Low-weight Neonatal Survival Paradox in the Czech Republic

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Analysis of vital statistics for the Czech Republic between 1986 and 1993, including 3,254 infant deaths from 350,978 first births to married and single women who conceived at ages 18–29 years, revealed a neonatal survival advantage for low-weight infants born to disadvantaged (single, less educated) women, particularly for deaths from congenital anomalies. This advantage largely disappeared after the neonatal period. The same patterns have been observed for low-weight infants born to black women in the United States. Since the Czech Republic had an ethnically homogenous population, virtually universal prenatal care, and uniform institutional conditions for delivery, Czech results must be attributed to social rather than to biologic or medical circumstances. This strengthens the contention that in the United States, the black neonatal survival paradox may be due as much to race-related social stigmatization and consequent disadvantage as to any hypothesized hereditary influences on birth-weight-specific survival. *Am J Epidemiol* 1999;149:447–53.

birth weight; ethnic groups; gestational age; infant mortality; neonatology; racial stocks; socioeconomic factors; survival analysis

It is well known that in the United States, the mortality rate for black infants is twice that for white infants. Controlling for social class, educational background, marital status, and other dimensions of social structure fails to dispel the race-based contrast in survival rates. This contrast remains the highest profile, most perplexing, and most studied disparity in life chances in the United States. Higher black infant mortality in part reflects the fact that black infants, more often than white infants, weigh less than 2,500 g at birth (1–3). At normal birth weights (2,500 g or more), black infants also have a higher risk of dying during the first year than do white, Asian, or Hispanic infants (4), but the excess of black low-weight births accounts for the largest share of the total difference in infant mortality (5), since most infant deaths occur after low-weight births.

However, when low-weight births are considered separately, black infants have a lower mortality rate than white infants of a similar birth weight (6, 7), at least during the first weeks of life (8). After the neonatal period, higher death rates for black infants reappear even for low-weight births. The temporary survival advantage is due particularly to lower rates of death from congenital anomalies among black low-

weight infants (9–12). Even at normal birth weights, blacks have a lower death rate than whites for congenital anomalies, whereas some studies (e.g., (3)) have found that for blacks, rates of death from other causes such as infections and injuries far exceed those for whites.

In an attempt to understand this inversion in survival chances, Wilcox and Russell (7) have called for population-specific standards for birth weight. They point out that a weight considered dangerously low for a white infant might be more normal for a black infant if normal birth-weight distributions differ by race, as they are known to do for sex (7, 13). However, racial categories in part may be labels for other factors whose impact on infant mortality should not be lost. "Race" is a social construct with no generally agreed-upon or understood intrinsic scientific meaning (14). In the United States, groups conventionally defined as white or black, for example, are composed of genetically diverse persons of varying degrees of mixed ancestry. In a society in which perceived, ascribed, or self-identified race is a stigmatizing source of social disadvantage, with intrinsic consequences for education, employment, marriage, and many other aspects of life, any inherent biologic racial elements are at most part of the explanation for differences in birth weight and infant survival.

Differences in birth-weight distributions and infant survival may be due to social conditions as much as to race-linked heredity (15, 16). Otherwise, it is difficult to understand why the increase in the incidence of low

birth weight among children who have one black and one white parent (relative to those who have two white parents) is twice as large when the father is black as when the mother is black (17). This is reinforced by the finding that the birth-weight distribution of births to African-born blacks in Illinois (1980–1995) resembles that of births to US-born whites more than that of births to US-born blacks (18). Commenting on the persistent effect of race found in data for the United States, Hogue and Hargraves place most of the blame on socioeconomic inequalities but conclude that "we must question whether eliminating poverty is sufficient to eliminate social differences in infant mortality" (19, p. 9). The call for population-specific standards for birth weight may apply equally well to other groups definable by lifestyle (20, 21) or a distinctive niche in society (22).

