Demography (2013) 50:615–635
DOI 10.1007/s13524-012-0148-2

Maternal Education, Birth Weight, and Infant Mortality in the United States

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Published online: 17 October 2012

Abstract This research determines whether the observed decline in infant mortality with socioeconomic level, operationalized as maternal education (dichotomized as college or more, versus high school or less), is due to its "indirect" effect (operating through birth weight) and/or to its "direct" effect (independent of birth weight). The data used are the 2001 U.S. national African American, Mexican American, and European American birth cohorts by sex. The analysis explores the birth outcomes of infants undergoing normal and compromised fetal development separately by using covariate density defined mixture of logistic regressions (CDDmlr). Among normal births, mean birth weight increases significantly (by 27–108 g) with higher maternal education. Mortality declines significantly (by a factor of 0.40–0.96) through the direct effect of education. The indirect effect of education among normal births is small but significant in three cohorts. Furthermore, the indirect effect of maternal education tends to increase mortality despite improved birth weight. Among compromised births, education has small and inconsistent effects on birth weight and infant mortality. Overall, our results are consistent with the view that the decrease in infant death by socioeconomic level is not mediated by improved birth weight. Interventions targeting birth weight may not result in lower infant mortality.

Keywords Maternal education · Birth weight · Infant mortality · CDDmlr

Introduction

The strong correlation between birth weight and infant mortality is empirically so well documented (Buehler et al. 1987; Frisbie et al. 1996; Institute of Medicine 1985;

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McCormick 1985; Solis et al. 2000) that birth weight is often used as proxy for infant mortality (Kallan 1993; Van Den Oord and Rowe 2000), and current U.S. policy to reduce infant mortality is to improve birth weight outcomes (U.S. Department of Human Heath Services 2000). Similarly, social research has identified a consistent correlation between socioeconomic level and infant mortality, which many studies conclude is mediated at least in part by birth weight (Conley et al. 2003; Frisbie et al. 1996; Hummer et al. 1999; Kramer 1987). In general, these interpretations are situated within Mosely and Chen's "proximate determinants" model of infant and childhood mortality (Frisbie et al. 1996; Mosely and Chen 1984). Briefly, the proximate determinants model proposes that social variables (e.g., socioeconomic level, race/ethnicity, and nativity) influence mortality through a hierarchy of proximate determinants. For example, socioeconomic level might influence maternal nutrition, which might influence birth weight and hence affect infant mortality—in which case, nutrition and birth weight would be proximate determinants of infant mortality, at least compared with socioeconomic level. The goal of the proximate determinants model is to map the causal pathways through which such social variables and their more proximate determinants influence infant mortality (Frisbie et al. 1996; Mosely and Chen 1984).

On the other hand, the causal role of birth weight on infant mortality has been challenged by a number of theoreticians, including the originators of the proximate determinants model (Mosely and Chen 1984; Wilcox and Russell 1990; Wise 2003). The Wilcox-Russell/Hernández-Diaz hypothesis (Hernández-Diaz et al. 2008; Wilcox and Russell 1990) is perhaps the best-developed theory. They contend that, at least among normal births (basically, those found within a Gaussian distribution: i.e., not including those births in the heavy upper and lower tails of the birth weight distribution), birth weight is not on the causal pathway to infant mortality. Hence, the (potential) social effects on birth weight are not passed on to infant mortality (i.e., mediated) through birth weight. The implication based on the proximate determinants model is that socioeconomic factors influence both birth weight and infant mortality—hence, the correlation between birth weight and infant mortality—but that some determinant other than birth weight (e.g., gestational age or other maternal–fetal interactions) mediates the relationship between socioeconomic factors and infant mortality independently of birth weight.

A statistical method of examining the causal role of birth weight, within the context of the proximate determinants model, has been developed (Gage et al. 2004, 2009), based on the Wilcox-Russell definition of "causality" (Wilcox and Russell 1990). The aim of this research is to determine whether maternal education, as a measure of socioeconomic level, affects infant mortality indirectly (though birth weight), directly (independent of birth weight), or both. If no indirect effect through birth weight is observed given a shift in the birth weight distribution, then birth weight cannot be on the causal pathway to infant mortality. If so, then socioeconomic factors must influence infant mortality through other pathways. These analyses are repeated on six populations (i.e., African Americans, European Americans, and Mexican Americans by sex from the 2001 U.S. national linked birth–death files) to establish the consistency, or lack thereof, of socioeconomic effects on birth weight and infant mortality across different populations.

Background

Wilcox and Russell (1990) argued that birth weight is not on the causal pathway to infant mortality, because the birth weight distribution and the birth weight–specific mortality curve shift horizontally in the same direction by a similar amount in response to a stressor (Fig. 1, panel a). Consequently, there is no net change in the infant mortality despite a shift in the birth weight distribution. For example, fetal development at high altitude is associated with lower birth weight, but it results in little or no change in infant mortality. On the other hand, if the shift in the birth weight–specific mortality curve does not match the shift in the birth weight distribution, then birth weight could be on the causal pathway to infant mortality (Fig. 1, panel b). This will be referred to here as an indirect effect of the stressor through birth weight on infant mortality. In addition, Wilcox and Russell (1990) posited that there also may be a consistent increase (or decrease) in infant mortality at all birth weights (independent of birth weight) because of a stressor (e.g., smoking during pregnancy) (Fig. 1, panel c). Although strictly speaking, socioeconomic status must operate through some more-proximate determinants, for simplicity, the birth weight–independent effect will be referred to as a direct effect of the stressor (direct in the sense that it does not involve birth weight). The Wilcox-Russell hypothesis (Wilcox and Russell 1990) does not address whether compromised births (basically those births in the heavy upper and lower tails of the birth weight distribution) behave in the same manner as normal births. Nevertheless, to fully examine the Wilcox-Russell hypothesis requires a method that distinguishes between normal and compromised births. By including an exogenous covariate, covariate density defined mixture of logistic regressions (CDDmlr) can estimate the significance of indirect as well as direct effects separately for normal and compromised births.

