Long-Term Trends in Childhood Infectious Disease Mortality Rates

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ABSTRACT

Objectives. This study assessed long-term trends in US childhood infectious disease mortality rates (CIDMR).

Methods. We calculated age-adjusted and age group–specific US CIDMR (1968–1996) by using data from the Compressed Mortality File (1968–1992, 1996) and Multiple Cause of Death Files (1993–1995) of the National Center for Health Statistics and English data for historical comparison (1861–1964).

Results. US CIDMR declined continuously from 1968 to 1996, although the rate of decline slowed after 1974. Respiratory and central nervous system categories declined most; HIV-related deaths offset these declines somewhat.

Conclusions. CIDMR declined nearly 200-fold between 1861 and 1996, but no substantive improvement occurred after 1986. (*Am J Public Health.* 1999; 89:1883–1885)

Pinner et al. recently reported that US mortality from infectious disease increased 38% between 1980 and 1992.[1] Children (birth–4 years) constituted the only group with a discernible decline, but the limited age categories (birth–4 years, 5–24 years) made meaningful assessment of trends in childhood infectious disease mortality rates (CIDMR) difficult. The Pinner report covered a relatively short period, 1980 to 1992, which included only part of the HIV epidemic and did not include subsequent prevention and treatment strategies. Likewise, their analysis preceded some changes attributable to *Haemophilus influenzae* B immunization.[2] To more completely understand recent trends in CIDMR, we analyzed US data from 1968 through 1996. We also examined English data from 1861 to 1964 to compare these recent trends with a longer perspective.

Methods

We obtained English mortality data (1861, 1871, 1881, 1891, 1901, 1911, 1921, 1931, 1940, 1951, 1960, 1964) from Registrars General reports, which provide 4 separate infectious disease mortality rates—"Respiratory/TB," "Other Infectious and Parasitic," "Influenza/Pneumonia/Bronchitis," and "Diarrheal"—for 19 age categories.[3] We combined these to obtain composite CIDMR for each of the following age groups: birth to 1 year, 1 to 4 years, 5 to 9 years, 10 to 14 years, and 15 to 19 years.

We obtained US mortality data for 1968 through 1992 from the Compressed Mortality File,[4] for 1993 through 1995 from the Multiple Cause of Death data files,[5] and for 1996 from the Centers for Disease Control and Prevention Wonder Web Page.[6] For both US and English data, we calculated mortality rates for birth to 364 days, 1 to 4 years, 5 to 9 years, 10 to 14 years, and 15 to 19 years and then calculated age-adjusted rates, using direct standardization (1980 US population reference).[7]

The authors of the Pinner report kindly provided their *International Classification of Diseases, 9th Revision (ICD-9)* infectious disease codes, which we further subcategorized into "neonatal," "respiratory," "central nervous system," "gastrointestinal," "septicemia," "AIDS and other immune disorders," and "all other."[8] Review of *International Classification of Diseases, 8th Revision (ICD-8)* codes led to an analogous set.[9] In addition to tabulating CIDMR, we also calculated the annual rate of change of CIDMR according to the following formula:

$$\ln(R) = [\ln(r_1/r_0)]/t$$

where R is the annual rate of change, r_0 and r_1 are the actual rates at the start and end of the time period of interest, and t is the duration of the period.

Results

Age-adjusted CIDMR declined nearly monotonically between 1861 and 1996, although the rate of decline appeared to moderate after the first 100 years (Figure 1). Each individual age category beyond infancy demonstrated a pattern similar to the age-adjusted rates—steeply declining curves for approximately 1 century, followed by smaller decrements in the 1980s and 1990s. Among infants (<1 year), however, the mortality decline did not appear until 1921, 5 or 6 decades later than among older children. Mortality attributable to respiratory and central nervous system infections fell sharply between 1979 and 1995, while mortality from AIDS and other immune disorders increased following 1984. The 13% increase at the English/US data boundary (1964–1968) and 30% decrease at the *ICD-8–ICD-9* transition (1978–1979) may reflect artifacts. The rate at which CIDMR declined initially accelerated from 1881 through 1974 and then reversed course, becoming essentially 0 for 1986 through 1996 (Figure 2).

