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Forum: The Epidemiological Transition

One, many or none?

Bruce Fetter

Department of History, University of Wisconsin, Milwaukee

Demographic transition theory has given birth to a number of variants whose terms are contested but in which many researchers nonetheless have a stake. A case in point is that of C.J.L. Murray and A.D. Lopez who have, under the auspices of the World Bank and the World Health Organization, just produced a ten-volume series on The Global Burden of Disease [and Injury]. After lamenting the lack of comparable and commensurate runs of health statistics, they go on to assume that 'most developed and a number of developing countries have undergone demographic and "epidemiologic transitions" to lower fertility and mortality rates during this century' (Murray and Lopez 1996:740-741).

Increasingly scholars disagree about the nature of these transitions. Dalla Zuanna (1996:216) in reporting on the 1992 Chaire Quetelet observes that 'we are not dealing with a single demographic transition, but rather transitions which occurred differently according to time and place'. Let us look more closely at the epidemiologic transition, that transformation in the cause of death from infectious to chronic and degenerative diseases. Despite scholarly investigations into the causes of that shift—and the undisputed contributions of this journal to the cultural, social and behavioural determinants of health—many questions remain. Here are three of them.

1. What are the immunological preconditions for an epidemiologic transition? That is, are the pathogens which kill humans sufficiently limited in number so that a common set of medical preventive and curative procedures could have lowered mortality? Beyond the number and nature of pathogens, one must also consider human resistance to them. Recent work suggests that different human populations have very different resistance to infectious diseases. These conclusions are based on the study of Class I and II histocompatibility antigens, which control the immune system and which reflect a human population's experience with infectious diseases and that population's ability to resist them. Francis Black (1994) reports that Old World populations (from Europe, Asia, and Africa) have at least 35 histocompatibility variants while New World populations (from the Americas, Australia, and the South Pacific) have no more than 17. He therefore argues 'that people of the New World are unusually susceptible to diseases of the Old...'. Given the diversity in the capacity of various human (and animal) populations to resist infectious disease (Parham and Ohta 1996), one might well ask if a single set of medical and sanitary techniques would lower mortality among all populations in the same way.

2. Can we treat medical interventions, even those focused on a single pathogen, as a single independent variable? Let us consider the case of Jennerian vaccination against

smallpox. Nearly a century separates Jenner's discovery of the efficacy against smallpox of exposure to cowpox and the establishment of compulsory vaccination in England. Some of the delay can be attributed to medical imperfections in the procedure: the danger of infection with other diseases through arm-to-arm vaccination; ignorance of the need for periodic revaccination in order to maintain immunity (Hopkins 1983; Skold 1996). Other delays were due to cultural and social resistance and the question of who should pay for the procedure (MacLeod 1967). While these medical, social, and economic obstacles were being overcome, the path to smallpox eradication was by no means direct. Is it not better to see mortality reduction from infectious disease as resulting from the accumulation of the impact of a number of initiatives of varying efficacy rather than as part of a single global phenomenon?

3. How do societies pay for medical care and advances in public health? The changing nature of the financing of medical procedures and public works linked to mortality reduction has been neglected in the transition literature. Variants include fee for service; voluntary organizations, both national and international; and governmental activities, local, national, and international.

Fee for service arrangements are not limited to the United States. Many, if not most, non-allopathic services are provided by healers who are reimbursed by the sick and their families rather than by national health services or insurers. This was particularly true in colonial regimes, where governments actively discouraged non-allopathic healers (Fetter 1996).

Voluntary organizations, particularly religious groups, provided a large proportion of medical services during the nineteenth century. In Western Europe and North America, these included both Protestant and Catholic religious orders which filled the needs not met by professional medicine. These religious efforts were supplemented by single-purpose secular organizations designed to relieve victims of famine, war, or particular illnesses.

Finally, one needs to sort out the role played in health care by various levels of government. Roger Lee (1988) argues that the local governments' role in British health care reached its zenith between the first and second world wars. By the same token, Catherine Rollet (1992) finds that French rural departments played an important role in child health during the Third Republic. Although local and even provincial governments have declined in importance, international agencies and foreign aid have come to pay for a substantial proportion of health costs in many of the world's poorest countries. The prospect that such funds will continue at current levels is doubtful indeed. In predicting future trends, one might well want to examine the health systems of countries such as China and South Africa, which have developed without substantial external support.

In summary, several objections to transition theory in its current form need to be considered. Thanks to *Health Transition Review*, contributors to this forum can enter a lively debate.

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Epidemiology and the demographic transition in the New World

Philip R. P. Coelho

Department of Economics, Ball State University, Muncie IN 47306 USA

Bruce Fetter (1997) has raised an interesting issue: what does it mean to say that a society has undergone an epidemiological or demographic transition? When we speak of societies are we speaking of some people who are genetically close, such as Icelanders, or are we speaking of people who have similar skin pigmentation, or the people who inhabit a geographic or political region? To answer these questions, I believe it is useful to examine a historical episode; to do this I turn to the history of the post-contact New World.

The demographic transition in the New World was literally a change in populations. Subsequent to the Old World's 'discovery' of the New World, the aboriginal peoples of the New World died and their ecological and economic niches were taken by their Old World counterparts. The reasons that Native Americans died and the Old Worlders did not were the differential epidemiological responses that Old and New World human populations had to the evolving disease ecologies of the New World. And, in turn, the differential responses of the disparate populations and the evolving ecologies were a function of their ancestral disease environments and the interchange of biological organisms between the hemispheres. So the demographic and epidemiological transitions in the history of the Western Hemisphere refer to a complex series of events that relates the biological sciences, demography and the social sciences.