Conceptually, the basic CDDmlr (i.e., without any exogenous covariate) fits a multicomponent Gaussian finite mixture model to the birth weight distribution, which divides the population into several (two in this application) latent subpopulations, and simultaneously estimates a separate logistic regression on infant mortality by birth weight for each latent subpopulation (Gage et al. 2004). This procedure is useful when the latent subpopulations account for otherwise unobserved heterogeneity with respect to mortality. For the purposes of identification, the subpopulation accounting for the majority of individuals is labeled the primary (p) subpopulation, and the remaining minority component is labeled the secondary (s) subpopulation. The logistic regressions on infant mortality are parameterized as second-degree polynomials of birth weight to account for the reverse J–shaped relationship of birth weight and infant mortality. Previous applications (Gage 2002; Gage et al. 2004) indicated that (1) the primary subpopulation represents births with birth weights generally within the normal range (Fig. 2, panel a) and hence is interpreted as representing births undergoing normal fetal development; (2) the secondary subpopulation accounts for most low birth weight and macrosomic births (Fig. 2, panel a) and is consequently interpreted as accounting for births undergoing compromised fetal development; and (3) the two components identified by the birth weight density submodel are heterogeneous with respect to infant mortality with the compromised subpopulation consistently displaying lower birth weight–specific

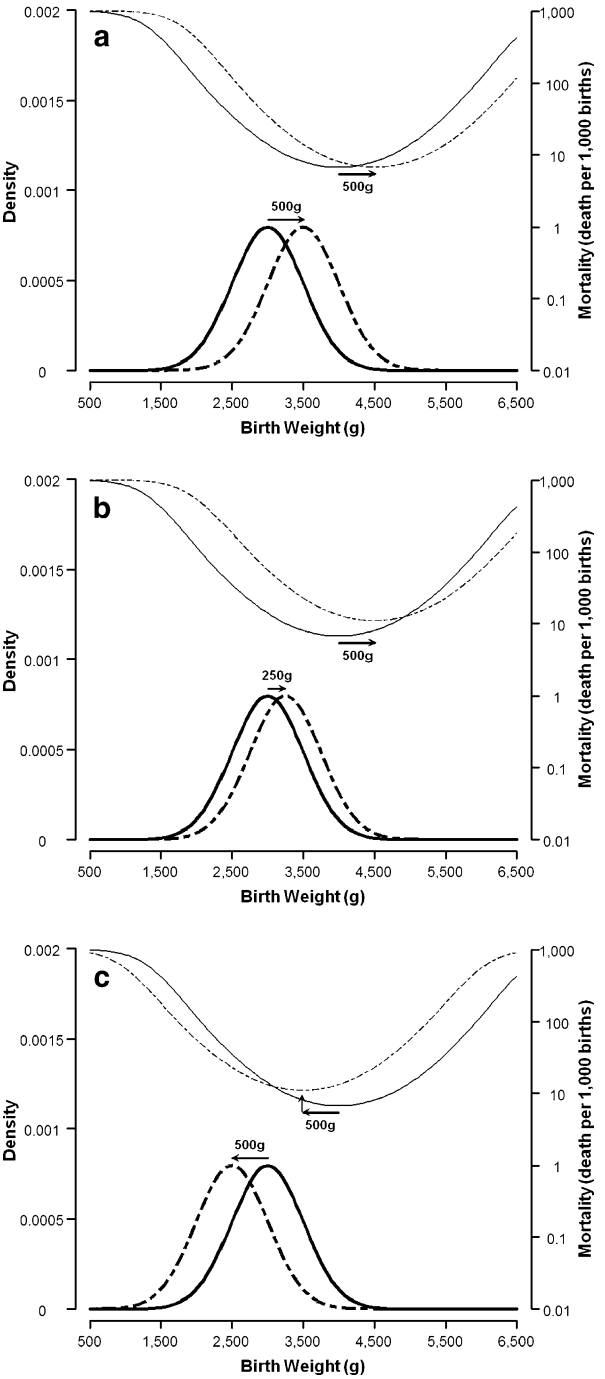

Fig. 1 Graphical illustration of the Wilcox-Russell (Wilcox and Russell 1990) definition of causality. Panel a represents a shift in the birth weight distribution that is accompanied by an identical shift in the birth weight–specific mortality curve owing to a stressor, so that there is no change in the overall mortality and birth weight is not causal. Panel b represents a shift in birth weight that is not accompanied by an identical shift in the mortality curve, so that the stressor has an indirect effect on mortality and birth weight could potentially be on the causal pathway to infant mortality. Panel c represents a birth weight–independent change of the mortality curve (i.e., a vertical shift of the whole curve) owing to a stressor when birth weight is not causal, so that a direct effect of the stressor occurs

mortality but higher overall mortality because of the less favorable distribution of birth weight (Fig. 2, panel b).

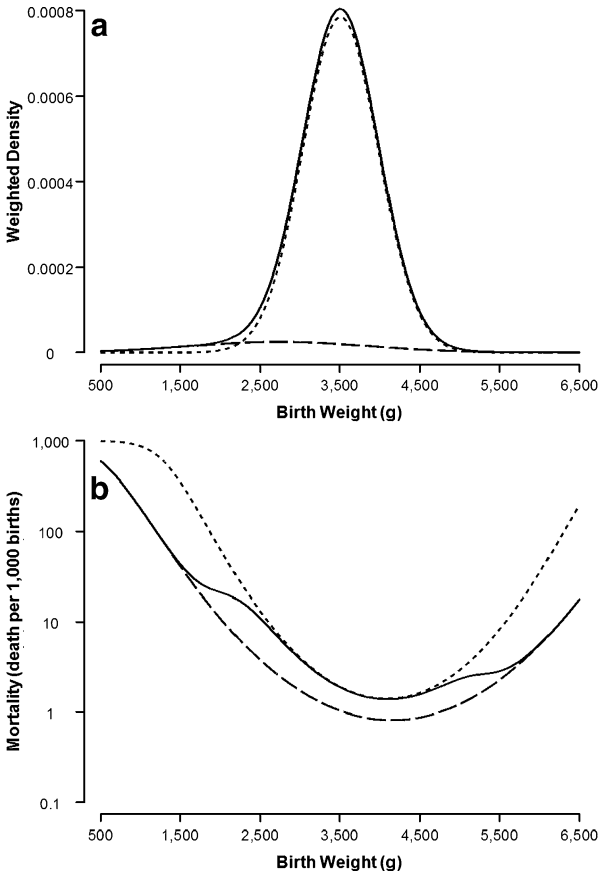

Fig. 2 Graphical illustration of covariate density defined mixture of logistic regressions (CDDmlr) without any exogenous covariate as applied to birth weight and infant mortality. Results presented are for European American males in 2001. The solid lines represent the total population, the dotted lines represent the primary subpopulation, and the dashed lines represent the secondary subpopulation. Panel a demonstrates the total distribution of birth weight as a weighted sum of two Gaussian densities (i.e., the primary and the secondary subpopulations). The secondary subpopulation is considered to consist of compromised births, because it accounts for most low birth weight and macrosomic infants. The primary subpopulation consists of the remaining births who are considered to be undergoing normal fetal development. Panel b demonstrates the total birth weight–specific mortality curve as a weighted sum of the primary and the secondary birth weight–specific mortality curves. Results for other five populations of interest are similar

