Health Risks Related to Family Formation in Nineteenth Century Western Massachusetts

R. Meindl
Kent State University

H. Temkin-Greener
University of Massachusetts - Amherst

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Evidence from both developing and developed countries indicate strong relationships between infant mortality and birth order, birth spacing, and maternal age. We have examined the mechanisms of family formation and fertility decline by means of reconstituted family data from a historical New England community. Aspects of mortality risks of a population which lacked modern medicine and community-organized public health measures are presented. Implications for late 19th century mortality transitions in rural America are discussed.
The second half of the 19th century saw considerable advancements in the longevity of most Americans. Crude death rates for most of this population were reduced by a third, and the average expectation of life at birth improved steadily through the turn of the century (Meeker 1972). Similarly, the cause-of-death structure in the United States assumed a new configuration. The infectious diseases, which prior to our century caused a sizable part of the mortality in all human populations, were partially replaced as major causes of death by the chronic diseases of old age. Regardless of the mechanisms involved, the "vital revolution" in mortality in urban and even rural America (Higgs 1973) came about primarily as a result of the control of infectious pathogens. The speed and pattern of change from 1850 to about 1915 was apparently without precedent.

To date, the explanations which have been advanced apply largely to the populations of urban centers, a portion of the nation's people. Arguments tend to focus on medical advancements, public sanitation, social reform and technological innovation, as well as improvements in housing, diet and hygiene. But it is the relative importance of each factor in the decline of morbidity and mortality over the previous two centuries that constitutes what Thomas McKeown has called the most important question in medical history (see McKeown & Brown 1955). Therefore the study of the transition in the late 19th century requires analyses that go beyond a mere comparison of summary vital rates. In addition, such studies might begin with rural communities in which the reasons for the great decline are less obvious.

There is little doubt that recent refinements in the medical sciences have resulted in a great saving of human lives and in general improvement in health. Yet before 1900 the effect of the biomedical professions on the average individual's well-being may have been small, as suggested by studies in England and Wales (McKeown & Lowe 1966). Rather it was government action and public attitude which partly improved health, not by the treatment of the individual patient, but by prompting deliberate sanitation policies among urban populations during the late 19th century. However, rural America never fully experienced the community-organized sanitation measures that caused the tremendous decline in mortality in American cities. Yet Robert Higgs (1973) and others have shown that low density regions paralleled the declines in urban centers in an equally dramatic way, based on estimates of some simple mortality statistics. Since the analyses which were operationalized from historical data have provided little in terms of explanation, Higgs and other economic historians have attributed rural health improvements almost exclusively to higher standards of living. Before it is concluded that mortality is exclusively determined by income, the portion of which devoted to health-related purchases will always remain unknown, another look should be given to demographic determinants.

Structural features of populations influence vital rates as strongly as do environmental factors. For example, demographers have
recently argued that childhood mortality has had a great effect on subsequent fertility levels, at least within those families which practiced some form of fertility control. Conversely, fertility levels themselves can exert great influence on health within families, a relationship which has not received its proper emphasis. We have found that such mortality determinants can be isolated from reconstituted family data, and that they may be quite important in the overview of 19th century medical history in rural America.

Patterns of fertility, family formation, and household composition are important variables which greatly influence the health status of a population. This seems to be the case in many developing areas in the world today, such as the Near East, India, and the Phillipines as observed by the World Health Organization (Omran and Standley 1976). We will emphasize the relationships of these factors to child mortality, which, by constituting the majority of "preventable" deaths, shows the highest amount of variability among human groups. This variability is evident in contemporary, historic, and prehistoric populations.

Our purpose in this analysis is twofold: first, we explain a portion of the variation in crude mortality statistics such as death rates and expectation of life at birth largely in terms of associated fertility levels and demographic structure, and second, we will apply these inferences to the final dramatic health improvements in a small portion of rural America previous to the advent of modern medical and public health care.

Materials and Methods

The fact that Massachusetts was one of the ten original registration states, the predominantly rural nature of Franklin County, and the existence of an excellent community genealogy make Deerfield generally suitable to the first purpose - the detection of fertility-influenced health levels. Deerfield families were reconstructed on the basis of fertility histories as revealed by the vital register of the town. The federal censuses served as an independent check. Sheldon's published genealogy was most useful in strengthening our observations of periods before the 1840 registration laws; it was rarely needed for the periods after this time.