The human immune system can be thought of as consisting of two parts: an innate resistance to disease and an acquired resistance to disease. Innate resistance is inherited from the parental gene pool. The chromosomal position of part of the immune system, the major histocompatibility complex antigens (divided into Class I and Class II MHC glycoproteins), has been identified (Black 1992; Engelhard 1994). Class I and II MHC glycoproteins are part of the body's defences against pathogens. If the pathogens survive the body's defences and are passed on to a genetically close individual, the varieties of the pathogen passed on have been preselected by the previous victim's immune system to combat the new victim's immune system. Consequently, the greater the diversity of MHC glycoproteins in a given population, the greater the resistance pathogens will encounter as they infect humans.

Individuals are estimated to have up to six varieties of MHC glycoproteins, but populations can have many more. Engelhard (1994:56) states that over one hundred forms of

MHC glycoproteins have been identified. In a study of ethnic groups, Black (1992) obtained the number of different types of Class I MHC glycoproteins from various sample populations. The sample of sub-Saharan Africans had 40 different forms of Class I MHC glycoproteins; the European sample had 37; the East Asian sample had 34; the North American Indian sample had 17; and the sample of South American Indians had 10 different forms.

The startling lack of genetic diversity in New World peoples meant that the Old World diseases introduced into the Americas had a devastating impact upon aboriginal Americans. Black (1992:1739) calculated that a pathogen passing between two individuals, each with six Class I MHC glycoproteins, chosen from a population of South American Indians (with ten different Class I MHC glycoproteins in the population) had a 32 per cent chance of just facing glycoproteins that it had already encountered in its first victim. This meant that in the South American sample the reproducing pathogens, which had survived the immune system defences long enough to infect someone else, had been evolutionarily selected to avoid many of their new victim's immune system responses. In contrast in a population of sub-Saharan Africans (with 40 different Class I MHC glycoproteins), a pathogen passing between two individuals (each with six Class I MHCs) had a 0.5 per cent chance of only encountering Class I MHC glycoproteins that it had encountered in its first victim.

The Americas, around 1500, were, for all practical purposes, a virgin soil for the diseases that were then prevalent in Africa and Europe. The native peoples of North and South America were descendants of wandering bands of Asiatic hunter-gatherers who had crossed the Bering land bridge to the North American continent during the ice ages. These bands were small groups of closely related peoples whose descendants migrated south into Central and South America. This explains the relative genetic homogeneity of the native peoples who inhabited the southernmost regions of the Western Hemisphere. The post-contact American Indian population was adversely affected by diseases because of the lack of genetic diversity and because of the variety of pathogens that were introduced. Aboriginal Americans, in general, had no exposures, hence no acquired immunities, to smallpox, chickenpox, measles, influenza, malaria, and a number of other diseases (Ramenofsky 1993; Kunitz 1993) that were brought to them post-contact. The voyages of discovery initiated a series of changes with far-reaching effects. The interchange of biological organisms, both human and non-human, between the Old and New Worlds altered the ecologies of both, and the subsequent epidemiological and demographic histories of the Americas.

With the decline in Indian populations the European conquerors sought additional supplies of labour to exploit New World resources. They sought labour in both Africa and Europe. Accompanying each group were the diseases endemic to their populations. The combination of 'European' diseases (tuberculosis, influenza, measles, and chickenpox), 'African' diseases (yellow fever, malaria, hookworm) and the ubiquitous smallpox destroyed all Indian societies that were not in remote places, or in the deep jungles or high mountain areas. The historical result is that with the exceptions noted, Meso-America, the Caribbean and South America are lands inhabited with peoples whose descent is primarily European or African. The remnants of the Aboriginal Americans have been assimilated into the prevailing Old World culture.

The introduction of African diseases into the tropics of the New World also affected the Europeans and Euro-Americans adversely. Conversely Africans and Afro-Americans were adversely affected by the introduction of 'European' diseases that became endemic to the temperate regions of the New World. People of European ancestry had few immunities, innate or acquired, to the disease pathogens prevalent in tropical West Africa which was the ancestral home of most of the Africans who were brought to the New World. Consequently in the tropical areas of the New World people of African descent lived longer, healthier lives

than those of European descent (Curtin 1968, 1989; Davies 1975; Crosby 1986; Steckel and Jensen 1986). Africans suffered similarly in the temperate regions of the New World (Klepp 1994; Coelho and McGuire 1997). The relative resistance of each ethnic group to the diseases that were endemic to their ancestral environments determined the ethnic composition of the various regions. Given the life expectancies of Europeans and Africans in the various climatic regions of the New World; interest rates; estimated regional morbidity in each ethnic group; and the prices of indentured servants (Europeans) and slaves (Africans), then simple financial calculations explain the regional distribution of Africans and Europeans in the New World (Coelho and McGuire 1997). Economic profitability ensured that Africans predominated in the tropical areas, Europeans in the more temperate areas of the New World.

