Class and Environment in "Fatal Years"

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McKeown and Record's classification of the causes of the nineteenth-century mortality decline has proved a fruitful one and forms a useful framework for considering the distinctiveness of the demographic findings of Preston and Haines. McKeown and Record detected four main controls on mortality: medical science, the standard of living, the environment, and the virulence of disease organisms. They argued that changes in the mortality from particular causes of death could be assigned to the operation of one or other of these main controls. For England and Wales, they argued that medical science had little to offer in fighting any of the main diseases that actually waned, except for smallpox, which was attacked through vaccination. Thus the small contribution of smallpox to the mortality decline registered the slight contribution that the medical profession had made to improving individuals' life chances during the second half of the nineteenth century.

Believing that diet (and thus the standard of living) controlled the level of mortality from tuberculosis, they proposed that the large contribution of the decline in tuberculosis mortality to the overall mortality decline was clear evidence that the main contribution to English and Welsh longevity came from a general rise in real incomes. The intermedi-

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ary position of the diarrheal diseases placed environmental changes (the sanitary revolution) above medicine but below economic growth. Finally, a small residual contribution from some childhood diseases whose decline could be explained in no other way attested the tiny contribution made by autonomous changes in the virulence of disease organisms.

This framework continues to shape discussion about the causes of mortality decline. It has, however, been criticized on both methodological and conceptual grounds. Problems of measurement, aggregation, and interpretation have been identified. Can the independent operation of these major controls on mortality really be measured by the relative contributions that drops in mortality from tuberculosis, diarrhea, and smallpox made to decreases in the standardized mortality rates? Doesn’t the national picture mask a set of important differences along, for example, rural/urban lines? Shouldn’t historians be looking for interactions between the factors controlling mortality—interactions that might render invalid the isolation of factors and their unique identification with particular causes of death? Is it really acceptable to exclude all reference to behavioral factors, such as health care practices, from the analysis?

Fatal Years on the Factors Controlling Mortality

Fatal Years addresses these big questions about the fundamental causes of mortality decline. Preston and Haines, however, approach the issue somewhat differently than McKeown and Record do. On the one hand, Preston and Haines do not have information on cause of death and thus cannot identify the operation of the main controls on mortality change in precisely the way that McKeown and Record claimed to be able to do. On the other hand, they do have a wealth of material on the individual circumstances of the children they study. They have thus been able to explore the interaction of a wide range of factors in explaining cross-sectional (and to a more limited extent, temporal) variations in child mortality. Furthermore, their focus on child mortality highlights a set of cultural variables which McKeown and Record ignored altogether. Following both historical studies and those conducted in today’s poor countries, Preston and Haines place breast-feeding practices and the literacy

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of the mother on the agenda alongside medicine, living standards, and the environment. These are important advantages.

For the late nineteenth-century United States, Preston and Haines echo McKeown and Record's reservations about the minor role played by medical science. Their grounds are rather different, however. It is not the absence of specific drugs for treating particular diseases to which they draw attention, but rather, the ignorance of germ theory's basic lessons with regard to domestic cleanliness and urban sanitation: "The ignorance to which we refer is some combination of ignorance about personal hygiene and ignorance about what public institutions could accomplish in the area of health." Their evidence for this conclusion is the lack of relationship (when other factors have been accounted for in the regression model) between the mother's literacy and child mortality and between the father's occupation and child mortality. Even the children of physicians were subject to a rate of mortality only 6 percent below the national average (p. 189). The higher mortality of children in urban areas "is clear evidence that political institutions were far from realising their potential for improving health conditions" (p. 207). After 1900, according to Preston and Haines, medical knowledge and/or techniques became more effective in reducing mortality:

Our subsequent research suggests that improved preventative measures were the principal forces of change in the United States between 1900 and 1930. The most important of these were probably measures taken by parents in the home, taking advantage of the new knowledge that disease was spread interpersonally by invisible micro-organisms.

It is this ignorance of basic domestic hygiene which explains why in the late nineteenth-century United States the literacy of the mother failed to offer the protection that it is usually observed to provide in poor countries today.