International research shows that when nonbiologic criteria are used to identify social disadvantage, low-weight infants born to socially disadvantaged women have the same temporary survival advantages associated with race in the United States. In Israel, Shoham-Yakubovich and Barell (23) documented a temporary survival advantage for low-weight infants born to women with only elementary schooling or less, compared with corresponding infants of more educated women, after controls for maternal age and parity were introduced. At the same time, the mortality rate for normal-weight infants born to women with a low level of education was more than twice as high as the rate for corresponding infants of more educated mothers. What is observed in the United States as a "race" effect is evident in Israel as an effect of educational level. While hereditary group differences in weight distributions may be a plausible hypothesis for explaining the race paradox in survival of US infants, the same can hardly be said when the survival paradox is associated with social factors such as education. In this paper, we add evidence to this growing literature by showing that the same neonatal survival paradox exists in terms of marital status and educational level for mothers in the Czech Republic.

MATERIALS AND METHODS

Data

The Czech Ministry of Public Health furnished individual birth records from the Czech Republic for 1986–1992. Infant death records from 1986 to 1993 were matched to these births. Only 0.5 percent of all death records were unmatched compared with 2.5 percent of all US death certificates in the 1993 matched file of natality/infant mortality data. Thus, the matched Czech records were of better quality than US national-level data and at least as good as the best state-level matched files.

The value of studying Czech data is twofold. First, the Czech Republic has one of the world's most ethnically homogenous populations available for study; fully 94 percent of the people are ethnic Czech (24). Therefore, it is highly unlikely that variations in birth-weight distributions based on marital status or education reflect any hereditary differences. Second, the access that Czech women have to prenatal and other health care is not sharply differentiated with respect to social factors such as marital status, education, and occupation. According to a World Health Organization and Centers for Disease Control study (25), 99 percent of pregnant Czech women obtained prenatal care during the years studied. More than 94 percent of them made 10 or more prenatal visits. Virtually all births in the country occurred in the maternity wards of state-run hospitals because physicians were all state employees who were not allowed to maintain private practices. Thus, all women were exposed to essentially the same institutional conditions during labor and childbirth.

On the basis of the ethnic homogeneity and the uniformity of health care in the Czech Republic, observed patterns in birth-weight distributions and associated infant survival rates should therefore result from underlying differences in lifestyles and social conditions rather than from differences in proximate exposure to health care during pregnancy or hereditary differences in the birth-weight distribution itself. Such factors cannot be held constant in the United States; ethnically, the population is very diverse, and, even within ethnic groups, differences in education and income mean very different patterns of exposure to health care during pregnancy.

Analysis

We defined mortality as the number of infant deaths in a specific population subgroup and segment of time divided by the corresponding number of person-years (or other person-time units) of exposure to risk. For cases who died, we assigned half a day of risk during the day that death occurred, whereas cases who survived that day contributed a whole day of exposure to risk. All counts of person-days represented aggregations of half-day units of time contributed by individual cases. Those who survived to day 365 were treated as right censored. Numbers of person-days lived and infant deaths, as well as the corresponding risks of death at various levels of variables, are shown in table 1. We converted infant deaths to annual rates per thousand by dividing the number of deaths by person-days lived and multiplying that number by 365.25 days per year and then by 1,000.

Also presented in table 1 are the numbers of newborn infants at risk in each category of each of the first

TABLE 1.   Risk of infant death for first births to Czech women aged 18–29 years, 1986–1992