Given enough time the native people of the Americas could have survived to become once again the dominant ethnic group of the Americas. But they were not given enough time. In pre-contact North America, agriculture was adopted later than in South and Meso-America. When the Old World diseases came (and the concomitant deaths of many native peoples) the resource mix available to the Indians changed. Fewer people meant less hunting, less hunting pressure meant the revival of populations of wild game and fish. Given the short (relative to humans) generational time among the North American animals, the increase in game must have been substantial. The native people reverted to relying more upon hunting and gathering, and less upon agriculture. Skeletal remains show a post-contact North American Indian population that increased its height (Larsen et al. 1997). This would reflect an enhanced protein intake due to the increased consumption of animal-based foods. There were fewer people, but they were taller.

The significance of this is that it indicates the dispersal of native peoples. The relative decline in the economic importance of agriculture meant that Indian societies were absolutely smaller and less settled than their pre-contact ancestors. A reduction in population density prevented disease pathogens from becoming endemic childhood diseases. Many diseases when contracted as a child are extremely mild, for example measles, chicken pox, yellow fever, and leave no long-lasting effects except that the body's immune system is primed for any subsequent re-infestation (acquired immunities); while these diseases if acquired as an adult can have a devastating impact including permanent disability and death. It was not just the peoples who reverted almost completely to a hunter-gatherer existence, such as the Sioux and Comanche, but even the Indians who remained agriculturalists were affected. The population of North American Indians was spread similar to a checker-board throughout the landscape. There would be clusters of permanent settlement, and then tracts of unsettled territories. This prevented the Indian population from obtaining the absolute numbers of people required in the various regions to make diseases endemic there. When the Old World diseases and peoples came the Indian population was differentially affected because of the lack of genetic diversity and innate immunities; and the lack of acquired immunities. The population of Indian peoples declined both absolutely and relatively until by 1890 the Census of the United States counted 1,235,000 Indians out of a total population of 62,947,000 (US Bureau of the Census1975:14), or a little under two per cent of the entire population of the United States.

What does this tell us about demographic and epidemiological transitions? The only generalization that comes from it is that no sweeping statement can explain this history. The details matter. The importation of Old World biological organisms, human and non-human, fundamentally altered the New World ecologies. These events had feedback effects that sometimes magnified the direction of change, at other times feedback effects ameliorated the direction of change. Peoples of different ethnic backgrounds were differentially affected depending upon the disease ecology that prevailed in the area of the New World they inhabited, and the evolutionary environment of their ancestors. As we examine each group,

'black' or 'white', more closely, we see that in the New World these groups themselves were heterogeneous; and each subgroup has its own epidemiological and demographic history. There was a gradual intermixture both within and between major groups that led to the present New World population. The depopulation of the New World and its repopulation with peoples of different ethnic backgrounds was a complex process that can be understood, but it cannot be characterized as a discrete historical event, nor was it accomplished in a generation. Neither was the process of epidemiological and demographic transition the same for all ethnic groups both within and between regions and at different times. It is useful to use the transition terms to describe historical events, but it is misleading, at best, to believe that there was just one such phenomenon.

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The epidemiologic transition revisited: or what happens if we look beneath the surface?*

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John Rogers[a] and Marie C. Nelson[b]

[a]*Department of History, Uppsala University*

[b]*The Tema Institute, Department of History and Department of Health and Society, Linköping University*

In a broader context the decline in mortality in Europe has spawned studies in a number of disciplines. The decline and its nature have been carefully charted, but the reasons for the decline have occasioned debate concerning the role of factors such as urbanization, industralization, changes in living standards and conditions, improvements in health and medical care and the introduction of sanitation in towns and cities. While each explanation has had its defenders, the tendency is now toward illuminating the complexity of the situation. McKeown (1976) launched much of the current discussion by arguing the case for the primacy of improvements in nutrition as opposed to medicine in the decline of mortality in Europe. Many volumes have appeared since then emphasizing the role of various factors, and the debate is still very much alive. Recently Schofield and Reher (1991) have summarized some main trends in this debate, concluding that no single cause may be awarded the sole explanatory role and arguing for an interdisciplinary approach to questions of the mortality decline. There are numerous studies that could be named here which, while recognizing the complexity of the problem, plead the case for one or the other variable. In his study of the European mortality decline, which primarily focuses on England, Mercer (1990)emphasizes the role played by improvements in nutrition and standards of living, but casts his vote for prevention and public health measures. Livi-Bacci (1991) has, for example, again taken up the discussion of nutrition and pointed out the short-term effects in the mortality decline, but reached the conclusion that it does not have the decisive role in the long run.

Sweden was very much a part of this development. The decline of mortality in Sweden resulted in substantial increases in population during the nineteenth century. The death rate declined from 28 deaths per thousand population in 1749 to half that rate in 1910. By the 1940s the figure had reached ten per thousand.

Fragments of the Swedish picture have been presented, but a real overview of the components is still missing in the published literature. For example, southern Sweden has been dealt with in a number of articles. Imhof and Lindskog (1973) studied causes of death in that region for 1749-1773. In his study of the relationship between harvest fluctuations and demographic change 1751-1858 in southern Sweden, Bengtsson (1984) reached the conclusion that the effect of nutrition on mortality increased over time. With evidence for the same part of the country, the case for changes in disease virulence as the cause of major fluctuations in the mortality decline has been pleaded by Fridlizius (1984).