Discussion

Unlike reported infectious disease mortality among US adults, CIDMR declined during the past 2 decades, reaching an all-time low in 1993. Virtually all of

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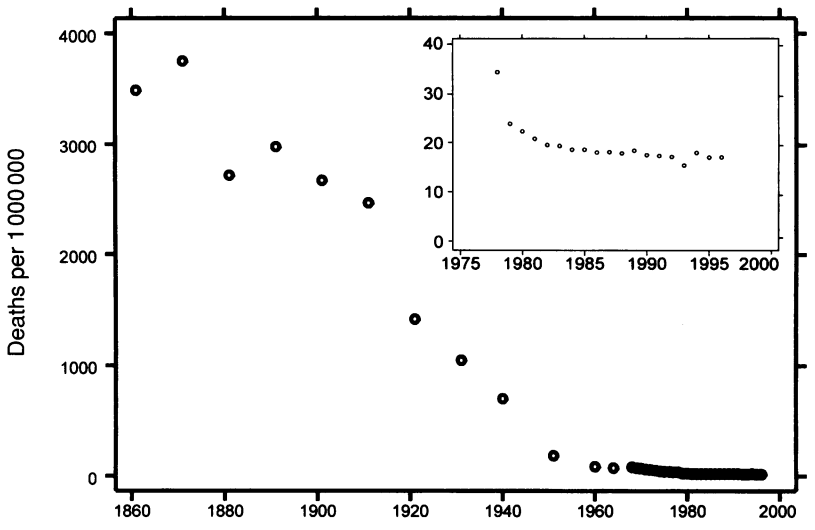


FIGURE 1—Age-adjusted childhood infectious disease mortality rates, 1861–1996 (1861–1964, England; 1968–1996, United States); inset: 1978–1996, United States.

the adult mortality increase resulted from 2 *ICD-9* codes: 486 (pneumonia—no organism specified) and 038.9 (septicemia—no organism specified); childhood rates for these codes declined throughout this period (1980–1992). Pinner et al. suggested emerging infections as possible explanations for rising adult septicemia and pneumonia mortality rates. The childhood data seem to refute this hypothesis—unless children are somehow protected from these organisms.

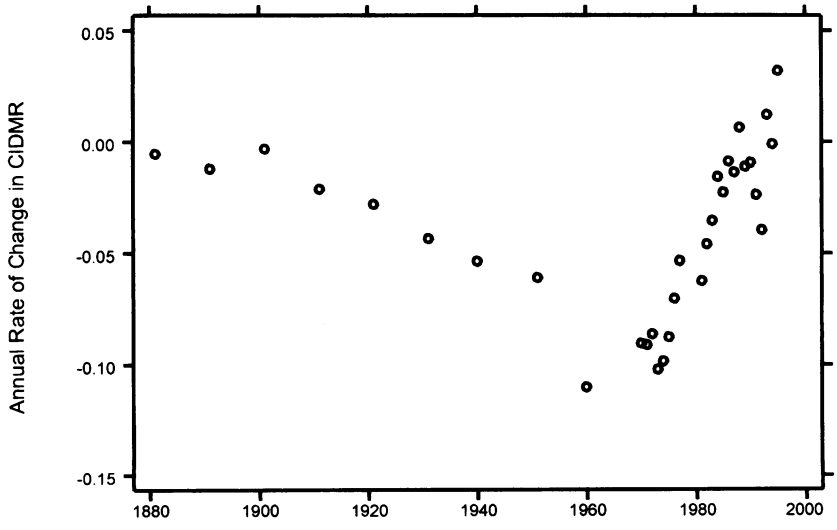
We examined several possible artifacts in our results and their interpretation. A linear scale on the y-axis—such as that used in Figure 1—may potentially lead to misinterpretation of the rate of change. Over extended time periods, a constantly declining rate will actually appear to plateau. Although logarithmic transformation avoids this potential visual artifact, we chose to calculate and display the compound annual rate of change (Figure 2). Although the resulting unit—annual proportion of change—is mathematically similar to the log of CIDMR, we believe it is more interpretable. We also attempted in Figure 2 to minimize the effects of the (possibly artifactual) abrupt increase in CIDMR at the English–US transition (1964–1968) and the decline at the *ICD-8–ICD-9* transition (1978–1979) by using 3-period moving averages (the mean of 3 consecutive data points) to smooth the data and by dropping the transition period data points. Clearly, CIDMR stopped declining toward the end of the study period, following a trend that began in the mid-1970s.