We expand this basic model by adding exogenous covariates to the birth weight density submodel and the conditional mortality submodel (i.e., the mixture of logistic regressions). In the case presented here, the covariate is maternal education: high education (i.e., college or more, $z = 1$) versus low education (i.e., high school or less, $z = 0$) as a proxy for socioeconomic level. In the birth weight density submodel, all parameters (i.e., the mixing proportion, means, and variances) are each defined as a linear function of education. In addition, the logistic regression for each subpopulation identified is stratified on education by adding education as an indicator variable on all terms of the second-degree polynomials. Finally, we define each logistic regression of mortality as a second-degree polynomial of the standardized birth

weight based on the Gaussian subpopulation that it represents. Thus, the regression for each latent component is similar to the homogeneous model proposed by Solis et al. (2000). The result is a model that can test the Wilcox-Russell hypothesis (Wilcox and Russell 1990) and its extensions (Hernández-Diaz et al. 2008) of direct and indirect effects (Fig. 1) separately for normal and compromised births. Parameterization is based on the standard (nonlinear) quadratic specification $\text{logit}\left(P\left(y=1\,|\,x\right)\right)=A+C\left(x-B\right)^2$, where A and B are Cartesian coordinates of the vertex where the minimal mortality occurs. A significant change in the value of A because of education can be interpreted as a direct effect of maternal education, which is constant at all birth weights. Because of the use of standardized birth weight, an insignificant change in the values of B and/or C indicates that the birth weight–specific mortality curve shifts in concert with the change of birth weight density (i.e., both mean and variance). Thus, no indirect effect of education is possible as argued by the Wilcox-Russell hypothesis (Wilcox and Russell 1990). On the other hand, any significant change in the value of B because of education accounts for a horizontal shift in the birth weight–specific mortality curve relative to mean birth weight, which results in a potential indirect effect. Similarly, any significant change in C because of education accounts for changes in shape of the birth weight–specific infant mortality curve relative to the standardized birth weight distribution. This is not considered in the Wilcox-Russell theory, but is by Hernández-Diaz et al. (2008), and represents a second way that birth weight–specific infant mortality could be uncoupled from the birth weight density. Again, an insignificant change in C indicates no possible indirect effect, while a significant change in C indicates a significant potential indirect effect. Here, we examine shift and shape effects together as one potential causal effect, since they both represent an uncoupling of the birth weight density and birth weight–specific mortality. A formal definition of this model is provided in the methods.

A limitation of Wilcox's original theory and CDDmlr as defined here is that they do not completely account for all potential influence of birth weight on infant mortality. In particular, the original Wilcox and Russell theory (1990) assumes that the quadratic coefficient of the reverse J–shaped birth weight–specific mortality curve is constant. CDDmlr, following Hernández-Diaz et al. (2008), relaxes this assumption a bit by allowing the shape to change (i.e., a change of C's value). Nevertheless, it remains possible that birth weight is responsible for the reverse J shape of the infant mortality curve. Recently, Basso et al. (Basso and Wilcox 2009; Basso et al. 2006) have provided an extension of Wilcox's original theory, which attributes the reverse J shape to confounding. CDDmlr could be used to explore this possibility as well. However, this is beyond the scope of the present article. Here, we use CDDmlr and a statistical decision theory approach (Geneletti 2007) to decompose direct from indirect effects of maternal education on infant mortality and determine whether differences in the birth weight density associated with socioeconomic level result in changes in infant mortality. If differences in the birth weight distribution do not result in changes in infant mortality, then birth weight cannot be on the causal pathway to infant mortality. If, on the other hand, differences in infant mortality do result, then birth weight may or may not be on the causal pathway since a shift or

shape change could be attributable to confounding and not a causal effect of birth weight.

Data and Methods

Statistical Model: CDDmlr

Formally, the CDDmlr model is defined as the joint conditional probability of one-year death indicator (y) and birth weight (x) given the exogenous dichotomous covariate (z) (Eq. (1)). In particular, it is a product of the conditional birth weight density submodel $f_1\left(x \mid z;\theta\right)$ and the conditional mortality submodel $f_2\left(y \mid x,z;\beta^*,\theta\right)$:

$$f\left(y,x \mid z;\beta^*,\theta\right) = f_2\left(y \mid x,z;\beta^*,\theta\right)\cdot f_1\left(x \mid z;\theta\right), \tag{1}$$

where θ represents the collection of parameters of the birth weight distribution, and β^* the parameters of the subpopulation specific logistic regressions of mortality by standardized birth weight.

In this study, each birth cohort is assumed to consist of two truncated Gaussian subpopulations. So for $i = s$ and p, $f_1\left(x \mid z;\theta\right)$ is given as

$$\begin{aligned} f_1\left(x \mid z;\theta = \left(\pi_s\left(z\right),\mu_s\left(z\right),\sigma_s\left(z\right),\mu_p\left(z\right),\sigma_p\left(z\right)\right)\right) \\ = \pi_s\left(z\right)\cdot\tilde{N}\left(x;\mu_s\left(z\right),\sigma_s\left(z\right)\right)+\left[1-\pi_s\left(z\right)\right]\cdot\tilde{N}\left(x;\mu_p\left(z\right),\sigma_p\left(z\right)\right). \end{aligned} \tag{2}$$

$$\operatorname{logit}\left(\pi_s\left(z\right)\right) = \eta_s\left(z\right) = \eta_{s,0} + z\cdot\eta_{s,1}. \tag{3}$$

$$\mu_i\left(z\right) = \gamma_{i,0} + z\cdot\gamma_{i,1}. \tag{4}$$

$$\sigma_i\left(z\right) = \lambda_{i,0} + z\cdot\lambda_{i,1} \tag{5}$$

$\pi_s\left(z\right)$ is the proportion of births belonging to subpopulation i given z. $\tilde{N}\left(x;\mu_i\left(z\right),\sigma_i\left(z\right)\right)$ represents a Gaussian density, truncated at 500 g, with mean $\mu_i\left(z\right)$ and standard deviation $\sigma_i\left(z\right)$ given z. The conditional mortality submodel $f_2\left(y \mid x,z;\beta^*,\theta\right)$ is given by

$$f_2\left(y \mid x,z;\beta^* = \left(\beta_s^*,\beta_p^*\right),\theta\right) = q_s\left(x \mid z,\theta\right)\cdot P_s\left(y \mid x_s^*\left(z\right),z;\beta_s^*\right)+\left[1-q_s\left(x \mid z,\theta\right)\right]\cdot P_p\left(y \mid x_p^*\left(z\right),z;\beta_p^*\right). \tag{6}$$

$q_s\left(x \mid z,\theta\right)$ is the conditional probability of an infant with birth weight x belonging to subpopulation s given z. $f_1\left(x \mid z;\theta\right)$ determines that

$$q_s\left(x \mid z;\theta\right) = \frac{\pi_s\left(z\right)\cdot \tilde{N}\left(x \mid z;\mu_s\left(z\right),\sigma_s\left(z\right)\right)}{f_1\left(x \mid z;\theta\right)}. \tag{7}$$