Of the various age groups, it is infant mortality which has been most carefully studied. Breastfeeding, the life situation of women, and general economic factors have all been emphasized in various studies (e.g. for Sweden, Lithell 1981; Brändström 1984). There has been research on urbanization and its relationship to decreases in infant mortality, taking into account size of urban areas, the establishment of water and sanitation systems and overcrowding (Edvinsson 1992; Nelson and Rogers 1994, 1995).

Studies have also been made of specific epidemic diseases. This work may be divided into several categories: works dealing with the sources and their reliability; studies of specific epidemic diseases and/or geographical areas during a limited time period (early

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epidemiological or aetiological studies by either members of the medical profession or by demographers), and so-called disease histories. Surveys of a more general nature are less common.[1]

The epidemiologic transition

The mortality decline *per se* has often been interpreted in terms of the epidemiologic transition as conceived by Omran (1971, 1982). To apply the epidemiologic transition to Swedish history is somewhat misleading, as this concept was actually developed from a study of Swedish source material. The theory is based upon five basic propositions: (1) the fundamental role of mortality in population dynamics; (2) the long-term shift of mortality in three basic stages from high and fluctuating mortality due to infections and famine, through a period when epidemics lose their clout, to a level where degenerative and man-made diseases are the major killers, with a relatively low level of mortality; (3) the transition favours the young and females with a maintenance of class differentials; (4) medical progress made itself felt first in the twentieth century, while nutrition and the living standard were most significant in earlier times; (5) there are four basic models: classical; accelerated classical; the delayed; and the transitional delayed.[2] Even though the model has been modified to accommodate other situations and parts of the world than those initially studied, it seems worthwhile to try to evaluate its validity when the components of the model are scrutinized more closely.

In the classical or Western model of the epidemiologic transition the previous two hundred years have seen a shift from high death rates of about 30 per thousand annually to low death rates of less than ten per thousand. Communicable diseases have given way to degenerative diseases as major causes of death. In Sweden life expectancy steadily increased during the transition period. During the period 1749-1930 the life expectancy of males which was 33.7 years at birth for the period 1751-1790 had increased to 61 for the period 1921-1930; the corresponding figures for females were 36.6 years for 1751-1790 and 63.2 years for 1921-1930 (see Table 1). Life expectancy at age 50 was 18.2 years in 1751-1790 and 24.2 years in 1921-1930 for males; the increase in female life expectancy for the same periods was from 19.6 to 25.1 years (*Historisk Statistik* 1969:118). According to Omran's model, females should be favoured over males during the transition period. It is questionable whether this was the case in Sweden as female mortality was lower even during the pre-transitional period. Males experienced an 81 per cent improvement in life expectancy at birth, 27.3 years, and females 73 per cent, 21.8 years. True, during the transitional period in the middle of the

[1] An early classic in the field of disease studies in Sweden is von Rosenstein 1771. During the late nineteenth century, especially after the birth of modern bacteriology, an increased number of medical topographical studies began to appear. Bibliographical information concerning some of the older material may be found in Åman 1990; Sköld 1996.

[2] These are clearly described in Omran 1982. A more recent modification and clarification of these models has been presented in Omran 1983. Swedish material inspired one of the original models, the classical or Western model.

nineteenth century, females showed more improvement but this lasted only for a couple of decades. There are increases for both e_0 and e_{50}, but much of the improvement that took place was among the young, a point emphasized if one looks at the life expectations of 15-year-olds. Life expectancy for 15-year-old males in 1751-1790 was 40.5 and had increased to 52.7 for the 1901-1930 period; for females the changes were from 43.1 to 53.9 (*Historisk Statistik* 1969:118). The decline in the rate of infant mortality further substantiates this as IMR dropped from 205 per thousand in the period 1751-1760 to 59 per thousand in 1921-1930 (*Historisk Statistik* 1969:115).

Table 1
Expectation of life at different ages

Year	Males at age of						Females at age of					
	0	1	15	50	65	80	0	1	15	50	65	80
1751-1790	33.72	42.06	40.50	18.16	10.02	4.60	36.64	44.65	43.09	19.59	10.51	4.76
1791-1815	35.35	43.84	40.64	17.26	9.29	3.92	38.44	46.24	42.90	18.68	9.84	4.14
1816-1840	39.50	47.10	41.23	17.55	9.58	4.03	43.56	50.50	44.65	19.60	10.44	4.46
1841-1845	41.94	49.26	42.98	18.32	9.82	3.84	46.60	53.22	46.52	20.48	10.77	4.35
1846-1850	41.38	48.46	42.60	17.89	9.43	3.73	45.59	51.96	46.18	20.02	10.40	4.16
1851-1855	40.51	47.17	42.03	17.86	9.63	4.09	44.64	50.73	45.50	19.83	10.40	4.48
1856-1860	40.48	46.87	43.75	19.17	10.46	3.12	44.15	49.80	46.38	20.72	11.17	4.91
1861-1870	42.8	49.3	44.9	19.4	10.4	4.3	46.4	52.1	47.8	21.3	11.4	4.7
1871-1870	45.3	51.6	46.3	20.8	11.2	4.6	48.6	54.1	49.0	22.6	12.2	5.2
1881-1890	48.55	54.14	48.14	21.94	11.96	4.81	51.47	56.19	50.30	23.64	12.92	5.40
1891-1900	50.94	56.25	48.66	22.44	12.24	4.88	53.63	58.04	50.61	24.04	13.13	5.40
1901-1910	54.53	59.06	49.79	23.17	12.81	5.22	56.98	60.64	51.48	24.74	13.69	5.64
1911-1920	55.60	59.18	49.06	23.35	12.84	5.26	58.38	61.16	51.13	24.79	13.69	5.66
1921-1930	60.97	64.18	52.70	24.15	13.24	5.45	63.16	65.50	53.90	25.14	13.85	5.73
1931-1940	63.76	66.17	53.94	24.08	13.06	5.31	66.13	67.78	55.32	25.13	13.64	5.55
1941-1950	68.06	69.19	56.26	25.01	13.60	5.55	70.65	71.33	58.17	26.27	14.30	5.83
1951-1960	70.89	71.35	58.01	25.54	13.85	5.70	74.10	74.27	60.73	27.47	15.00	6.10
1961-1965	71.60	71.81	58.31	26.65	13.88	5.82	75.70	75.68	62.06	28.56	15.80	6.44
1966	71.87	71.93	58.40	25.79	14.00	6.02	76.49	76.30	62.64	29.12	16.26	6.74
1967	71.85	71.88	58.27	25.74	13.99	6.05	76.54	76.42	62.72	29.22	16.31	6.77