Variable	Livebirths	Person-days lived	Infant deaths	Deaths/1,000 person-years*	Infant mortality rate†
Total	350,978	126,867,531.5	3,254	9.37	9.27
Birth weight (g)					
<1,500	2,041	390,948.0	996	930.53	488.00
1,500–2,499	16,097	5,589,738.5	812	53.06	50.44
2,500–3,499	220,875	80,163,936.5	1,144	5.21	5.18
3,500–4,999	111,965	40,722,908.5	302	2.71	2.70
Gestation (weeks)					
25–32	3,305	822,918.5	1,079	478.91	326.48
33–36	13,076	4,553,812.5	616	49.41	47.11
37–38	40,871	14,774,416.0	385	9.52	9.42
39–41	293,726	106,716,384.5	1,174	4.02	4.00
Marital status					
Single	26,027	9,379,661.0	330	12.85	12.68
Married	324,951	117,487,870.5	2,924	9.09	9.00
Education					
Basic	27,090	9,752,150.5	382	14.31	14.10
Vocational	137,504	49,681,926.5	1,349	9.92	9.81
Higher	186,384	67,433,454.5	1,523	8.25	8.17
Time since birth‡					
0–23 hours	350,978	350,425.5	1,105	1,151.75	
1–2 days	349,873	542,378.5	464	312.47	
3–6 days	349,409	1,396,706.0	404	105.65	
1 week	349,005	2,441,837.5	285	42.63	
2–3 weeks	348,720	4,880,605.0	176	13.17	
4–7 weeks	348,544	21,946,746.0	332	5.53	
8–12 weeks	348,212	31,675,349.0	250	2.88	
13–51 weeks	347,962	63,651,485.0	238	1.37	
52 weeks	347,724				

* Deaths divided by person-days lived multiplied by 365.25 days per year multiplied by 1,000.
† Deaths per 1,000 livebirths.
‡ In completed units; the first column lists the number of liveborn infants who survived to the beginning of each interval.

four variables shown and the corresponding infant mortality rate, computed as the number of infant deaths per 1,000 newborn infants. The two measures of mortality largely coincide for low to moderate risks but deviate when mortality is high because of the uneven timing of deaths. The overall average risk of 9.37 deaths per 1,000 person-years lived is comparable with the infant mortality rate of 9.27 deaths per 1,000 livebirths calculated by using the same data. The more detailed measure of risk was slightly higher than the traditional infant mortality rate but can be interpreted in much the same way.

As shown, the risk of infant mortality declined strongly as birth weight increased. Following extensive exploration of alternatives, we merged these birth-weight categories into two larger groups: less than 2,500 g (low birth weights) and 2,500–4,999 g (normal birth weights). Excluded from analysis was a tiny category of infants who weighed 5,000 g or more at birth. Using these two conventional categories facilitated comparison with the previous literature without distorting most

significant patterns observed for finer-weight gradations and also provided more events in each category.

Besides birth weight, gestational age at birth is usually an important indicator of the level of infant mortality (15, 26). In our study, the mortality pattern showed that the risk of death based on gestational age was similar to that based on birth weight. Again, to enable comparisons with previous studies, we grouped the first two categories shown in table 1 (25–32 weeks, 33–36 weeks) into less than 37 weeks since onset of the last normal menstrual period (premature births) and the last two categories (37–38 weeks, 39–42 weeks) into 37–41 weeks since the last normal menstrual period (term births). We excluded the tiny number of pregnancies that lasted 42 weeks or more after onset of the last normal menses. For post-term gestational-aged infants, the risk of death again increases rapidly and would distort the patterns for normal, full-term births.

We restricted our detailed analysis to women aged 18–29 years, since most births occur at these ages in the Czech Republic. This restriction removed most

substantive concerns about the effects on birth outcomes of particularly young or old mothers. We also restricted our attention to first births, as was done in a similar analysis of black-white differences for first births to college-educated women in the United States (27). Doing so removed concerns about the effects of variations in birth order and interval on outcomes of subsequent pregnancies and about any dependence between outcomes across births to the same mother.

We studied both single and married women and found a higher risk for term births to single mothers (table 1). Few divorced or widowed women who gave birth were included in these records, and even fewer of these births were first births at ages 18–29 years; therefore, divorced and widowed women were excluded from analysis.

Besides marital status, educational level also measures social position. In the Czech Republic, basic education is equivalent to completion of the eighth grade in US schools, a level attained by virtually all Czech citizens. At that point, schooling diverges into two tracks (vocational training or the academic high school called gymnasium), as it does in many other European countries. Students who complete gymnasium have one more year of schooling than US high school students do. Some women with a gymnasium diploma go on to university. Since almost all university graduates in our study married before they gave birth, we combined university graduates with those who had a gymnasium diploma to form a category called higher education. Risk values for these two groups were quite similar (not shown), unlike the sharply different values for the marital-status groups. Also, university graduates are drawn almost exclusively from those who have a gymnasium diploma, and many of the latter may be prediploma university students when they give birth, although we could not determine whether this was true in our study.