For i = s and p, $P_i\left(y \mid x_i^*\left(z\right),z;\beta_i^*\right)$ is given by

$$P_i\left(y \mid x_i^*\left(z\right),z;\beta_i^* = \left(A_i^*\left(z\right),B_i^*\left(z\right),C_i^*\left(z\right)\right)\right) = \frac{e^{y\cdot\left\{A_i^*(z)+C_i^*(z)\left[x_i^*(z)-B_i^*(z)\right]^2\right\}}}{1+e^{A_i^*(z)+C_i^*(z)\left[x_i^*(z)-B_i^*(z)\right]^2}}. \tag{8}$$

$$x_i^*\left(z\right) = \frac{x-\mu_i\left(z\right)}{\sigma_i\left(z\right)}. \tag{9}$$

$$A_i^*\left(z\right) = A_{i,0}^* + z\cdot A_{i,1}^*. \tag{10}$$

$$B_i^*\left(z\right) = B_{i,0}^* + z\cdot B_{i,1}^*. \tag{11}$$

$$C_i^*\left(z\right) = C_{i,0}^* + z\cdot C_{i,1}^*. \tag{12}$$

Model Fitting

The model was fitted to individual data by the method of maximum likelihood (*ms* routine in the SPLUS statistical library). To facilitate the fitting process, the linear expansions of Eq. (8) were used, as opposed to the nonlinear quadratic mortality functions. The parameters were transformed to their respective nonlinear forms after fitting. In all, there are 22 parameters for the CDDmlr model given the dichotomous covariate education (z). Bias-adjusted (point-wise) 95 % confidence intervals for the parameters and estimates were calculated from 200 bootstrap samples. Our model shows no evidence of lack of fit based on the Hosmer-Lemeshow statistic at the α = .05 level.

Decomposition of the Maternal Education Effect on Infant Mortality

Decomposition of the maternal education effect was carried out in two steps. First, using the standard Kitagawa decomposition method (Gupta 1978), the overall infant

mortality disparity between births born to high- and low-educated mothers was decomposed into effects attributable to (1) the differences in the mixing proportion, (2) the difference in the death rates of the primary subpopulations, and (3) the difference in the death rates of the secondary subpopulations.

Then the maternal education effect on the infant death rate in each subpopulation was further decomposed into two multiplicative components by factoring the respective subpopulation specific relative risk (RR_i, $i = s$, and p) of death for mothers receiving high education as compared with that for mothers receiving low education into a direct factor ($F_{i,1}$) and an indirect factor ($F_{i,2}$) of maternal education:

$$RR_i = \frac{\overline{P_i\left(y=1 \mid z=1\ \mu_i(z),\sigma_i(z),\beta_i^*\right)}}{\overline{P_i\left(y=1 \mid z=0;\mu_i(z),\sigma_i(z),\beta_i^*\right)}} = F_{i,1}\cdot F_{i,2}. \tag{13}$$

$$\overline{P_i\left(y=1 \mid z;\mu_i(z),\sigma_i(z),\beta_i^*\right)} = \frac{\sum\limits_x \left[\tilde{N}\left(x_i \mid z;\mu_i(z),\sigma_i(z)\right)\cdot P_i(y=1 \mid x_i^*(z),z;\beta_i^*)\right]}{\sum\limits_x \tilde{N}\left(x_i \mid z;\mu_i(z),\sigma_i(z)\right)}. \tag{14}$$

$$F_{i,1} = e^{A_{i,1}^*}. \tag{15}$$

$$F_{i,2} = \frac{\sum\limits_x \left\{\tilde{N}\left(x_i \mid z=1;\mu_i(z),\sigma_i(z)\right)\cdot e^{A_{i,0}^* + C_i^*(z=1)\left[x_i^*(z=1)-B_i^*(z=1)\right]^2}\cdot\left[1-P_i\left(y=1 \mid x_i^*(z),z=1;\beta_i^*\right)\right]\right\}}{\overline{P_i\left(y=1 \mid z=0;\mu_i(z),\sigma_i(z),\beta_i^*\right)}\cdot\sum\limits_x \tilde{N}\left(x_i \mid z=1;\mu_i(z),\sigma_i(z)\right)}. \tag{16}$$

$F_{i,1}$ is a constant, and therefore it is independent of birth weight. $F_{i,2}$ represents the combined effects of all birth weight–related factors on the mortality disparity between infants born to mothers with different levels of education in subpopulation i. In particular, birth weight–related factors include differences in shape and horizontal shift of the reverse J–shaped standardized birth weight–specific mortality curve, as well as nonlinear transformation between the probability and the logit of infant death at any standardized birth weight.

Data

The data for these analyses were obtained from the 2001 NCHS U.S. vital statistics linked birth–infant death data files. Race and ethnic origin is based on mother's reported race and ethnic origin. Births with missing information or gestational age < 20 weeks or birth weight < 500 g are excluded. The data are not further selected or adjusted by gestational age. Maternal education is dichotomized as high (i.e., college or more, $z = 1$) versus low (i.e., high school or less, $z = 0$). Summary statistics for the birth cohorts of interest are presented in Table 1.

Table 1 Descriptive statistics for the sample populations

Maternal Education	Birth Cohort	No. of Births	CDR[a]	Birth Weight Distribution (in grams)					
				Mean	5 %	25 %	Median	75 %	95 %
High School or Less ($z = 0$)									
	European American females	432,034	4.73	3,278	2,410	2,977	3,290	3,628	4,111
	European American males	454,249	6.36	3,393	2,460	3,090	3,430	3,751	4,253
	African American females	162,394	7.72	3,061	2,055	2,778	3,095	3,420	3,912
	African American males	167,571	10.22	3,165	2,097	2,863	3,210	3,544	4,038
	Mexican American females	222,480	3.57	3,299	2,495	3,005	3,315	3,610	4,090
	Mexican American males	230,989	4.45	3,393	2,523	3,090	3,402	3,735	4,224
College or More ($z = 1$)									
	European American females	583,889	2.45	3,390	2,580	3,090	3,402	3,714	4,180
	European American males	614,327	3.05	3,512	2,637	3,204	3,540	3,856	4,338
	African American females	89,290	6.04	3,150	2,154	2,863	3,203	3,515	4,005
	African American males	92,281	7.86	3,266	2,211	2,977	3,317	3,655	4,155
	Mexican American females	43,746	2.97	3,322	2,523	3,033	3,335	3,629	4,111
	Mexican American males	45,275	3.31	3,424	2,551	3,135	3,446	3,761	4,252

[a]CDR = Crude death rate (death per 1,000 births). Refers to infant death (i.e., death in the first year of life).

Results

Maternal education level influences the birth weight distribution through changes in the mean and standard deviation of both the primary and the secondary birth weight distributions (Fig. 3 and Table 2). The mean of the primary subpopulation increases significantly with education in all six birth cohorts examined. The increase is largest for European Americans (~100 g) and smallest for Mexican Americans (~30 g). The standard deviation of the primary subpopulation birth weight density declines for the European (~20 g) and Mexican (~4 g) American cohorts, but these declines are significant only for European Americans. Among African American births, the primary standard deviation increases for both sexes (~9 g) but is significant only for males. Overall, these shifts in the birth weight distribution represent improved birth outcomes using the standard metrics, such as mean birth weight, or the estimated rate of low birth weight (i.e., the proportion of births < 2,500 g). In particular, the rate of low birth-weight declines for the primary subpopulation of all populations (Table 2; Fig. 3, panel a).