The table refers to the average number of years to be lived by persons of certain ages, who are assumed to be subject to the mortality rates observed in the period indicated, for the remainder of their lives.

In the earliest period the general mortality pattern in Sweden shows clearly the extremes that mark the phase characterized by pestilence and famine, which may be assumed to be a continuation of earlier patterns. Studies have been conducted which provide glimpses into an era for which statistics are not commonly available and often extremely difficult to compile. At least 22,000 people died in the city during the plaque between August 1710 and February 1711, at a time when the city's population numbered about 55,000..

A quick look at the eighteenth century shows that 1772-1773 stands out; in 1772 the death rate for the nation soared to 37.4 per thousand, only to reach new heights of 52.4 per thousand in 1773 (*Historisk Statistik* 1969:91). This was the classic case of famine and disease going hand in hand. A study of the town of Örebro has shown that mortality in the area more than doubled during the crisis.[3] 1808-1812 were also years which were outstanding, especially 1809, when the crude death rate again exceeded 40 per thousand.[4] After this period of high death rates, which coincided with Sweden's involvement in its last war, mortality gradually declined with occasional setbacks due to epidemics. But these reverses did not affect the aggregate figures to the extent of the crises of the earlier period.

The epidemiologic transition model considers the diminishing role of epidemic or infectious diseases for a general decline in mortality. During the first phase of Omran's model, the age of pestilence and famine, such diseases are assumed to be fairly common and a major cause of high death rates and low life expectancy. In Sweden during the last quarter of the eighteenth century the five biggest killers, 'fevers', measles, whooping cough, diphtheria and smallpox, accounted for about one-quarter of all deaths. There were substantial fluctuations as would be expected. By 1900 these epidemic diseases accounted for approximately five per cent of all deaths. In this paper we concentrate on the period during which epidemic diseases begin to decline in importance, that is, the period 1796-1820. We further consider whether or not there were any significant regional variations. It would be convenient to begin at an earlier date and it certainly would be helpful to continue forward in time. As historians too often experience, the providers of the sources were not particularly interested in our historical questions. Regional figures are available from 1749 to 1830, but the administrative areas are not directly comparable. For the period 1830 to 1860 only a few causes of death were recorded.[5] The material presented here is based primarily upon statistics formerly available in the archives of *Statistiska centralbyrån* (the Swedish Central Bureau of Statistics - SCB) for the period before 1830, and after 1860 on the statistics found in *Bidrag till Sveriges officiella statistik. (A) Befolkningsstatistik* and *Sveriges officiella statistik.*[6] The material concerning deaths from communicable diseases, commonly known in Swedish (together with other common maladies) as *folksjukdomar* or 'folk diseases' is derived from these sources, which have their limitations. Generally causes of deaths were reported on printed forms, which were revised periodically from the inception of the *Tabellverket,* a forerunner of SCB, until 1830 and again after 1860; during the interim only deaths due to

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[3] For a general study of such crises, see Ohlander and Norman 1984; Nelson 1988:23-25. For 1772-1773, see Ohlander and Norman 1984:170-172; Norman 1983; Emanuelsson 1982.

[4] *Historisk Statistik* 1969:93; Norman 1983. Sweden was at war with Russia during 1807-1809. Much of the fighting took place in Finland and generally the east central provinces of Sweden were affected with considerable troop movements in the area.

[5] For the period, statistics were collected by county(several counties have significant gaps) and the figures were aggregated by ecclesiastical administrative districts; after 1795 the tabulation of data was again by county. For a discussion of cause-of-death registration and additional references, see Rogers forthcoming.

[6] The archives at the Central Bureau of Statistics (SCB) have been closed. Some material is now available in the collections of the National Archives, Stockholm, while some is still deposited at SCB.

childbirth, smallpox, suicide, accidents, and other major epidemics were reported (see Rogers forthcoming).

