenous infant mortality (as well as all-cause infant mortality) relative to having private insurance. As a mediator, insurance status is highly related to family income, but Medicaid may also indicate a level of negative selectivity, in that mothers with greater propensities for health problems or negative health histories may be enrolled. That is, the negative effects of Medicaid may not be associated with poor medical care that leads to increased risk of death, but, rather, may indicate both the SES and health histories of mothers.

These results are interesting in that they provide a way to minimize reverse-causation effects, given that the mortality risks for infants are entirely subject to the conditions of their mothers' health and social environment. Therefore, the mortality risks for infants cannot have been said to cause the SES of the parents. However, a recent study (Conley and Bennett 2000) demonstrated that some of the socioeconomic effect may actually have been due to the legacy of poor birth outcomes transferred to the mothers of these children. These analyses attempted to minimize this influence by controlling for maternal BWT, and it was demonstrated that the gradient persisted net of maternal BWT.

It is also expected that income effects may be biased downward because data were not collected on wealth and debt. That is, cases that may be heavily under- or overpredicted may be so because of suppressed effects of worse health among those with more debt and moderate incomes and/or better health among those with lower incomes but a greater reliance on wealth for living circumstances.

Future research should test these results for other birth outcomes (e.g., preterm birth and intrauterine-growth retardation) and other infant-child health outcomes, while paying attention to unobserved heterogeneity. This unobserved heterogeneity may be due to unmeasured factors at the family level and/or genetic propensities for health that vary across individuals of various socioeconomic positions, yet may also be due to structural factors (e.g., race and socioeconomic segregation) that individuals with the same characteristics have in common. In particular, specific attention should be paid to the role that material conditions and governmental interventions may play in minimizing compromised birth outcomes that may have resulted from poverty. Future research should also continue to attempt to explain the peculiarity of the gradient with the realization that advantaged health benefits do not accrue across the whole of the SES distribution.

Finally, research on the mediating processes that translate income into lower risks of mortality (e.g., bacterial vaginosis) is necessary to determine potential interventions that can be undertaken to lower rates of infant mortality. Concomitantly, studies that attempt to make causal connections between income and mortality are crucial to ensure that interventions are viable.

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