The mean birth weight of the secondary subpopulation increases significantly (by 227–253 g) with higher maternal education level for the European American cohorts (panel b of Fig. 3; Table 2). The standard deviation of secondary birth weight declines

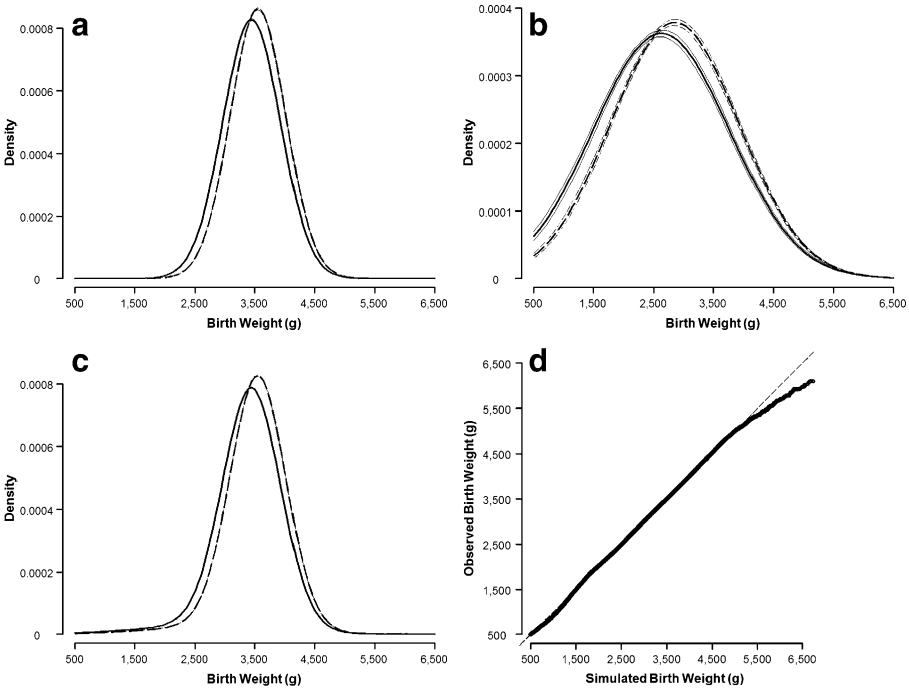

Fig. 3 Birth weight densities by maternal education: European American males, 2001. In panels a–c, the solid lines represent low education, and the dashed lines represent high education. Corresponding fine lines are the bias-adjusted 95 % confidence intervals, which, when they overlap the predicted line, are not visible. Panel a represents the primary subpopulation, panel b represents the secondary subpopulation, and panel c represents the total population. Results for all six populations studied are similar. However, differences in birth weight distributions by maternal education for Mexican Americans are smaller compared with European American and African American births. Panel d is a Q-Q plot indicating the goodness of fit of the mixture model to the birth weight density. The example given is European American male births to mothers with high educational levels. The results for the other populations also indicate excellent fits

significantly (by 51–66 g) with education among the European American cohorts, but increases significantly (by 114–152 g) among the African American cohorts. Overall, the increase in mean birth weight and decline in the standard deviation clearly improve European American birth weight densities. The decline in the secondary low birth-weight rate in these populations exceeds 8 % (Table 2). The increase in the standard deviation among secondary African American births has little effect on the rate of low birth weight (Table 2), since the mean of secondary birth weight is below 2,500 g and the density is truncated at 500 g. Consequently, among African American and Mexican American birth cohorts, there is little or no change in the secondary birth weight densities associated with educational level, at least based on the standard metrics, such as the rate of low birth-weight.

Finally, higher education is associated with an increase in the proportion of primary births, although this is typically less than 1 % of total births and is significant only for European American and African American male births. Nevertheless, it represents an improvement in birth outcomes using the standard metrics of mean birth weight and the rate of low birth weight because the primary subpopulations have

Table 2 Model-estimated changes (with bias-adjusted 95 % confidence intervals in parentheses) in birth weight distribution and mortality characteristics by maternal education

		Females			Males		
Birth Cohort	Parameters	European American	African American	Mexican American	European American	African American	Mexican American
Primary Subpopulation							
	μ_p (g)	103 (101; 105)*	88 (83; 93)*	27 (22; 31)*	108 (106; 110)*	95 (91; 99)*	35 (30; 41)*
	σ_p (g)	−20 (−22; −18)*	7 (1; 11)*	−3 (−10; 3)	−19 (−22; −17)*	9 (3; 14)*	−4 (−12; 2)
	LBW[a] rate (%)	−2.0 (−2.1; −1.9)*	−2.0 (−2.3; −1.7)*	−0.5 (−0.7; −0.3)*	−1.4 (−1.5; −1.3)*	−1.5 (−1.8; −1.3)*	−0.4 (−0.5; −0.2)*
	Death rate[b]	−1.8 (−2.0; −1.6)*	−1.6 (−2.2; −1.2)*	−0.1 (−0.6; 0.5)	−2.2 (−2.4; −1.9)*	−2.2 (−2.7; −1.5)*	−0.8 (−1.4; −0.3)*
Secondary Subpopulation							
	π_s (%)	−0.4 (−0.9; 0.0)*	−0.8 (−1.5; 0.1)	−0.3 (−1.4; 0.9)	−0.8 (−1.2; −0.4)*	−1.6 (−2.4; −0.8)*	0.2 (−1.2; 1.7)
	μ_s (g)	253 (202; 304)*	−65 (−247; 97)	−36 (−183; 73)	227 (169; 273)*	−136 (−317; 20)	4 (−119; 126)
	σ_s (g)	−51 (−80; −28)*	114 (41; 175)*	−24 (−101; 47)	−66 (−87; −42)*	152 (93; 217)*	−29 (−95; 29)
	LBW[a] rate (%)	−8.3 (−9.9; −6.5)*	−0.5 (−3.4; 2.7)	1.3 (−2.7; 6.3)	−7.6 (−9.1; −5.5)*	0.4 (−2.4; 3.5)	−0.2 (−4.5; 4.3)
	Death rate[b]	−8.7 (−13.1; −5.4)*	0.2 (−6.1; 6.2)	−6.3 (−13.0; 1.7)	−16.0 (−19.6; −11.9)*	4.1 (−3.3; 12.5)	−5.1 (−10.8; 3.0)
Total Cohort							
	LBW[a] rate (%)	−2.6 (−2.6; −2.4)*	−2.3 (−2.5; −2.0)*	−0.5 (−0.7; −0.3)*	−2.1 (−2.2; −2.1)*	−2.2 (−2.4; −2.0)*	−0.3 (−0.5; −0.1)*
	Death rate[b]	−2.3 (−2.5; −2.1)*	−1.7 (−2.3; −1.0)*	−0.6 (−1.1; 0.0)	−3.3 (−3.6; −3.0)*	−2.4 (−3.0; −1.6)*	−1.1 (−1.7; −0.5)*

[a] LBW = Low birth weight (i.e., < 2,500 g).

[b] Death per 1,000 births.