Sailing in uncharted waters

The concept of the epidemiologic transition was developed on the basis of aggregate Swedish statistics. In reality Sweden's mortality pattern showed considerable variation throughout the country. One of Sweden's best known demographers, Gustav Sundbärg, observed at the turn of the century, 1900, significant differences in demographic development, not least with regard to mortality, during the nineteenth century; discussions concerning demographic variations within the country often use his categorization. Sundbärg described three distinctive demographic regions: the eastern, the western and the northern. In the western region marriages were relatively few and were late, fertility was high and mortality low. In the eastern region the nuptiality rate was high, the birth rate low, and the death rate higher than in other regions. The northern region had the highest fertility and a low death rate (Sundbärg 1910:4-9).

With this in mind we explore the distribution of epidemic diseases at the provincial level during the period 1796-1820, to determine if there were any significant regional differences at the outset of the transition and consider any changes which might have occurred. Was there one general pattern or several transitions? Does the disease panorama vary and does it change over time? Are the patterns of change similar or dissimilar? If there are any regional differences in the epidemic mortality level, the disease panorama or the pace of change, can these be related to regional variations in population density, urbanization or some other variable? We shall not consider age-specific, sex-specific or social class-specific mortality, as the figures do not allow such analysis. Furthermore, dealing with causes of death during this early period creates numerous problems of interpretation. We are forced to use those classifications used by the compilers of the statistics, which influences the reliability of the information. We therefore use only simple tables and figures, as any more sophisticated statistical analysis would amplify the problem of unreliable sources.

Table 2
Crude death rate per thousand by province 1796-1905

Years	1796-1800	1801-1810	1811-1820	1821-1830	1831-1840	1341-1850	1851-1860	1861-1870	1871-1880	1881-1890	1891-1900	1901-1905
Jämtland	17.02	19.10	15.23	14.14	14.18	12.95	13.86	14.94	15.96	15.93	16.08	14.40
Gävleborg	22.38	23.12	19.89	18.65	18.49	17.44	18.59	20.38	20.31	18.30	16.36	15.09
Värmland	22.99	28.35	24.71	21.83	19.16	18.52	20.90	18.70	15.88	14.17	15.24	15.47
Älvsborg	23.15	28.18	25.23	21.38	21.52	19.40	21.12	19.55	16.26	15.29	15.31	15.40
Västernorrland	23.47	24.76	22.21	20.45	21.35	18.02	18.52	20.89	20.11	18.61	17.33	15.21
Kalmar	23.54	27.36	24.73	24.00	22.03	22.41	22.22	19.54	17.13	15.93	15.93	16.07
Gotland	23.67	22.67	20.76	19.11	19.75	18.66	16.67	15.85	16.18	15.52	16.51	16.74
Jönköping	23.75	25.40	24.50	20.05	19.26	18.15	22.40	18.03	15.16	14.78	14.55	14.30
Södermanland	23.76	25.31	24.88	23.07	21.84	21.61	21.19	21.26	18.84	17.19	15.79	14.56
Halland	23.92	26.07	26.83	23.35	23.03	20.50	19.44	20.54	17.49	16.15	15.68	16.04
Kronoberg	24.00	27.09	25.47	23.21	21.40	19.70	19.09	19.30	15.74	16.01	15.94	15.67
Kopparberg	24.11	26.45	22.83	20.54	22.77	19.93	20.62	19.89	20.42	17.41	16.37	15.90
Kristianstad	24.13	25.15	23.46	23.88	22.04	19.17	20.16	18.29	17.40	17.16	17.39	16.34
Uppsala	24.56	28.65	27.67	25.76	24.70	21.47	23.97	21.68	20.59	18.59	17.02	15.66
Malmöhus	26.39	25.89	23.47	23.82	23.38	19.96	19.69	18.62	18.07	17.60	16.52	15.38
Västerbotten	26.66	30.31	21.73	20.45	20.04	14.95	17.32	20.06	16.87	16.93	16.53	14.99
Norrbotten	26.66	30.31	22.16	20.40	22.06	18.76	20.77	24.50	20.00	21.56	20.47	17.56
Stockholm province	26.84	31.33	30.86	29.05	27.34	24.53	25.55	23.08	20.65	18.12	17.24	15.93
Skaraborg	27.22	28.10	27.37	23.70	23.25	20.85	22.47	18.36	15.40	14.61	14.90	14.74
Blekinge	27.25	31.68	27.38	27.58	23.49	22.84	24.63	22.09	20.35	18.27	17.64	16.92
Östergötland	27.46	27.24	27.43	24.99	22.32	21.58	22.21	20.02	18.18	15.57	15.87	14.72
Västmanland	27.61	30.24	28.03	26.06	24.79	21.91	23.80	21.50	21.00	18.35	16.18	14.88
GöteborgBohuslän	27.63	33.47	28.12	24.68	25.50	20.86	22.96	21.67	18.75	17.73	16.54	15.95
Örebro	28.72	28.62	26.89	21.65	21.49	20.51	20.96	18.55	16.23	14.95	14.28	14.20
Stockholm city	42.21	49.61	43.93	44.39	46.08	38.11	41.51	32.25	30.28	22.60	19.06	16.05

Regional variation in crude death rates and epidemic mortality

As can be seen in Table 2, crude death rates (CDR) varied little with the exception of the city of Stockholm and the province of Jämtland. If we look at the period under study, 1796-1820, we find that in the first sub-period Jämtland had 17 deaths per thousand and the city of Stockholm more than 42 per thousand. For the remaining provinces the difference between the high and the low was slightly above six per thousand (1796-1800). Yet within this group there were two major categories: one with a range of around 22 to 24.5 per thousand, the other ranging from 26.4 to 27.6 per thousand. Örebro province was above the level of this last group at 28.7 per thousand. Both groups of provinces present a geographical mix.