With regard to living standards, Preston and Haines again give qualified support to McKeown and Record. Although they draw attention to the lack of relationship (after other economic variables have been considered) between the father's occupation and child mortality, and although they believe that "lack of know-how rather than lack of resources


was primarily responsible for foreshortening life in the United States in the 1890s" (p. 209), they also find a series of economic variables to have been important in explaining variations in child mortality. Security of employment had an effect: “For the native population, and in both rural and urban areas, a period of unemployment by the principal income earner in the family raised the child mortality index by 12–22 percent” (p. 168). Another significant factor was the presence of boarders in the household, which, among other things, would be “an indicator of economic distress” (p. 168). The condition of the local labor market (measured by state income levels) appears to have been important, at least among native-born populations in rural areas (p. 169). Preston and Haines find a very significant difference between the levels of child mortality in black populations and comparable levels in white populations, and conclude that “the large mortality variation by race that we have demonstrated is most plausibly ascribed to the enormous economic disparities that existed between the races at the time” (p. 175). It is the importance of the cultural or behavioral correlates of class that are called into question by an analysis that finds the father’s occupational label to be relatively unimportant once a set of more tightly defined economic variables have been considered. However, the variations in child mortality accounted for by economic circumstances were about a mean that was relatively high in historical terms. By the standards of the poor countries of today, the United States in 1900 was a relatively wealthy country, yet it had a much higher level of child mortality. In this respect, Preston and Haines place medical knowledge above living standards in explaining child mortality.

The importance of the environment is clearly established by the important geographic variations they find in mortality. Other things being equal, it made a great deal of difference what part of the United States people lived in. For example, the rural areas of the “Mountain region” and the “New England region” had mortalities about twice that of the rural South Atlantic region (p. 167). In addition, for all groups, residence in cities with a population of twenty-five thousand or more meant a mortality between 20 and 36 percent higher than that seen in towns with populations of between one thousand and five thousand (p. 168). With the mother’s age considered as a proxy for the date of birth of the child, they also conclude that child mortality was declining quickly in late nineteenth-century America, and that this decline was most rapid in urban rather than rural areas, and among the children of foreign-born mothers (p. 166). In other words, the sanitary revolution was reaching some of the very poorest groups in society.

Turning, finally, to the behavioral factors they introduce into the
analysis alongside the controls already considered by McKeown and Record, they reach negative conclusions:

The variables that do not appear to be very important in child mortality, individually or as a group, are those which we expect to be most closely associated with child-care practices: mother's literacy, her ethnicity, her English-speaking ability, and her husband's occupation. None of these variables significantly explains variation at a 5 percent level . . . once other variables are controlled. Whatever behavioral variation was associated with these variables seems to be swamped in its effects by broad geographic and economic factors. (p. 175)

Demographic historians will be loath to concede this point. It is clear that to some extent the emphasis on the quality of mothering as a crucial control on child mortality in the late nineteenth century comes from a contemporary horror of the women who worked outside the home and thereby appeared to go against the prevailing ideology of domesticity. Preston and Haines comment that although relatively few women worked outside the home after giving birth, the Children's Bureau was obsessed with this phenomenon: "The concentration on women's work as an influence on child health by contemporaries probably says more about social expectations regarding parenthood and the family than it does about major factors in mortality" (p. 41). The European evidence points to marked differences in infant mortality—often within the same country—between areas with relatively high rates of breast-feeding and those with relatively low rates. These differences may have been based on religion or may have been imposed by variations in the nature of women's agricultural work. Perhaps immigrants adjusted their breast-feeding practices to the changed labor market conditions of the United States (and the availability of artificial foodstuffs there) to an extent that eliminated the geographic variations of their homelands. However, country of birth may be too crude a variable to discriminate clearly between early-weaning and late-weaning mothers, and thus these behavioral factors may not be detectable in this data set.

The general picture that emerged from McKeown and Record's analysis was of a population gradually released from the mortality check of

poverty. Preston and Haines show us a population buffeted by the external pressures of labor market and urban environment. In the former study, medical science was allowed no part in the improvements that occurred. In the latter, ignorance of medical technique separates a past of high child mortality from a present of much lower mortality. In neither case do the people of the nineteenth century emerge as having had much control over their life chances: "In place of a sharp differentiation now commonly associated with behavioral differences among classes were important variations in mortality according to factors over which individuals had little or no control" (p. 209).

The evaluation of the findings from Fatal Years will occupy demographic and medical historians for some time to come. At least three sets of considerations are important. First, the specification of the central variables in the study and implications drawn from the findings will come under scrutiny. Second, comparison with the experience in other places could highlight the distinctiveness of the situation of the United States. Finally, historians might examine the importance of aspects of mortality to which Preston and Haines are unable to give specific attention. There are three dimensions of the great mortality decline on which Preston and Haines have little direct evidence: cause-specific patterns of mortality, mortality patterns after childhood, and intraurban variations in mortality.

