The Relationship Between Social Stratification and All-Cause Mortality Among Children in the United States: 1968–1992

John H. DiLiberti, MD, PhD

ABSTRACT. *Background.* US childhood poverty rates have increased for most of the past 2 decades. Although overall mortality among children has apparently fallen during this interval, these aggregate mortality rates may hide a disproportionate burden imposed on the least advantaged. This study assessed the impact of social stratification on long-term US childhood mortality rates and examined the temporal relationship between mortality attributable to social stratification and childhood poverty rates.

Methods. Using US childhood mortality data obtained from the Compressed Mortality File (National Center for Health Statistics) and a county-level measure of social stratification (residential telephone availability), I evaluated the impact of social stratification on long-term trends (1968–1992) in age-adjusted mortality and compared the resulting attributable proportions to trends in childhood poverty rates.

Results. Between 1968 and 1987 the proportion of US childhood deaths attributable to social stratification decreased from .22 to .17. Subsequently, it increased to .24 in 1992, despite continuous declines in overall childhood mortality rates. These proportions correlated strongly with earlier childhood poverty rates, taking into account an apparent 9-year lag. Among black children comparable trends were not observed, although throughout this time period their mortality rates were far higher than among the rest of the population and declined more slowly.

Conclusions. Despite declining childhood mortality rates between 1968 and 1992, children living in the least advantaged counties continued to die at higher rates than those living in the most advantaged counties. This differential worsened considerably after 1987, and by 1992 had a substantive impact on US life expectancy at birth, resulting in perhaps the most significant (in terms of years of life lost) reversal in the health of the US public in the 20th century. *Pediatrics* 2000;105(1). URL: http://www.pediatrics.org/cgi/content/full/105/1/e2; *socioeconomic status, children, mortality, social stratification, life expectancy.*

ABBREVIATION. SS, social stratification.

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Mortality rates among infants and children in the United States and other industrialized nations declined throughout the 20th century, except for relatively short periods of extreme economic and political turmoil.[1,2] Suggested reasons for these declines include improving standards of living, decreases in family size, public health services (sanitation, water, and immunizations), and more recently, modern individual medical care. The relative contributions from each remain uncertain and controversial.[3–8] Not all members of a population participate equally in these mortality declines, however. For example, among blacks, mortality and morbidity rates generally exceed those of whites; life expectancy in mid-17th century England increased among the peerage ~100 years before the general population—the earliest documented systematic improvement in human life expectancy.[7–12] Population-wide infant mortality did not decline substantively until the early 20th century—long after comparable trends were evident among older children and adults.[13,14]

Mounting evidence supports consistent associations among social stratification (SS), the health of a population, and the individuals who comprise it.[15–18] (SS suggests a broader scope of influences than the more commonly used socioeconomic status.) A wide range of health outcomes has been linked to SS, including mortality, morbidity from various diseases, and health services utilization.[3] Relationships between SS and health outcomes among children have received far less attention than for adults. Few studies have examined this association among US children; investigations of long-term trends have apparently not been done.[19–22]

During the past 3 decades, children in the United States experienced several large swings in economic fortune as a consequence of changing government programs and policies, and periodic variation in the economic cycle. The poverty rate among children, ~26% in the early 1960s, dropped to 14% in 1969. It remained primarily in the 15% to 16% range until 1980 when it surpassed 18%, more recently rising to exceed 20% for the 1980s and 1990s.[23,24] Throughout this period, however, mortality rates among children generally fell.[25] It is uncertain whether all children benefited equally, or whether changes in the US economy since the early 1980s have resulted in increasing mortality gradients between less and more advantaged US children. To investigate this possibility, I evaluated trends in the association between SS

and all-cause mortality rates (1968–1992) among US children.