Finally, measuring risk as the number of events per person-time unit of exposure enabled us to compute a life table for the first year of life (table 1). As shown, the risk of infant death declined exponentially with time since livebirth. Age was measured in correspondingly longer successive intervals to provide comparable numbers of deaths for each interval. After extensive exploration using alternative intervals, we also collapsed those intervals shown into the neonatal period (the first 28 days of life) and the postnatal period (the remainder of the first year of life) to summarize those survival patterns that were compatible with findings from previous studies.

RESULTS

Mortality rates for the neonatal and postneonatal periods, by birth weight, gestational age, and mother's marital status and educational level, are shown in table

2. For normal-weight births (2,500–4,999 g), the standard pattern of higher infant mortality among disadvantaged mothers occurred with few exceptions during both periods. (The risk of death for infants born to those women with higher education who remained single throughout pregnancy seemed unaccountably high.)

As in other studies, a temporary survival advantage for low-weight infants born to disadvantaged women was also found in the Czech data. During the neonatal period, infant death rates for low-weight births among both single and married mothers increased rather than decreased systematically with educational level. Furthermore, for low-weight infants born to mothers with either basic or vocational education, the neonatal mortality rate for single mothers was lower than that for married mothers. This finding constitutes the neonatal survival paradox of interest and replicates findings for the US population in terms of racial differences. The same neonatal survival advantage for low-weight infants born to disadvantaged women also occurred for premature infants born to these women (table 2), providing a different way to observe the same phenomenon.

To show that both birth weight and gestational maturity are important, separate ways of identifying the survival-risk inversion, we analyzed both measures simultaneously. The survival inversion during the neonatal period was clearest for those infants who were both low weight and premature but largely absent for those who were both normal weight and full term. Signs of the survival inversion were evident for "mixed" cases (those who were low weight but full term or normal weight but premature) but were not as consistent and clear when one or the other measure of potential fetal distress was missing. We concentrated on the neonatal period because the numbers of postneonatal deaths were insufficient to enable simultaneous disaggregation by weight and gestational age.

When we used more detailed birth-weight categories (not shown), we found that the survival advantages for very-low-birth-weight infants (less than 1,500 g) born to disadvantaged women were not as consistent as those that occurred for infants in the upper range of low birth weights. Using more detailed infant-age intervals also showed that during the first day of life, there was less deviation than there was later in the neonatal period from the standard trend of improved survival with better education. Although the survival inversion under study appears to be a feature of the neonatal period for low-weight births, it cannot be traced to the birth event itself, nor does low birth weight show a consistent dose-response relation to the chance for survival. Similarly, the inverse survival advantage for low-birth-weight black infants in the United States also has been found to be concentrated

TABLE 2. Infant deaths per 1,000 person-years at risk for first births to Czech women aged 18–29 years, by birth weight, gestational age, maternal education, and marital status, 1986–1992

Marital status and education	Birth weight (g)		Gestation (weeks)		Gestation <37 weeks, birth weight (g)		Gestation 37–41 weeks, birth weight (g)	
	<2,500	2,500–4,999	<37	37–41	<2,500	2,500–4,999	<2,500	2,500–4,999
Neonatal period								
Single								
Basic	895.04	34.27	971.79	51.87	1,433.73		264.83	35.72
Vocational	1,009.58	38.00	1,153.77	40.10	1,661.64	209.46	207.78	33.59
Higher	1,445.21	34.70	1,403.93	36.53	2,379.48	159.72	241.94	31.77
Married								
Basic	1,162.14	44.37	1,366.15	50.09	2,079.47	159.85	260.22	41.68
Vocational	1,271.49	34.47	1,398.88	37.87	2,164.58	260.03	329.35	30.35
Higher	1,374.85	30.71	1,379.23	32.07	2,315.91	229.82	290.51	27.14
Postneonatal period								
Single								
Basic	20.31	2.80	21.33	3.23				
Vocational	18.15	2.79	18.40	2.96				
Higher	23.78	1.79	29.68	1.49				
Married								
Basic	13.25	3.81	12.85	3.98				
Vocational	12.45	2.18	10.14	2.36				
Higher	12.39	1.64	10.57	1.72				

more clearly at low birth weights than at very low birth weights, again directly paralleling these results (6, 7).