During the years 1801-10, Stockholm city and Jämtland still represented the extremes with a high of 49.6 and a low of 19 deaths per thousand, respectively. The difference among the remainder was 11 per thousand. The war years during this decade caused both the high figures and the unequal distribution as some of the provinces were not affected by the war.

During the third period, 1811-20, both the extremes dropped sharply, Jämtland to a level slightly lower than the starting point and Stockholm to a level slightly above. During this period the CDR for the remainder ranged from about 20 per thousand to over 30. These fall generally into two categories: provinces with falling death rates and those with stable or slightly increasing rates.

By the beginning of the period, three of the provinces in Sundbärg's eastern region and Stockholm city were among the areas with the five highest CDRs in 1796-1800. The only misfits here were Göteborg and Bohuslän, which had a high death rate and belonged to the western region. The provinces of Uppsala and Stockholm had respective ranks of 14 and 18 (1 was lowest and 25 highest). Provinces that were part of this eastern region, but not among the half with the highest rates, were Jämtland, Gävleborg and Södermanland. By the end of the period studied in depth here, 1811-1820, Jämtland, Gävleborg, and Göteborg and Bohuslän retained the same or similar relative positions. Västerbotten, Norrbotten, Malmöhus and Kristianstad had improved their relative positions. Table 2 shows that, in terms of mortality as expressed in CDR, Sundbärg's division into three regions does not appear to have validity early in the nineteenth century, although some provinces seem to be moving in that direction.

On the basis of these observations we can group the provinces as follows: (1) provinces with a CDR above 25 per thousand which experienced declining death rates; (2) provinces below 25 per thousand with declining death rates; (3) provinces with a CDR above 25 per thousand and stable or slightly decreasing death rates; (4) provinces with a CDR below 25 per thousand with stable or increasing death rates (see Table 3).

Table 3
The percentage of dealths caused by infectious epidemic diseases of all deaths 1796-1820 by province

Provinces with declining crude death rates of those provinces with crude deaths rates above 25 0/00 1796-1800	
province	%
Malmöhus	22.2
Old Västerbotten	11.5
Örebro	26.9
Provinces whose crude death rates do not decline of those provinces with crude deaths rates above 25 0/00 1796-1800	
Province	%
Province of Stockholm	23.8
Skaraborg	27.6
Blekinge	24.6
Östergötland	23.9
Vastmanland	25.5
Göteborg and Bohus	29.4
City of Stockholm	21.4
Provinces with declining crude death rates of those provinces with crude deaths rates below 25 0/00 1796-1800	
Province	%
Gävleborg	22.3
Old Västernorrland	16.7
Gotland	18.0
Koparberg	27.7
Kristianstad	22.1
Provinces whose crude death rates do not decline of those provinces with crude deaths rates below 25 0/00 1796-1800	
Province	%
Värmland	29.6
Älvsborg	27.6
Kalmar	26.4
Jönköping	25.7
Södermanland	26.6
Halland	22.3
Kronoberg	25.4
Uppsala	25.3

Of the three provinces that fall into group 1 (high and declining death rates), Old Västerbotten[7] shows a clear decline in deaths from infectious diseases. The provinces of Malmöhus and Örebro showed no marked decrease in the proportion of epidemic diseases. Furthermore, the pattern is more stable, without exaggerated peaks.

In the second category (low and declining death rates) two provinces, Kristianstad and Gotland, had no outstanding peaks and showed no pronounced decrease in the proportion of epidemic diseases. The other three provinces, Kopparberg, Gävleborg and Old Västernorrland, still had a number of epidemic peaks. The disappearance of the peaks towards the end of the period tends to explain their declining death rates.

For provinces with declining death rates we can observe two patterns: first, for one group of provinces (low and declining rates), the declining crude death rates appear not to be related to a decline in the proportion of epidemic diseases; secondly, there is a group of provinces (high and declining) where the decline seems to be related to the elimination of mortality peaks caused by epidemics.

For group 3, provinces with high crude death rates which showed no tendency to decline, the pattern is less clear. For the province of Östergötland the proportion of epidemic deaths of all deaths remained at a fairly high and stable level. For the other provinces in the group there is some peaking, but the mortality peaks are not nearly as pronounced as in provinces with declining crude death rates. The provinces which had relatively low crude death rates from the outset and showed no improvement (group 4), also had varied patterns. Two provinces, Värmland and Jönköping, had epidemic peaks which tended to eliminate any improvements. Among the others only moderate mortality peaks occur.