*Estimate is significantly different from 0 at the $\alpha = .05$ level.

a higher mean and smaller standard deviation, and hence a lower rate of low birth weight. Consequently, considering all birth weight density factors, overall birth outcomes improve significantly with higher maternal education in all populations.

The logistic regression results indicate that mortality generally declines with increasing education (Fig. 4). The Kitagawa decomposition attributes the majority of the absolute decline in mortality with education to the primary subpopulation for all birth cohorts except Mexican American females (Table 3; Fig. 4, panel a). Changes in secondary subpopulation mortality are smaller and are significant only for the European American birth cohorts (Table 3; Fig. 4, panel b). Finally the mixing proportion effect reduces infant mortality for most birth cohorts, significantly for African and European American males. However, this latter effect is small.

Further decomposition of the change of infant mortality into direct (independent of birth weight) and indirect (associated with changes in birth weight) effects of maternal education level indicates a strong direct effect in the primary subpopulation, which reduces infant mortality (Table 4). This direct effect is significant for all birth cohorts except Mexican American males. There is also an

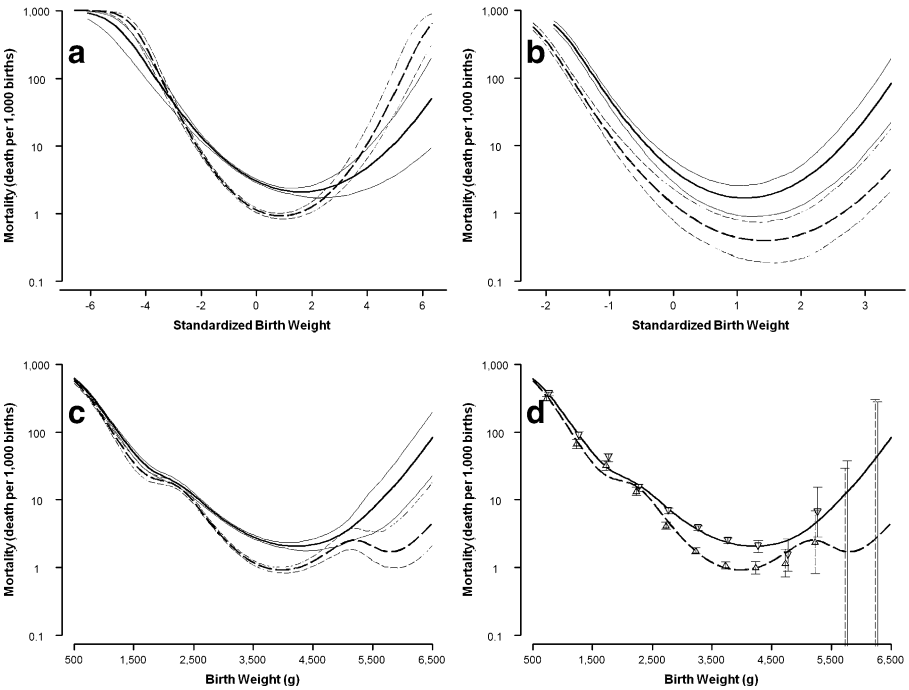

Fig. 4 Standardized birth weight–specific mortalities by maternal education: European American males, 2001. The solid lines represent low education, and the dashed lines indicate high education. Corresponding fine lines are the bias-adjusted 95 % confidence intervals. Panel a represents the primary subpopulation, panel b shows the secondary subpopulation, and panels c and d are for the total population. In panel d, the inverted triangles and the triangles are the observed birth weight–specific mortalities (estimated from binned data) for low and high education, respectively. Results for all six populations studied are similar. However, differences in mortality curves by maternal education are smaller for Mexican American births compared with European American and African American births

Table 3 Kitagawa decomposition of the infant mortality (death per 1,000 births) disparity by maternal education, with bias-adjusted 95 % confidence intervals in parentheses[a]

Birth Cohort	Mixing Proportion Effect	Rate effect		Total disparity
		Secondary	Primary	
European American Females	−0.08 (−0.16; 0.00)	−0.52 (−0.79; −0.33)*	−1.68 (−1.90; −1.47)*	−2.28 (−2.54; −2.06)*
European American Males	−0.19 (−0.28; −0.09)*	−1.09 (−1.34; −0.78)*	−2.03 (−2.26;−1.78)*	−3.31 (−3.58; −3.03)*
African American Females	−0.23 (−0.51; 0.02)	0.02 (−0.58; 0.59)	−1.50 (−2.04; −1.05)*	−1.71 (−2.34; −0.97)*
African American Males	−0.75 (−1.16; −0.34)*	0.38 (−0.30; 1.20)	−2.00 (−2.48; −1.31)*	−2.37 (−3.07; −1.57)*
Mexican American Females	−0.05 (−0.26; 0.14)	−0.42 (−0.89; 0.12)	−0.12 (−0.59; 0.49)	−0.60 (−1.11; 0.01)
Mexican American Males	0.04 (−0.28; 0.34)	−0.41 (−0.91; 0.29)	−0.77 (−1.26; −0.25)*	−1.14 (−1.72; −0.51)*

[a]Based on the estimated death rates shown in Table 2.

*Estimate is significantly different from 0 at the $\alpha = .05$ level.

indirect effect for the primary subpopulation, which tends to increase mortality with higher education. It is significant for all three female cohorts but not for male birth cohorts. That the indirect effects increase mortality with higher education is surprising given that the primary birth weight distribution improves with increased education as described earlier. The results for the secondary subpopulation are less consistent. In general, a direct effect in the secondary subpopulation reduces mortality but is significant only among European American and African American males and Mexican American females. The indirect

Table 4 Subpopulation-specific relative risk of infant mortality by maternal education decomposed into direct and indirect multiplicative factors with bias-adjusted 95 % confidence intervals in parentheses[a]

Birth Cohort	Relative Risk	Direct Factor	Indirect Factor
Primary Subpopulation			
European American females	0.47 (0.42; 0.52)*	0.40 (0.35; 0.47)*	1.16 (1.03; 1.36)*
European American males	0.48 (0.44; 0.52)*	0.45 (0.37; 0.56)*	1.07 (0.87; 1.21)
African American females	0.66 (0.57; 0.76)*	0.46 (0.37; 0.57)*	1.45 (1.22; 1.76)*
African American males	0.63 (0.55; 0.74)*	0.58 (0.47; 0.72)*	1.07 (0.86; 1.30)
Mexican American females	0.94 (0.74; 1.25)	0.55 (0.10; 0.90)*	1.69 (1.03; 8.65)*
Mexican American males	0.69 (0.49; 0.89)*	0.96 (0.65; 1.33)	0.72 (0.50; 1.02)
Secondary Subpopulation			
European American females	0.66 (0.55; 0.77)*	1.33 (0.50; 3.10)	0.50 (0.18; 1.55)
European American males	0.54 (0.47; 0.63)*	0.23 (0.07; 0.57)*	2.31 (1.27; 4.71)*
African American females	1.01 (0.83; 1.20)	0.53 (0.07; 3.34)	1.88 (0.25; 15.57)
African American males	1.08 (0.94; 1.28)	0.14 (0.02; 0.74)*	7.60 (2.93; 40.89)*
Mexican American females	0.71 (0.46; 1.10)	0.22 (0.07; 0.96)*	3.23 (0.30; 23.04)
Mexican American males	0.80 (0.59; 1.16)	0.47 (0.12; 1.74)	1.68 (0.54; 4.07)

[a]Based on the estimated death rates shown in Table 2.