About 700 Deerfield women ranging in year of birth from 1810 to 1860 formed the core of the sample. About 2/3 of these families were in full observation, i.e., from the birth of the women to the death or survival to adulthood of her last child. The children themselves were traced only to their 21st birthday, unless of course they later began their own families in town.

Our collection does not constitute a random sample of Deerfield residents, but we believe it to be quite representative of those individuals who remained in the community throughout their reproductive history. This defense of the sampling procedure is probably most
Figure 1. Birth order in Deerfield.
appropriate for what is by far the largest subclass in the file—native New Englanders who farmed their own land.

Results

During the time in question, Deerfield ranged in size from 2200 to 3600 inhabitants. It largely retained its agricultural character although several neighboring regions became somewhat industrialized. Except for a few Irish and German residents, the surnames remained predominantly English until just before the turn of the century when Polish-speaking immigrants began to acquire some of the farms. Their influence on the demographic characteristics of this part of the Valley was limited to only one neighboring community until the turn of the century.

Characteristics generally associated with high fertility populations include early age at marriage and brief interpregnancy intervals as well as a predominance of high order births and large mean completed families. It can be shown that such variables are correlated with family health levels, particularly early childhood mortality risks. This is illustrated by comparisons of adolescent cohort life tables selected from the Deerfield family data.

First, children were selected on the basis of birth order, regardless of the year of birth (Figure 1). 1700 children, observed from birth until young adulthood provide 5 adolescent life tables. We report only infant mortality. There was a small but regular increase in first year mortality as we move from 1st children through 2nd, 3rd, 4th, and finally 5th and higher-ordered births. Members of the last cohort have at least 4 children ahead of them, should these still be alive during this single year of observation. Our family file is not yet large enough for us to present comparisons independent of time. But we present the temporal composition of these cohorts by treating the year of birth as an "attribute" of each child. The range of the 5 mean years of birth is only 9; all 5 standard deviations are identical at 15. No birth was included of mothers over 38 or under 16 years of age. Thus we think that the gradual increase in risk to the infant is not only a function of maternal age. The risk might also be directly related to the number of older children in the family, and the size of the household itself, and so forth. In addition, the economic and maternal physiological resources expended on each child are probably parity-dependent.

In the next comparison (Figure 2), children are selected on the basis of the preceding birth interval. The bottom figures, 15-18 months, 19-22, and so on also refer then to the age of the previous sibling at the time of this birth. Obviously, no 1st-order births can be included here. The frequent omission of month of nativity in some families further reduces this sample to about 600 children. Again, this refers only to mortality risks during the first twelve months. Mortality risk for the 15-18 month interval children is almost twice the average of those of the next 25 months. This is similar to a disease-incidence study of Cleveland children during the 1950's. However, mortality in the recent clinical survey was,
Figure 2. Birth interval in Deerfield.
Figure 3. General fertility rates for Shelburne and Deerfield.
Figure 4. Enteritis in Shelburne and Deerfield.
of course, much reduced (Dingle and others 1964). I should point out that overmortality is generally male mortality. 60% of the high-risk infants who died were male. The figures for all other cohorts mentioned were only slightly over half. Our observation that these cohorts are not greatly biased by any particular portion of the century is an important methodological point that will be emphasized later. While the births span the better part of a century, the range in mean birth year of all 4 cohorts is only 2 years.

These data from the Deerfield community hint at some interesting associations between high infant mortality and some of the variables commonly associated with high fertility. But none of these are strongly associated with time - aggregate levels declined very slowly over the 19th century. Thus we have observed more demographic variation in fertility and childhood mortality within any decade than we see across decades (or, over time). In other words, Deerfield has been very useful in illustrating structural relationships in family formation, but particular social and environmental conditions preclude a direct application using this town to the more common historical health improvements we have mentioned. For this purpose, we turn briefly to conditions in a neighboring community.

By contrast, Shelburne, Massachusetts experienced a rapid decrease in birth rates some years after the registration improvements of the 1840's. The general fertility rate in Shelburne declined by almost 2/3 of its mid-century level. We offer no explanation for this and treat it for our purposes as an independent variable. The associated health improvements in Shelburne were impressive.

General fertility rates for each community over time are presented in terms of 3-year averages every ten years beginning 1855 (Figure 3). A fertility decline is evident in both towns but the changes in Shelburne are far more pronounced.