The Urban Penalty: Effects of Environment and Class

For the nineteenth century we may speak of urban living as carrying a demographic penalty exacted through relatively high mortality. Preston and Haines place environment second only to race in accounting for patterns of child mortality. In studying how cities levied this mortality penalty, the three dimensions unavailable to Preston and Haines may prove instructive.

It would appear that by the late nineteenth century many of the largest European cities were probably healthier than cities of the second rank. This may be testimony both to their lead in sanitary measures and to their earlier decentralization through suburbanization. In the 1890s, life expectancy at birth was 46.3 years in London but only 41.9 years in the other cities that had passed a population of fifty thousand by mid-century.6 In 1886–98, Paris had a crude annual death rate of 21.0 per

thousand living inhabitants, whereas the other French cities with more
than one hundred thousand inhabitants had a rate of 24.2 per thou-
sand. This difference was most marked in infant mortality (135.5 infant
deaths per thousand births, compared to 187.4), showing up in deaths
from diarrheal diseases and pneumonia (which might be thought of as
filth diseases in this context), but not showing up in respiratory tubercu-
losis (which might perhaps be viewed as a disease of overcrowding at
this time). Although it is beset by interpretative problems, the study of
patterns of cause-specific mortality may throw further light on the timing
of the impact of the sanitary revolution in different countries and in
different types of cities.

In considering the operation of the urban penalty across the life cycle,
four sets of considerations have an important bearing on how conclu-
sions from a study of child mortality may be set in a broader context.

First, it is clear that different causes of death bore most heavily on
different age groups. A study of patterns of mortality in agricultural and
urban districts of England and Wales in the 1850s and 1890s shows that
infectious diseases such as scarlet fever went a long way toward explain-
ing the fact that child mortality was higher in the city than in the
country. For adults, respiratory tuberculosis played much the same role.
The same difference emerged when mortality improvements over the
second half of the nineteenth century were considered.

Second, over the life cycle there was clearly a set of complicated
relationships between exposure and immunity which operated rather
differently in urban areas than in rural ones. The timing of bursts of
migration and urbanization will have stirred up disease pools at particu-
lar points in time, and some of the changes in the lethality of various
diseases at different ages may reflect long-term cycles of exposure and
accommodation occurring over the second half of the nineteenth cen-
tury. Therefore, a cohort effect combined with the increasing frequency
of childhood epidemics in larger places will modify the age-specific
patterns of disease mortality quite independently of any sanitary im-
provements.

7. Gerry Kearns, "Zivilis or Hygaeia: Urban Public Health and the Epidemiologic
8. Kearns, "Mortality Decline" (n. 6).
56: 559-82; Gretchen Condran and Rose A. Cheney, "Mortality Trends in Philadelphia:
Third, it is important to consider whether there may be thresholds in the effect of the environment on the human organism at different ages. In other words, if the resilience of people to a given level of environmental challenge varies with age, then a gradual improvement in sanitary conditions will actually show up as a series of falls in age-specific mortalities at different dates as the threshold for ever more vulnerable groups is surpassed. For England and Wales, at least, the mortality decline of the second half of the nineteenth century displayed just this sort of pattern. Adolescents and young adults show a fall in mortality beginning in 1860s; and infants, children, and the elderly do not join them until the end of the century. Whereas the gradual rippling of mortality improvement up the age pyramid may be evidence of a healthier cohort passing through the life cycle, the contemporaneous migration of improvement down the pyramid may attest to something rather like this threshold effect. A study based on the most vulnerable age groups would, therefore, perhaps present the most pessimistic picture of the severity of the urban penalty.

Fourth, we need to look at the likelihood that the urban penalty operated through insult accumulation: in other words, it was their continual exposure to the city which progressively broke adults down. Work on the Swedish lumbering town of Sundsvall for the period 1860–92 suggests that new migrants had lower mortality than permanent residents at all ages, and that this gap was widest for children and young adults. Staying in the city seems to have been bad for people’s health. The ratio of urban mortality to rural mortality is widest for the very young and for mature adults in England and Wales during the second half of the nineteenth century. Whereas the children’s mortality might reflect the prevalence of specific infectious diseases in the city, the progressive widening of the gap over the age range twenty to fifty is consistent with this idea of insult accumulation (although it is also consistent with the importance of infectious diseases of relatively long latency, such as respiratory tuberculosis). It is also clear that this penalty fell most heavily upon males, perhaps registering their experiences at work and perhaps

10. These results come from work with colleagues in Sweden, both at Umeå (Anders Brändström, Sören Edvinsson, Göran Broström, Bengt Frank, Carin Sjöstrom) and Uppsala (John Rogers).