Birth-weight distributions

The survival advantage for low-weight infants born to disadvantaged women disappears when 1) the birth-weight curves for different groups are shifted along the *x*-axis so the means of their predominant distributions coincide, and 2) the neonatal-mortality curves are shifted in lockstep, as suggested by Wilcox and Russell (7, 28). Disadvantaged women have higher rates of loss at all such standardized birth weights, just as Wilcox and Russell found when comparing US blacks and whites. Likewise, the inversion paradox also disappears when a birth weight is defined as "low" if it is among the lowest 4 percent in a particular social group. (The 4 percent cutoff point suggested itself for our data because it is the fraction of birth weights below 2,500 g in our most favored social group: married women with higher education.)

All of these results parallel previously documented differences in birth-weight distributions based on race (3, 6, 7, 16). Birth weights of infants born to disadvantaged groups versus more favored groups of women in the Czech Republic tend to be lower. Women in less advantaged social groups also tend to give birth earlier, a feature that our parallel study of fetal period (29) showed to result in higher risks of premature livebirth. However, for all groups in the present study, the distri-

bution of gestational age spiked sharply at 40 weeks, and no shifting procedure was available for these distributions analogous to that developed by Wilcox and Russell (7, 28) for birth-weight distributions.

Causes of death

We grouped causes of infant death into the following four categories by using codes from the *International Classification of Diseases*, Ninth Revision: perinatal conditions (codes 760–761, 764–766, and 772–779), delivery complications (codes 762–763 and 767–770), congenital anomalies (codes 740–759), and all other residual causes (10, 30). The numbers of deaths occurring during the postneonatal period did not enable us to make statistically reliable generalizations about cause-of-death-patterns, but we were able to consider the difference between low-weight and normal-weight births in relation to deaths during the neonatal period (table 3). Regarding the risk of neonatal mortality from all causes (repeated from table 2), risk inversions were evident in both marital status and basic and vocational educational categories for low-weight births, but they occurred only for single women with a basic education who gave birth to normal-weight infants.

Regarding cause-specific mortality, as for all causes together, there was little evidence of the neonatal survival paradox at normal birth weights, at least in terms of education. A lower level of education among mar-

TABLE 3.   Neonatal deaths per 1,000 person-years at risk for first births to Czech women aged 18–29 years, by cause group, birth weight, maternal education, and marital status, 1986–1992

Marital status and education	Cause of death and birth weight (g)									
	All causes of death*		Congenital anomalies†		Delivery complications‡		Perinatal conditions§		Residual causes¶	
	<2,500	2,500–4,999	<2,500	2,500–4,999	<2,500	2,500–4,999	<2,500	2,500–4,999	<2,500	2,500–4,999
Single										
Basic	895.04	34.27	94.93	10.71	189.86	12.85	583.13	2.14	27.12	8.57
Vocational	1,009.58	38.00	139.79	10.48	279.58	17.03	543.62	1.31	46.60	9.17
Higher	1,445.21	34.70	105.75	14.61	211.49	10.96	1,127.97	0.00	0.00	9.13
Married										
Basic	1,162.14	44.37	121.84	16.00	440.49	12.36	581.07	4.36	18.74	11.64
Vocational	1,271.49	34.47	217.97	15.35	326.96	10.90	703.06	2.45	23.51	5.78
Higher	1,374.85	30.71	223.68	14.54	379.87	10.11	728.88	2.41	42.42	3.65

* Figures from table 2.
† International Classification of Diseases, Ninth Revision (ICD-9) codes 740–759.
‡ ICD-9 codes 762–763 and 767–770.
§ ICD-9 codes 760–761, 764–766, and 772–779.
¶ All other ICD-9 cause-of-death codes.