METHODS

Mortality data for this report were obtained from the Compressed Mortality File (National Center for Health Statistics), which summarizes all US deaths for 1968–1992, stratified by age group, race, sex, year, and 4-digit *International Classification of Disease* code and aggregated by county of residence.[26] (For records covering the period 1968 through 1978, although all the data are present, the aggregations were incomplete. This error was taken into consideration in all analyses, so it will not affect any of the results.) Population data are provided in a companion file. For the youngest age group, the number of live births, rather than population count, was provided. During the study period, extensive changes took place in the geographic coding system among counties in the states of Alaska, Hawaii, and Virginia, which were therefore excluded from analysis. All-cause mortality rates were calculated for each remaining US county for age groups: 0 to 364 days, 1 to 4 years, 5 to 9 years, 10 to 14 years, and 15 to 19 years. Age-adjusted mortality rates for all causes, 0 to 19 years, were calculated using the direct standardization method, with 1980 as the reference year.[27]

SS was represented by the proportion of occupied housing units having a telephone, using 1970, 1980, and 1990 census data. This variable has been shown to have the strongest correlation ($\rho = .33$; $P < .00005$) at the county level with all-cause mortality among US children, compared with other commonly used aggregate-level measures of SS, including per capita income, educational achievement, income distribution, or median housing value.[20] Data used for the analysis of potential SS variables were obtained from the Area Resource File (February 1995), a dataset distributed by the Office of Research and Planning, Bureau of Health Professions.[28] For each census year, continuous data were converted to categorical data, divided into approximate quintiles (of population, not counties). The 1970 telephone categories were used for analysis of the 1968–1975 interval, 1980 categories for 1976–1984, and 1990 categories for 1985–1992. The value for each county level SS category was linked to county levels of deaths and population by age group.

I calculated 4 mortality ratios comparing the mortality rate for each quintile of SS with the most advantaged quintile. Piecewise linear regression, using year as the independent variable, was performed with the mortality ratios split into 1968–1986 and 1986–1992 time intervals. This analysis was performed using the *regress* command in the Stata statistical software package, Release 3.1 (Stata Corporation, College Station, TX). Attributable proportions were calculated using standard methods.[29]

Poverty time series data from 1959 through 1995 for US children <19 years of age were obtained from the US Bureau of the Census.[23] Cross-correlation between the attributable proportion time series and the US poverty time series was performed using the *Trans* subroutine from the ITSM software package for time series analysis, with lag times varying from 5 to 10 years.[30,31] Life table analysis of the effect of SS on life expectancy at birth was performed using standard methods.[32]

RESULTS

Age-adjusted all-cause mortality among children declined for each quintile of SS throughout the entire time period, except for occasional minor year-to-year fluctuations (Fig 1). None of the trend lines crossed except for a very slight overlap of quintiles 3 and 4 in 1987 and 1992. Trends in the mortality ratios, comparing mortality rates among children living in successively less advantaged quintiles of counties with those in the most advantaged, seemed to decline (with the exception of a slight increase in the quintile 4:quintile 5 ratio) from 1968 through 1986 and then increase until 1992 (Fig 2). This biphasic trendline was modeled by segmenting the data into 2 time periods: 1968–1986 and 1986–1992. Coefficients for these piecewise linear regressions are presented in
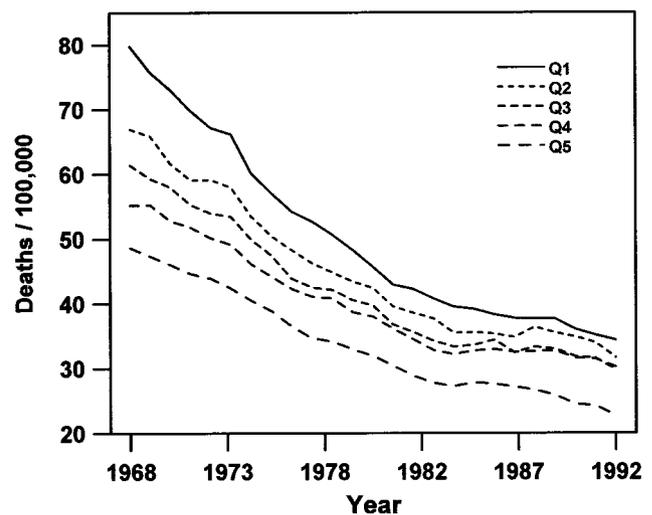