Age group–specific CIDMR declined in parallel over the entire study period, except among infants, for whom mortality changed little before 1921. This later onset of decline in the youngest category may reflect an important role for family size, which did not decline substantively until after the turn of the century, although other factors have been suggested.[10–12] Increasing poverty among children during the later portions of the study period did not directly cause the later plateau in CIDMR, because CIDMR declined more rapidly among the less-advantaged childhood population.[13] As CIDMR among less-advantaged children approaches those of the more advantaged, however, the decline in overall CIDMR might be expected to slow.

The decline in central nervous system mortality started well before *Haemophilus influenzae* B immunization became available, while varicella vaccine was released too recently to have had any impact on mortality. HIV deaths recorded after 1984 comprised a small proportion of total childhood infectious disease deaths and did not contribute substantively to CIDMR trends. Removal of HIV mortality from this analysis does, however, lead to a very small annual rate of decline in CIDMR as opposed to the observed plateau.

It is too early to tell whether the plateau in CIDMR since 1986 reflects an important departure from the infectious disease mortality dynamics of the preceding century or merely represents a minor stochastic fluctuation without long-term significance.

We should consider the possibility that the most achievable reductions in CIDMR have been accomplished; future reductions



Note. Apparent artifacts at 1964–1968 and 1978–1979 boundaries have been removed. CIDMR = childhood infectious disease mortality rates.

FIGURE 2—Annual rate of change in childhood infectious disease mortality rates, smoothed with 3-period moving average.

may be more difficult and may require incrementally greater resources as well as new strategies. □

Contributors

J. H. DiLiberti did most of the writing and data analysis. C. R. Jackson participated extensively in manuscript revisions, conception of the study, and interpretation of the data.

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Preventable Inpatient Time: Adequacy of Electronic Patient Information Systems

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ABSTRACT

Objectives. This study assessed hospital electronic patient information systems (EPIS) for inclusion of variables associated with avoidable and extended hospitalization (preventable inpatient time).

Methods. We searched MEDLINE and HealthSTAR databases to identify predictors of preventable inpatient time. We then audited the admissions process and the handwritten medical record at 1 hospital, and the EPIS at all hospitals, affiliated with the Yale University School of Medicine for inclusion of the predictors.

Results. Whereas the written medical record included all 58 predictors, the EPIS of the 10 hospitals surveyed included an average of only 38% of the predictors.

Conclusions. The conventional approach to information gathering during hospital admission is highly inefficient. Revising EPIS to include predictors of preventable inpatient time could enhance efficiency and quality, while reducing costs, of hospital care. (*Am J Public Health*. 1999;89:1885–1889)

Avoidable hospitalizations and extended hospital stays (collectively referred to as preventable inpatient time) confer a substantial economic and human cost.[1–4] Hospitalization is a major risk for elderly patients and is often followed by irreversible functional decline.[2] Outpatient primary care is more cost-effective than inpatient care[5]; delays in seeking such care result in longer hospital stays and higher mortality.[6]

Medicare's adoption in 1983 of diagnosis-related groups for prospective capitation of payment increased hospitals' financial incentive to limit lengths of stay,[7] particularly among Medicare patients. Numerous studies conducted since 1983 have reported factors associated with preventable inpatient time.[8–21] Hospitals routinely obtain information related to these factors, whether by questionnaire, personal interview, or examination of the preexisting medical record. This process, however, is inherently inefficient. Some critical data may not be obtained, while some may be obtained redundantly in the multiple interviews of a single patient. Virtually all of the information in the handwritten chart is difficult to retrieve after the patient's discharge.

Although the handwritten record is useful in managing the medical care and hospital course of a patient prospectively, it is of limited use for assessing patterns in patient populations retrospectively. Electronic patient information systems (EPIS), commonly used to obtain demographic data on hospital registration, readily allow access to data for retrospective patient population studies. To assess the adequacy of these systems, we compared the electronic information system at 1 teaching community hospital with the written record for the inclusion of predictors of preventable inpatient time.

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