Enteric diseases were a major cause of death in young children in the Middle Connecticut Valley (Figure 4). This is a category made up of those entries entitled "dysentery" and "cholera infantum." The second one bears no relationship to the Asiatic cholera; rather it is probably similar to the diarrheal diseases common to urban centers. Any illusion of regularity in these age-disease specific rates is an artifact of 5-year running averages. Even after smoothing, the incidence in Deerfield is erratic, whereas the decline in Shelburne is more apparent.

Tuberculosis was far more prevalent in the total population, and declined fairly rapidly in the 60 years shown here (Figure 5). "Consumption" was a disease rich in visible symptoms, but whether relatives preferred to accurately report this cause of death is another question. The rates are probably too low for this reason. This was a disease of young adults, especially women in their reproductive years. A number of consumptive deaths to women occur directly after childbirth. Also, the incidence of this disease might be closely tied with the opportunity for
Figure 5. Tuberculosis per 10,000 in Shelburne and Deerfield.
Figure 6. Child mortality in Shelburne and Deerfield.
Figure 7. Survivorship in Shelburne and Deerfield.
infection for all household members. Dr. Whitlock of Cornell has suggested that the mysterious 19th century decline in tuberculosis might be linked with reduction in average household size. This is affected not only by lowered fertility but also by the break-up of the extended family.

However, concentrating again on early mortality by all causes we find that the net result is a real decline in the force of mortality in childhood (Figure 6). These are running life table death rates for children only. The Shelburne rates decline by 60% in 60 years; Deerfield which maintains high fertility declines by only 30%. This last figure presents time-specific or transverse survivorships (Figure 7). Since these are small populations each mortality profile is an average for a decade. The figure presents only survivorships, but from the life tables we see that expectation of life at birth increased by almost 12 years in Shelburne; by 2 1/2 in Deerfield. The crude death rate in Shelburne dropped from 21 to 15.

Our point is simply this: the important differences among these profiles are brought about almost solely by changes in mortality under 5. The only reliable inference we can make therefore has to do with the close correspondence between child mortality and overall fertility levels. These were first detected in family analyses of Deerfield (fairly independent of time) and confirmed in another community within its secular context. Also, if any overall relationship exists between infant and adult mortality, it is inverse. Expectation of life at age 40 is actually diminishing somewhat in Shelburne. While limited data reduce the strength of our inferences about the higher ages, there is no support here for a standard of living argument in its usual sense.

Discussion and Conclusion

The greater proportion of infants and neonates which experience the highest mortality risks of any age class can easily inflate a crude death rate. But more important, lowered birth rates can result in real changes in childhood mortality resulting in actual changes of \( e_0 \) for a given individual. How rapid and high overall parity affects infant health risks is not clear. It may be related to several conditions: 1) the opportunity for infection in children increases with the size of the family, 2) the physiological vitality of the mother and even the resources of the family itself can be greatly diminished, 3) our comparisons are still not fully independent of maternal age, and finally, 4) our analysis is at the mercy of observational selection in that occupation, nativity, and probably wealth vary among the cohorts to some degree.

The following inferences, presented from weakest to strongest, concerning the "vital revolution" can be drawn from the study. First, we do not find support for the traditional argument that improved standard of living was the primary condition resulting in decreasing mortality. Second, considerable evidence suggests that changes in mortality were strongly affected by changes in fertility. The Deerfield analysis indicates the link between increased infant mortality
and two characteristics of high fertility populations: the predominance of high-order births and brief interpregnancy intervals. The comparative analyses at Shelburne and Deerfield already suggest that mortality change, particularly that due to tuberculosis and enteritis, was related to fertility change. While specific mechanisms relating high fertility with high mortality were suggested, e.g., high probability of infection from siblings, low time per parent per child, fewer resources per child those remain hypothetical.

Further research will concentrate on illuminating these mechanisms. Also, the conditions underlying the fertility decline need attention. Directing attention to these issues is a major conclusion of our work. Explanations for the "vital revolution" will not be found with simplistic lines of reasoning relating mortality change with non-demographic change. This crucial demographic event will be best understood with sophisticated consideration of the impact of modernization on all aspects of the population. Clearly, fertility change, and the concomittant changes in family size, family structure, interpregnancy intervals and vitality of the mother, was crucial to mortality change in the rural nineteenth century population of the Connecticut River Valley.
REFERENCES CITED

Dingle, J.H., G.F. Badger and W.S. Jordan

Higgs, R.

McKeown, T. and R.G. Brown

McKeown, T. and C.R. Lowe

Meeker, E.

Omran, A.R. and C.C. Standley (eds.)

Sheldon, G.

Whitlock, J.H.