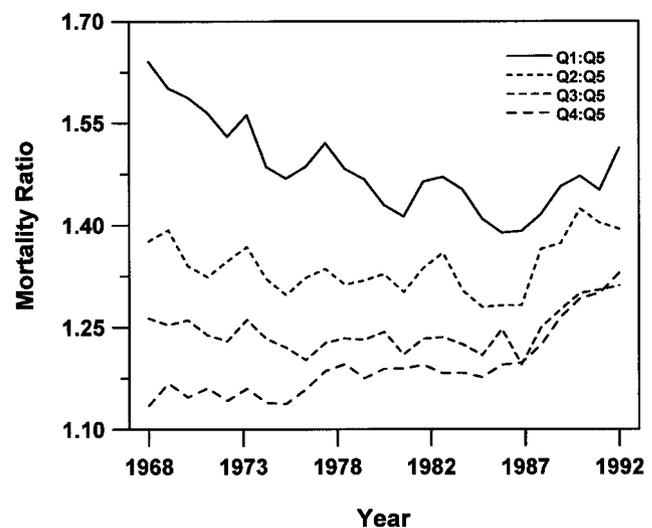**Fig 1.** Trends in all-cause mortality by quintile of SS.

Fig 2. Trends in all-cause childhood mortality ratios comparing quintile of SS.

Table 1. These coefficients represent the linear trend in the annual rate of change for each mortality ratio for the designated interval. There was very little slope for the 1968–1986 segments, but 3 of 4 were slightly negative, indicating a small downward trend in mortality ratios. For 1986–1992, however, all 4 slopes became positive, with steeper slopes corresponding to an increase in mortality. This may be seen more clearly in Fig 3, which shows the time trend for the attributable proportion—that fraction of total mortality associated with SS.

Between 1968 and 1987 the proportion of US childhood deaths attributable to SS declined from .22 to .17, corresponding to an absolute reduction in attributable deaths from ~28 000 to 12 000 annually (Fig 4). Subsequently, the attributable proportion increased to nearly .24 in 1992, with a corresponding increase in childhood deaths attributable to SS to 16 000 in 1990. This figure dropped slightly in 1991 and 1992 as the all-cause mortality rates for children declined. Despite this decline, SS accounted for an increasing proportion of the remaining mortality.

TABLE 1. Regression Analysis of Trends in Attributable Risk

Quintile Ratio	1968–1992		1968–1986		1986–1992	
	β	R^2	β	R^2	β	R^2
Q1:Q5	−.0067	.55	−.11	.83	.020	.87
Q2:Q5	.00066	.015	−.0036	.44	.023	.75
Q3:Q5	.0013	.10	−.0015	.24	.016	.73
Q4:Q5	.0059	.71	.0029	.58	.024	.97

Linear regression coefficients, β, and R^2 values for trends in mortality ratios comparing successively more advantaged county quintiles to the most advantaged quintile.

When examined on an age group basis, mortality trends by SS quintile were quite similar to the age-adjusted trends—downward over time with little crossover between SS quintiles. No clear trends among mortality ratios, comparing the least advantaged with the most advantaged quintiles, were evident, although for each age range the nadir in mortality ratio occurred more toward the middle than either end of the time period.

Among black children there was no consistent association between mortality rates and SS category. The gradients in mortality observed by SS quintile among all children did not seem to be present for either the age-adjusted rate or the individual age group rates. For all but 1 year and age group, however, age-adjusted mortality rates for black children living in the most advantaged counties exceeded the rates for the white population living in the least advantaged (Fig 5).

The cross-correlation between the proportion of children living in poverty and the proportion of childhood deaths attributable to SS reached .96 ($P <$ .001) if a lag of 9 years was built into the model, ie, when poverty rates were compared with the attributable proportion 9 years later (Fig 6). Similarly, the correlation coefficient obtained, comparing the 1959–1978 poverty data with the 1968–1987 mortality data was .75, whereas the corresponding coefficient for the 1978–1983 to 1987–1992 intervals was .91.

The life expectancy at birth calculated from the Compressed Mortality File data for 1992 was 75.85 years for males and females combined. When the measured effects attributable to SS were removed, ie, the childhood mortality rates from the most advantaged counties were used in the life table, the life expectancy at birth increased to 76.05 years.