ried mothers translated into a higher neonatal mortality rate for infants in every cause category. Among single women, the education gradient was less clear within cause categories; it was disrupted by particularly high mortality rates associated with higher education for deaths from congenital anomalies and with vocational training for deaths from delivery complications. Among normal-weight infants born to married mothers (by far the modal category in these data), congenital anomalies accounted for at least half of all neonatal deaths. However, among single women with less than a higher education, delivery complications replaced congenital anomalies as the modal cause-of-death category for normal-weight infants.

On the other hand, and in relation to marital status and cause-of-death category, the neonatal survival paradox occurred almost as frequently with normal-weight births as with low-weight births. Being single seemed to confer an actual survival advantage, rather than a penalty, to normal-weight infants in each cause category. Only in terms of the two associations just noted (higher education with congenital anomalies and vocational education with delivery complications) did single women have higher rates of loss than married women. Thus, the cause structure of neonatal mortality differed for single and married women, even for normal-weight infants.

For low-weight infants, whose general neonatal survival rates were better when their mothers were single or had less education, perinatal conditions replaced congenital anomalies as the leading cause of death, followed by delivery complications and then congenital anomalies (the reverse in every respect of patterns at normal birth weights). The survival-risk inversion was clearest and most consistent for congenital anom-

alies, again mirroring results from analyses of blacks and whites in the United States (9–12, 30). For example, for low-weight births, the ratio of the risk in the most disadvantaged category (single women with a basic education) to the risk in the most advantaged category (married women with a higher education) was below unity for every cause category, but it was smallest for congenital anomalies.

DISCUSSION

The neonatal survival paradox (higher neonatal survival rates for low-weight infants born to disadvantaged women) essentially is a signal that the procedure used to compare mortality rates among different population groups of infants of the same birth weight (7) and/or the same level of maturity at birth is too simple to enable identification of the mechanisms that produce differential chances of survival. It appears that women in more favored social groups are better able to keep even high-risk fetuses in utero longer and to deliver them as livebirths at normal weights and that low-weight or premature birth becomes a more important distress signal for them than for women in more disadvantaged groups.

The neonatal survival advantage for infants born to disadvantaged women, which is viewed as a black-white issue in the United States, appears in the Czech Republic in relation to educational differences and also largely to differences in marital status. In many respects, details of this paradox coincide in the two populations, including characteristics of the birth-weight distributions and associated birth-weight-specific mortality risks as well as patterns by cause of death. It seems implausible that there would be sys-

tematic hereditary genetic differences between single and married Czech women or between Czech women with a basic education and those who were better educated, differences that for some hereditary reason would predispose the disadvantaged groups to give birth earlier to systematically smaller, lighter-weight infants. These patterns must be understood to be the result of social outcomes, produced by the different circumstances that these groups of women experienced before giving birth. It is a pattern produced by nurture, not nature.

The patterns of neonatal survival documented here for the Czech Republic closely mirror findings already established for the black-white contrast in infant survival rates in the United States. Therefore, at least some element of the race-related pattern in the United States may be due to the social realities of racial identification rather than to hereditary influences on birth-weight distributions or birth-weight-specific survival.