11. This was pointed out to me by Samuel Preston at the Louisiana meeting of the American Association for the History of Medicine, May 1993. The mortality data on the relative severity of the urban penalty on men and women of different ages is presented in Gerry Kearns, “Biology, Class, and the Urban Penalty,” in Urbanising Britain: Essays on Class and Community in the Nineteenth Century, ed. Gerry Kearns and Charles Withers (Cambridge: Cambridge University Press, 1991), pp. 12-30, esp. p. 17.
also reflecting the role of lodging houses as hothouses of infectious disease transmission. It is certainly the case, both in Stockholm and Paris, that young single adult males had rates of mortality somewhat above those of their single sisters.

Finally, in considering how the urban environment levied its mortality toll, some consideration needs to be given to the implications of mortality patterns within cities. Suburbanization was a class-specific transformation in the quality of the urban environment. It was moreover, an environmental improvement that was urged on the middle class in terms of the requirements of health (as well as other desiderata such as public order). It did not wait upon the widespread acceptance of germ theory and was equally imperative within a miasmatic perspective.

This issue has further implications for Preston and Haines’s treatment of class. Finding that the cultural residue left in their use of the father’s occupation explains little of the variations in child mortality once more narrowly economic factors have been accounted for, they then proceed to compare this result with contemporaneous data for England and Wales. They discover that “urban location exacted about twice as high a penalty in England as in the United States” (p. 194) and that class had an independent effect on child mortality. They account for this social differentiation by the more complete spatial segregation of the classes in British cities than in American ones (p. 197).

This argument is persuasive. There is a limited amount of evidence to suggest that the dynamics of the spatial segregation of the classes was reflected in the evolution of class-specific differentials in mortality, with the earliest gap opening up between the urban gentry (merchants and professionals) and the rest of urban society, and the lower middle class later joining the gentry in suburban safety. For example, in London during the cholera epidemic of 1848, “tradesmen” and “mechanics” had similar rates of mortality, whereas the “gentry” suffered less mortality than this. In 1871, the class gap in mortality appears to have been wider in the great cities of England and Wales than in the countryside. At this date, white-collar workers (primarily clerks) actually had higher mortality rates than the skilled working class, although the gap narrowed with age (perhaps indicating the cumulative effect of the world of manual work). By 1900–1902, the gap between the two had closed, although it

was still clear that clerks were peculiarly subject to respiratory tuberculosis. The most significant mortality gains over the second half of the nineteenth century were made by these white-collar workers, whereas the least marked improvement fell to the general laborers. At mid-century the mortality gap was between the gentry/professional on one hand and the lower-middle and working classes on the other. At the close of the century the largest gap was between the upper- and lower-middle classes on one hand and the working class on the other. This shift is consistent with changing patterns of residential segregation and may provide some evidence for the importance of the later suburbanization of the English petty bourgeoisie compared to the gentry. It is certainly consistent with the explanation offered by Preston and Haines.

Yet Preston and Haines resist the conclusion that the widening class gap in child mortality in the United States over the period 1900–1930 might be due to subsequent suburbanization. The finer spatial scale of Higgs and Booth’s analysis of the effect of density variations within cities on mortality reveals correlations that are suggestive of a suburbanization effect as early as 1890, and Meckel finds an association between the decentralization of population within Boston and falling rates of mortality. It is not clear why the class effect observable after 1900 should be ascribed to class-specific familiarity with germ theory, rather than to class-specific patterns of suburbanization.

Conclusion

The introduction of a set of questions requiring information on mortality variations by cause, age, and intracity location suggests some of the ways we might try to specify more clearly the mechanisms whereby cities levy the mortality penalty that Preston and Haines so clearly demonstrate in the case of child mortality. They have convincingly shown the importance of the constraints of labor market and environment on child mortality. By contrast with the influential study of McKeown and Record, they have moved beyond aggregate studies to the examination of data on individuals, allowing Fatal Years to explore the interaction of many different variables. Future studies on the mortality decline of the nineteenth

14. Kearns, "Biology" (n. 11), p. 16.
century in the United States will have to offer explanations that are broadly consistent with the main outlines offered in this work. It is difficult to resist Preston and Haines's claim that the new data on which their book is based “converts the United States from the industrialized country with the poorest mortality data at the turn of the century to the country with perhaps the richest and most detailed data on infants and children” (p. xvi).