DISCUSSION

These observations should leave little doubt regarding the existence of an association between SS and childhood mortality. During the entire 25-year interval, children living in less advantaged county quintiles, measured by the availability of residential telephones, experienced higher mortality rates than those in the more advantaged county quintiles. The monotonic mortality gradient present in 1968 persisted throughout the study period, despite continuously declining mortality rates for each quintile. The risk of dying for children living in the least advantaged quintile was consistently 50% to 60% higher than the risk for those living in the most advantaged quintile. Several previous studies that examined
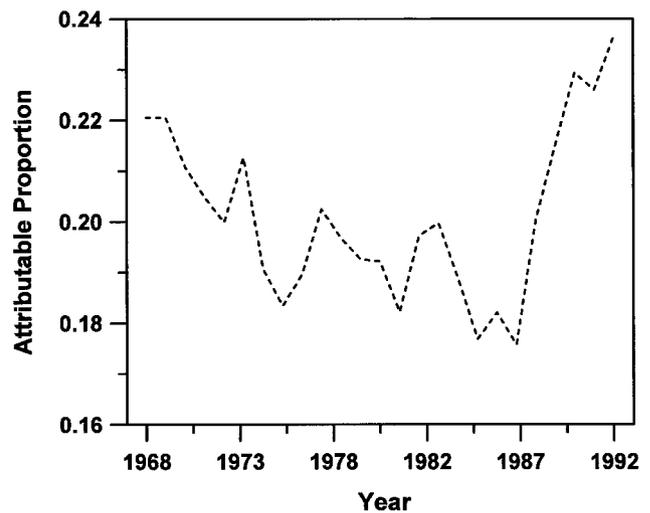

Fig 3. Proportion of childhood mortality attributable to SS.
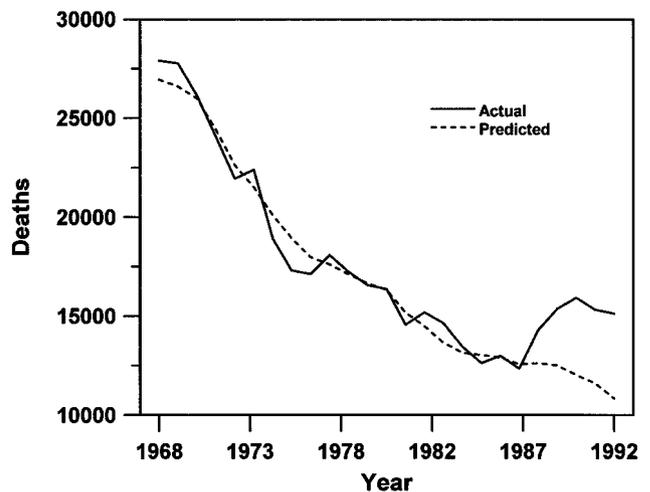

Fig 4. Childhood deaths attributable to SS. Predicted line based on linear trend in attributable proportion: 1968–1986.