*Estimate is significantly different from 1 at the $\alpha = .05$ level.

effect generally increases mortality with education but is significant only for European American and African American males. Again, this indirect effect is not consistent with the improvements in the birth weight distribution observed for European American male secondary birth weight densities described earlier. African American male secondary birth weight densities are largely unaffected by education. Perhaps the lack of consistently significant results is due to the relatively small absolute effect sizes associated with the secondary subpopulation (Table 3). Overall, effects of education on mortality are shown in Fig. 4, panel c.

It is possible that neonatal mortality is more closely or strongly associated with birth weight than is infant mortality. A second analysis based on neonatal mortality was also conducted. The results are not shown here because they are consistent with the analysis of infant mortality. No cases of significant indirect, or direct, effects were observed in the analysis of neonatal mortality that were not also observed in the analysis of infant mortality. On the other hand, in several cases, significant indirect and direct effects in the analysis of infant mortality were not significant in the analysis of neonatal mortality. In general, infant mortality is more informative with respect to the potential direct and indirect (through birth weight) effects of education than is neonatal mortality.

Discussion

Maternal education appears to have little effect on Mexican American birth outcomes compared with the other birth cohorts. In particular, the effect of maternal education on the Mexican American birth weight distributions tends to be small. Primary (normal) mean birth weight does increase by education, but the change is only approximately one-third of the effect observed for European American and African American birth cohorts. The impact of education on mortality is smaller as well. An overall effect is significant only for the male cohort. It is not clear why this population does not respond to the high level of maternal education like other birth cohorts. However, Mexican American birth cohorts have the lowest observed infant mortality rate of any of the populations examined (Table 1). In addition, approximately 64.3 % of Mexican American births are to Mexican-born mothers. Perhaps the ethnic difference in response to maternal education noted earlier is a part of the “Hispanic paradox” that is thought to influence infant mortality in recent migrant populations (Hummer et al. 1999; Iván 2002; Lopez-Gonzalez et al. 2005).

There may also be sex differences in the response of infant mortality to maternal education. Overall, male mortality appears to benefit more from higher education than female mortality does, particularly among African American and European American cohorts. This appears to be due largely to primary (normal) indirect effects, which tend to increase mortality more for females than males, even though birth weight distributions improve with higher maternal education for both sexes. Additional analyses are needed to determine whether this sex bias is a consistent difference and what factors might be responsible for this difference.

The African American versus European American racial disparity in infant mortality increases by maternal education: that is, the high-education population has a larger racial difference both in absolute difference in the death rates and in the relative risk. In particular, the low-education group has racial disparities (in terms of relative risk) of 1.63 (1.51 – 1.75) and 1.61 (1.52 – 1.69) for females and males, respectively. The high-education group has racial disparities of 2.46 (2.21 – 2.71) and 2.57 (2.36 – 2.81) for females and males, respectively. The fact that racial disparities increase with increasing education levels has been observed in a number of settings (Din-Dzietham and Hertz-Picciotto 1998; Singh and Yu 1995). Our decomposition attributes the majority of this increase in the racial disparity to the compromised subpopulation: that is, mortality improves significantly among compromised European American births and not among compromised African American births with increased education (Table 3). The normal subpopulation contributes slightly to the racial difference in that mortality declines more with increasing education among European Americans, but this difference is not significant for either sex. Finally, the mixing proportion effect reduces the racial disparity with increased education, by reducing mortality among African Americans more than among the remaining populations. This latter effect is significant only for males. Decomposition into direct and indirect effects is not consistent across the sexes in the compromised subpopulation, so whether these are birth weight–dependent or birth weight–independent effects is unclear.

It is well documented that birth weight increases and infant mortality declines with increased maternal education (Din-Dzietham and Hertz-Picciotto 1998; Gortmaker 1979; Kramer 1987; Singh and Yu 1995), although infant mortality has been reported to increase at very high levels of maternal education (e.g., more than 16 years of education) (Haglund et al. 1993; Shoham-Yakubovich and Barel 1988). The results presented earlier are consistent with these general findings. The division of maternal education levels by >12 years of school versus ≤12 completed years of school does not allow an examination of birth weight and infant mortality at very high educational levels. Overall, birth weight increases with educational level (as indicated by increased mean birth weight) declines in the standard deviation of birth weight, and hence a decline in the rate of low birth-weight. These are largely driven by the normal subpopulation of births, the dominant group occurring predominately in the center of the birth weight distribution. The compromised births, which account for the majority of births in the tails of the birth weight distribution but also account for births at all birth weights, are more heterogeneous. European American compromised birth weights improve, but African American and Mexican American compromised births remain more or less constant.

The decline in infant mortality with higher education is largely due to direct effects in the normal subpopulation. The beneficial effects of education on mortality are entirely independent of the improvements in the birth weight distribution. There are significant indirect effects of education. However, these all increase infant mortality despite general improvements in the birth weight density. These birth weight–dependent effects are overwhelmed by the direct effects of education, so overall infant mortality declines with increasing education. These results suggest that the current U.S. national policy of reducing infant mortality by reducing the rate of low birth weight (U.S. Department of Health and Human Services 2000) might be ineffective or even detrimental. There are at least some situations in which improved birth weight distributions are associated with higher—not lower—mortality, as is generally assumed.

Care must be taken with respect to interpreting the decomposition into direct and indirect effects. The methods of effect decomposition used here are based on statistical decision theory (Geneletti 2007), as opposed to the more common "counterfactual" approaches (Robins et al. 2000; VanderWeele 2009) qualitatively applied to infant mortality by Hernández-Diaz et al. (2008). Here, we model the educational level–specific birth weight density as a mixture of two Gaussian distributions with subpopulation birth weight–specific mortality curves as second-degree polynomials of standardized birth weight. By standardizing birth weight (within specific educational levels and subpopulations), the main effect of the association of education and birth weight is eliminated, and the regressions can estimate the direct effect of education on infant mortality versus any indirect effect as interaction terms of education and birth weight on infant mortality. Effect decomposition can then be estimated using a procedure similar to direct standardization (Geneletti 2007). Geneletti (2007) called this a "generated direct effect," which is similar to Pearl's "natural direct effect" (Pearl 2009). Because the birth weight density is modeled as the mixture of two Gaussian distributions (truncated at 500 g) and the direct/indirect decomposition are carried out separately for normal and compromised births, the appropriate distribution is theoretically available for direct standardization. See Geneletti (2007) for proof of identification and Gage et al. (2010) for an initial application to birth outcomes.