REFERENCES

1. Kleinman JC, Kessel SS. Racial differences in low birth weight. Trends and risk factors. N Engl J Med 1987; 317:749–53.
2. Eberstein IW. Demographic research on infant mortality. Sociological Forum 1989;4:409–22.
3. Adams MM, Berg CJ, Rhodes PH, et al. Another look at the black-white gap in gestation-specific perinatal mortality. Int J Epidemiol 1991;20:950–7.
4. Forbes D, Frisbie WP. Spanish surname and Anglo infant mortality: differentials over a half-century. Demography 1991; 28:639–60.
5. Behrman RE. Premature births among black women. N Engl J Med 1987;317:763–5.
6. North AF Jr, McDonald HM. Why are neonatal mortality rates lower in small black infants than in white infants similar in birth weight? J Pediatr 1977;90:809–10.
7. Wilcox A, Russell I. Why small black infants have a lower mortality rate than small white infants: the case for population-specific standards for birthweight. J Pediatr 1990;116:7–10.
8. Min KJ. Life chances in infancy: comparative birth weight influence on survival for races and sexes. Unpublished Master's thesis. University of South Carolina, Columbia, SC, 1983.
9. Eberstein IW, Parker JR. Racial differences in infant mortality by cause of death: the impact of birth weight and maternal age. Demography 1984;21:309–21.
10. Durant TM. South Carolina infant mortality: cause-specific analysis of racial mortality differences, 1980–1987. Presented to the Southern Demographic Association, Atlanta, GA, October 21, 1994.
11. Berry RJ, Buehler JW, Strauss LT, et al. Birth weight-specific infant mortality due to congenital abnormalities, 1960 and 1980. Public Health Rep 1987;102:171–81.
12. Binken NJ, Rust KR, Williams RL. Racial differences in neonatal mortality. What causes of death explain the gap? Am J Dis Child 1988;142:434–40.
13. Tompkins ME, Alexander GR, Jackson KL, et al. The risk of low birth weight. Alternative models of neonatal mortality. Am J Epidemiol 1985;122:1067–79.
14. Cooper R. A note on the biologic concept of race and its application in epidemiologic research. Am Heart J 1984; 108:715–22.
15. Foster HW Jr. The enigma of low birth weight and race. N Engl J Med 1997;337:1232–33.
16. Leck I, Lancashire RJ. Birth prevalence of malformations in members of different ethnic groups and in the offspring of matings between them, in Birmingham, England. J Epidemiol Community Health 1995;49:171–9.
17. Parker JD, Schoendorf KC. Influence of paternal characteristics on the risk of low birth weight. Am J Epidemiol 1992;136:399–407.
18. David RJ, Collins JW Jr. Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S.-born whites. N Engl J Med 1997;337:1209–14.
19. Hogue CJ, Hargraves MA. Class, race, and infant mortality in the United States. Am J Public Health 1993;83:9–12.
20. Meyer MB, Comstock GW. Maternal cigarette smoking and perinatal mortality. Am J Epidemiol 1972;96:1–10.
21. Terrin M, Meyer MB. Birth weight-specific rates as a bias in the effects of smoking and other perinatal hazards. Obstet Gynecol 1981;58:636–8.
22. Wang X, Guyer B, Paige DM. Differences in gestational age-specific birthweight among Chinese, Japanese and white Americans. Int J Epidemiol 1994;23:119–28.
23. Shoham-Yakubovich I, Barell V. Maternal education as a modifier of the association between low birthweight and infant mortality. Int J Epidemiol 1988;17:370–7.
24. Federalni Statisticky Urad. Pohyb Obyvatelstva v Ceske a Slovenske Federativni Republice v Roce 1989. (In Czech). Cast 1. Prague, Czechoslovakia: Czech Federal Statistical Office, 1989.
25. World Health Organization/Centers for Disease Control. 1993 Czech Republic reproductive health survey: final report. Atlanta, GA: Centers for Disease Control and Prevention, 1995. (US Government Printing Office publication no. 1995-636-496).
26. Kline J, Stein Z, Susser M. Conception to birth: epidemiology of prenatal development. New York, NY: Oxford University Press, 1989.
27. McGrady GA, Sung JF, Rowley DL, et al. Preterm delivery and low birth weight among first-born infants of black and white college graduates. Am J Epidemiol 1992;136:266–76.
28. Wilcox AJ, Russell IT. Birthweight and perinatal mortality: III. Towards a new method of analysis. Int J Epidemiol 1986;15:188–96.
29. Carlson E, Hoem JM, Rychtarichova J. Trajectories of fetal loss. Presented to the Population Association of America, Washington, DC, March 28, 1997.
30. Eberstein IW, Nam CB, Hummer RA. Infant mortality by cause of death: main and interaction effects. Demography 1990;27:413–30.