smaller populations during shorter time intervals have identified similar mortality risks associated with SS. Wise et al[33] found an inverse relationship between median family income aggregated at the census tract level and mortality among 1582 Boston children who died between 1972 and 1979. In an evaluation of mortality among North Carolina children between 1985 and 1988, Nelson[34] found an overall risk ratio of 2.7 for children who received Aid to Families with Dependent Children payments compared with those who did not. Singh and Yu[35] found an inverse relationship between maternal educational achievement and infant mortality for 1964–1966 and 1987–1988, with the mortality ratio between the least- and most-educated groups increasing from 1.6 in the earlier period to 2.2 in 1988. When household income was used as a measure of SS, these ratios were 1.4 and 1.6, respectively. They subsequently found a similar relationship (mortality ratio: ≈3), comparing low income (<$10 000) to high income (>$15 000) families among 39 152 children (1–4 years) followed by the National Longitudinal Mortality Study between 1979 and 1985. The small num-
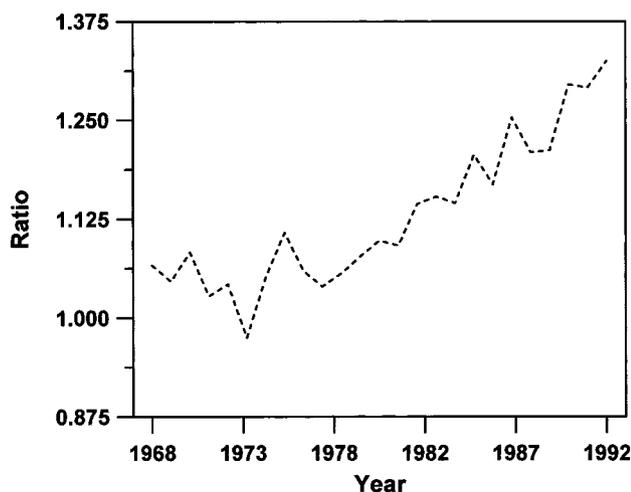
Fig 5. Ratio of age-adjusted all-cause mortality comparing black children in the most advantaged quintile to white children in the least advantaged quintile.
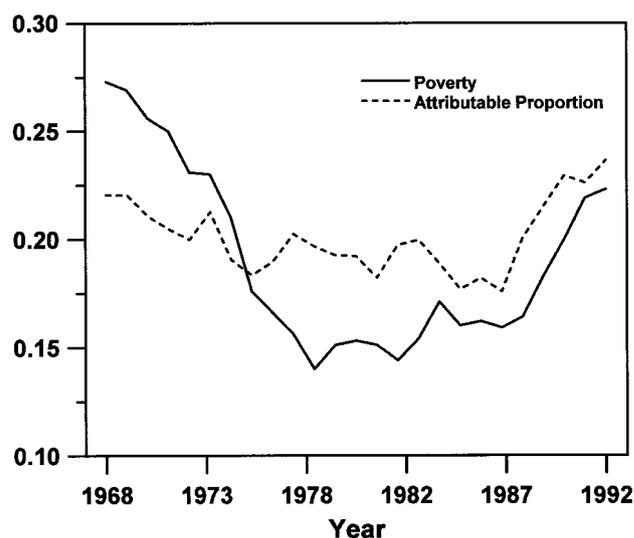

Fig 6. Trends in the proportion of US childhood mortality attributable to SS compared with US childhood poverty rates (with 9-year lag).

ber of deaths in the study (42) resulted in rather broad confidence intervals.[35]

The failure to detect a SS–mortality gradient among black children, although somewhat surprising, may reflect the study design. (The relatively small number of deaths among black children, when spread over ~3000 counties, might obscure any underlying gradient. The restricted geographic distribution of the black population also could tend to limit the ability of a study such as this one to detect a SS–mortality association.) The absence of a mortality gradient among black children should not be taken to imply that SS had no impact on their mortality rates. For each quintile of counties, black rates greatly exceeded those of other children. The impact of SS gradients on minority populations in the US has been examined by Keil et al,[36] who found that among adults dying with cardiovascular disease, differentials in mortality between whites and blacks almost completely disappeared when adjusted for individual level socioeconomic status, measured as a composite of educational and occupational indicators.

The choice of residential telephone availability as the measure of SS was driven by 2 considerations. First, with a study period covering 25 years, a variable measured at more than a single point was less likely to introduce bias. Second, previous work demonstrated that a measure of deprivation captures information not accessible to purely economic measures. The prevalence of residential telephones tells us something about the social and economic matrix of a community.[19] This fits nicely with the work of Carstairs and Morris,[37,38] who are proponents of the use of deprivation indices as measures of SS. They use variables such as the availability of an automobile in these analyses, claiming that purely economic measures of SS fail to reflect the observation that the impact of a given level of income may vary from household to household depending on a wide range of circumstances. Their deprivation scores attempt to capture how people are using their resources—the fewer discretionary items, the greater the level of deprivation. They have been able to demonstrate that deprivation captures at least some measure of the association between SS and health outcomes untapped by purely economic measures.[39] Use of any other SS variables would not have substantively changed the conclusions of this analysis, although the attributable proportions may have been somewhat lower. The fact that, with one minor exception, the mortality rates by quintiles of SS clearly were separated over a 25-year period argues in favor of the robustness of the use of telephone availability in the present study.

Studies such as this one, which use aggregate-level measures of an independent variable, have been criticized because of concern over introduced bias.[40] This phenomenon, cross-level bias, may occur when the level of inference differs from that of the measured independent variable. In this study, making an inference at the same (county) level as the measurement of the independent variable should not introduce bias. Thus, one may conclude that children living in less advantaged counties have increased mortality rates, rather than less advantaged children have increased mortality rates. The latter inference would be appropriate only if SS had been assessed at the individual rather than the aggregate level.