The decision theory approach, as well as the counterfactual approach, requires the same strong assumptions: (1) no unmeasured covariates that affect education and educational disparities in infant mortality, (2) no unmeasured confounding of education and birth weight, and (3) no unmeasured confounding of birth weight and infant mortality. Assumption 1 is required to estimate total educational effects, and all three are necessary for the decomposition into generated direct (Geneletti 2007) and indirect effects. Given our use of education as a proxy measure of socioeconomic level, it is unlikely that assumptions 1 and 2 are true, at least with respect to education. Consequently, the analysis presented here does not address whether education is a part of the causal pathway to birth weight or to infant mortality. It is best if the effects discussed here are interpreted as the result of socioeconomic level, which encompasses a spectrum of associated covariates (many of which are unmeasured or even unknown) as opposed to the effect of education *per se*. In addition, the analysis does not identify the proximate determinants that mediate the relationship (if it exists) of socioeconomic level with either birth weight or infant mortality. Nevertheless, these are the same data and assumptions that are commonly used to estimate socioeconomic disparities in infant mortality. The question addressed here is whether birth weight mediates socioeconomic disparities in infant mortality.

Following Hernández-Diaz et al. (2008) and based on the theories of Basso, Wilcox, and others (Basso and Wilcox 2009; Basso et al. 2006), we assume that the reverse J shape of the birth weight–specific infant mortality is due to confounding and not a causal effect of birth weight on infant mortality. We account for this confounding by modeling infant mortality as a second-degree polynomial of birth weight. This could cause bias if the second-degree polynomial does not fit the infant mortality data well. However, a second-degree polynomial is considered the appropriate model of birth weight–specific infant mortality, assuming homogeneity of the birth cohort (Fryer et al. 1984). Our CDDmlr model corrects for significant

unmeasured heterogeneity in the total birth cohort by using a weighted mixture of two second-degree polynomials (i.e., one for each subpopulation identified) and thus fits the overall birth weight–specific mortality curve better than a single polynomial (Gage 2002). Any significant education by birth weight interaction terms of the second-degree polynomials of standardized birth weight represent possible indirect effects mediated by birth weight. These could be due either to an interaction of education and birth weight on infant mortality (in which case, birth weight is on the causal pathway) or to an association of education with the unmeasured confounders affecting birth weight and infant mortality (in which case, birth weight is not on the causal pathway but cannot be distinguished from an interaction of birth education and birth weight). Thus the significant indirect effects reported earlier do not imply that birth weight is necessarily causal. However, if no indirect effect is observed, then birth weight cannot be on the causal pathway except in the unlikely event that all the effects of education on all unmeasured covariates and birth weight cancel each other out.

Consequently, despite the limitations, the results and interpretations presented in this article can provide statistical support to the hypothesis of Wilcox and others (Hernández-Diaz et al. 2008; Wilcox and Russell 1990) that birth weight is not on the causal pathway to infant mortality. In particular, the statistical evidence indicates strong associations of birth weight with socioeconomic level (e.g., maternal education), but that the improvements in infant mortality are generally due to factors independent of improvements in the birth weight distribution. The fact that significant changes in the birth weight density do not result in significant changes in infant mortality in 7 of 12 cases supports the hypothesis that birth weight is not on the causal pathway to infant mortality. Similar results have been found using the same methods with exogenous covariates maternal age (Gage et al. 2009) and race (Gage et al. 2010) instead of maternal education. In the case of maternal age (Gage et al. 2009), no convincing evidence for any indirect effects operating through birth weight was observed in either normal or compromised births. In the case of race (Gage et al. 2010), no indirect effects acting through birth weight occurred in the normal subpopulation, although possible indirect effects are observed in the compromised subpopulation.

Given that birth weight is not generally causal, explorations of other potential proximate determinants of infant mortality are needed. In this regard, it would be useful to repeat these analyses using gestational age in place of birth weight. Perhaps gestational age is the proximate cause of the variation in both infant mortality and birth weight. This will be described in a future analysis. A second possible cause of variation in birth weight and infant mortality might be fetal programming (Barker et al. 1990). This theory is based on the association between (low) birth weight and chronic disease later in life. However, fetal-programming theory explicitly assumes that birth weight is not causal and it argues that metabolic systems are programmed during fetal development as a function of (poor) maternal nutrition, which then influences both birth weight and adult disease. This process can be considered adaptive if it improves pre-reproductive survival and/or reproduction, a view that is consistent with the indirect effects reported earlier. Failure of this adaptive system to overcome maternal/fetal constraints might be the cause of infant mortality. Perhaps the compromised subpopulation represents

programmed births. CDDmlr would be useful for exploring either gestational age or fetal programming.

The advantage of CDDmlr over standard statistical methods is that it documents the changes in the birth weight density and the infant mortality curve and can distinguish between direct and indirect (through birth weight) effects of a covariate while controlling for unobserved heterogeneity, which we have interpreted as normal and compromised fetal development in the case of birth weight. This allows the identification and decomposition of differences in birth weight and infant mortality into a number of latent components. Although CDDmlr was designed specifically to examine birth outcomes, it is not limited to this application. Compared with conventional regression methods, CDDmlr is useful wherever the density of a potential mediator (e.g., birth weight) is well described by a finite mixture model, and this mixture accounts for some unmeasured heterogeneity in a dependent variable (e.g., infant mortality). The introduction of additional covariates as instruments can determine whether the proximate determinant could be on the causal pathway. For example, in a case similar to birth weight, another biomarker—body mass index (BMI), a measure of obesity—is closely associated with mortality in adults (Waaler 1984), again in a reverse J–shaped pattern. BMI–specific mortality tends to shift right or left along with changes in the mean of BMI (Su 2005). This is very similar to the dynamics of birth weight and infant mortality (Fig. 1, panel a or b) and is the original basis for arguing that birth weight was not on the causal pathway to infant mortality (Wilcox and Russell 1990).

Conclusions

Education, a proxy measure of socioeconomic status, is associated with significant changes in birth weight distributions and infant mortality.

1. Birth outcomes' responses to education are much stronger among African Americans and European Americans than among Mexican Americans.
2. Male birth outcomes respond more strongly to education than female birth outcomes, controlling for race and ethnicity.
3. Birth outcomes' responses to education are stronger among European Americans than among African Americans. Thus racial disparities increase with education.
4. In all racial and ethnic groups, birth outcomes—such as mean birth weight, standard deviation in birth weight, and rate of low birth weight—improve with higher education.
5. Mortality declines with higher education. This is entirely independent of the changes in birth weight and is due to direct effects.
6. The significant indirect effects (potentially causal through birth weight) all tend to increase infant mortality even though birth outcomes generally improve.

Our national policy of reducing infant mortality by intervening at the level of birth weight may not have the desired effect.

Acknowledgments This project is supported by NIEHS R01 HD037405. We'd like to thank Dr. Howard Stratton for his many useful discussions and extensive comments on the draft. We'd also like to thank Kiersten Westbrook for her work in preparing the manuscript.

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