Although the strong association between SS and mortality does not prove a cause–effect relationship, it does go a long way toward satisfying the criteria for causation proposed by Hill, especially with regard to strength, consistency, temporality, biologic gradient, coherence, and plausibility.[40] The hypothesis of reverse causality, suggested to explain previously reported SS–health associations, seems virtually impossible to invoke as an explanation for the present findings. Proponents of reverse causality suggest that poor health leads to lower socioeconomic status as a consequence of the burdens of disease.[41] With the exception of chronic mental illness, empiric evidence provides no support for this proposal.[42] For it to be operational at all in the

present study, families with children at high risk of dying would have had to migrate continuously over a 25 year period from more advantaged to less advantaged counties—an implausible conjecture at best. The US population generally tends to migrate from poorer to more affluent regions.[43–45]

The view of US childhood mortality presented here is both reassuring and worrisome. It is reassuring because mortality rates for all children, whether examined by age group, ethnicity, or SS quintile, fell substantively throughout the 25-year interval. The data are worrisome, however, because of the widening gap between the less and more advantaged. The considerable reduction in the proportion of US childhood mortality attributable to SS accomplished between 1968 and 1987 reversed during the 1988–1992 interval. The public health impact of this worsening of mortality apparently related to SS is considerable. The reduction in 1992 overall life expectancy of .2 years attributable to childhood SS is 11% larger than the impact of prostate cancer and nearly as large as the impact of breast cancer, the complete elimination of which would improve life expectancy by .18 and .3 years, respectively.

The robust correlation between US childhood poverty rates and the proportion of childhood mortality attributable to SS suggests that the poverty rate is a strong indicator/mediator of the relationship between SS and childhood mortality. Visual inspection of the corresponding graphs, the time series analysis, and the piecewise correlation coefficients, taken together, provide reasonable support for this conjecture. The 9-year lag between the increase in poverty rates and the parallel increase in childhood mortality attributable to SS nicely fits a theory of mortality decline proposed by Murray and Chen.[1] They suggest population mortality declines from the accumulation of health promotive assets, such as health knowledge and attitudes, nutrition, clean water and air, sewers, immunizations, and individual health care, which together provide a buffer against short term deprivations. They offer as support empirical evidence showing trends in life expectancy (and mortality) have, in effect, a memory. Examining the increased mortality induced by severe political and/or economic disturbance, such as war, they found that it took some time for life expectancy to decline, and that at the end of the disturbance, life expectancy returned to the same trend line projected before the disturbance. This model indicates that health is probably associated with a complex matrix of resources, the sum of which is more important than any individual part. The buffering effect of health promotive assets would delay the impact of increasing SS on mortality. In the present study, mortality rates continued to decline among even the least advantaged children, but the rate of decline slowed, compared with that of peers who lived in more advantaged counties. It could be argued reasonably that increasing poverty reduces the rate of accumulation of health promotive assets, eventually slowing the decline in mortality rates. Presumably, it would take more intensive socioeconomic disruption to increase previously declining rates.

This study provides additional support for the perspective that the SS–health relationship operates on a continuous, rather than a threshold basis. For each decrement in SS, mortality worsened, similar, for example, to the Whitehall study, which demonstrated lower risk of cardiovascular disease by increasing social class (as determined by occupational status).[46] The effects of SS on health therefore have an impact throughout the population, not just the most disadvantaged. These effects apparently persist beyond childhood, compounding the public health impact. Several studies suggest that the risks for several adult-onset diseases are related to prenatal and early childhood events.[47–49]

The global impact of SS on mortality is large enough for Williams[50] to suggest that lower socioeconomic status is probably the most powerful single contributor to premature morbidity and mortality. The negative impact of this contributor on US childhood mortality seems to be growing. Federal and state economic and social welfare policy decisions made during the 1980s have subsequently led to a substantial redistribution of income from the less affluent to the more affluent.[51] These decisions apparently have had highly adverse effects on the well-being of US children. The combined effects of a persistent wide economic disparity and the observed 9-year lag until maximum mortality impact suggest the relative well-being of less affluent children in the US may continue to decline.

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