In general then, there is no clear relationship between levels of mortality or changes in mortality levels and the proportion of epidemic diseases or changes in their relative strength. Is there any common pattern among the provinces with significant epidemic mortality peaks? All the provinces experienced smallpox epidemics in 1800-1801 which resulted in mortality peaks, although the impact of the epidemic varied. Whooping cough reached epidemic proportions during these years, but only in the northern provinces of Old Västernorrland and Old Norrbotten did whooping cough contribute significantly to mortality. Dysentery struck the province of Västmanland in east central Sweden in 1803 and the province of Halland in southwestern Sweden in 1806. The island of Gotland experienced an outbreak of whooping cough in 1806 followed by dysentery the following year. The other provinces, although the level and mix of epidemic mortality varied, did not experience a significant epidemic until the war years of 1808-1809, which brought dysentery to most of the provinces in east central Sweden, although the severity of the epidemic varied somewhat. Dysentery reached epidemic proportions in the provinces of Örebro, Jönköping and Älvsborg in 1819; and towards the end of the period we can observe the beginnings of a measles epidemic. The only clear patterns which emerge are that smallpox hit the entire country in 1800-1801 and that dysentery was prevalent in east central Sweden in 1808-1809. The epidemic disease panorama thus varied significantly.

Regional distribution of various epidemic diseases

Another way of considering the effects of infectious diseases is to consider the distribution of the diseases during the period studied. Smallpox shows a regular pattern with five to six-year intervals and decreasing intensity. Smallpox is clearly a childhood disease during this period.

[7] No province has existed with the name Old Västerbotten, but we have used the term here to refer to the northernmost province before the area was divided into two provinces, Västerbotten and Norrbotten, to distinguish it from the later province bearing the same name.

The province of Halland is typical of this pattern. The city of Stockholm, the island of Gotland and the provinces in the northern part of the country are more or less free from smallpox after 1810. The province of Kopparberg provides a good example. Sweden introduced vaccination at an early date and, through the use of the state church to record births and vaccinations and church officials as vaccinators, effectively combated smallpox.[8] Gotland's isolation and the effectiveness of Stockholm's early vaccination efforts may explain the early reduction of smallpox here but the disappearance of smallpox in the northern provinces is more difficult to explain. Certainly they were generally less densely populated but still had experienced significant epidemics earlier.

Another childhood disease, measles, shows three fairly distinct patterns. In northern Sweden epidemic outbreaks were on a lower level, representing 2-3 per cent of all deaths in an epidemic year. Only in one instance, in 1803 in Old Västerbotten, did deaths from measles reach ten per cent. In central Sweden epidemics were regular and generally in the ten per cent range. In southern Sweden outbreaks of measles produce moderate mortality figures until 1814, generally at the 5-6 per cent level. In 1814 and again in 1820 the proportion of deaths due to measles jumps to 12-16 per cent of all deaths. Northern Sweden was not only less densely populated but also little urbanized during this period and thus would have trouble sustaining a measles epidemic. The island of Gotland reveals a similar pattern. The rest of the provinces were more densely populated and more urbanized and thus could more easily sustain an epidemic. Measles epidemics and smallpox epidemics did not generally coincide and even the worst measles epidemics did not produce significant peaks in epidemic mortality in comparison with, for example, Värmland.

Dysentery affected generally the central part of the country. In a band of provinces from Uppsala in the east to Göteborg and Bohus in the west, dysentery was prevalent most years during the period. About half of these provinces had dysentery mortality rates generally between five and ten per cent of all deaths. The rest had a similar level of dysentery mortality but also experienced one or more severe epidemics. In provinces north and south of this band dysentery was associated with 1809 and the following couple of years. Here mortality was high, generally in the range of 25-35 per cent and at times reaching 50 per cent of all deaths. Only in the northernmost province of Old Västerbotten was dysentery a minor disease (2% or less of all deaths).

Diphtheria was present throughout the period but at low levels causing generally less than two per cent of all deaths; only in Malmöhus did it reach above three per cent. Diphtheria, which later would be a major killer, apparently was insignificant at the levels of epidemic mortality during this period.

Malaria deaths were often important in deaths due to infectious diseases, at times reaching epidemic proportions. In the provinces of east central Sweden (Stockholm, Uppsala, Västmanland, Södermanland and Östergötland) malaria deaths were in the 2-4 per cent range with an epidemic year in 1812 when the proportion of malaria-related deaths reached ten per cent. In the provinces of Kalmar, Kronoborg and Jönköping malaria mortality was lower but reached epidemic proportions also in 1812. This is also true for Göteborg and Bohus and Halland on the west coast. The island of Gotland experienced an epidemic in 1814-1815. In the rest of the country malaria deaths were few. Malaria gradually disappeared during the nineteenth century, largely as a by-product of extensive drainage projects.

Deaths due to whooping cough represented generally between ten and 15 per cent of all deaths. Only two provinces, Old Västerbotten in the north and Halland in the southwest, had higher mortality figures at 18 and 19 per cent, respectively. A group of provinces in north

[8] For studies of smallpox, consult the notes in Nelson and Rogers 1992. A recent comprehensive study of the disease in Sweden is Sköld 1996.

central Sweden (Gävleborg, Västmanland, Kopparberg, and Värmland) and two adjacent provinces in the southeast (Kalmar and Blekinge) had somewhat lower proportions of deaths due to whooping cough (7-9%). The pattern is regular throughout the country with outbreaks in 1796-98, 1801-02, 1805-06, 1812-13, and 1816-18. Whooping cough epidemics occurred between the measles epidemics.

Fevers which included scarlet fever and other unspecified fever illnesses represented by far the largest component of the epidemic panorama. Fevers accounted for between 40 and 50 per cent of all epidemic deaths during the period in most provinces. In the city of Stockholm fevers accounted for 60 per cent of the deaths attributed to infectious diseases. The pattern over time is fairly stable at around ten per cent with those provinces which were affected by the war in 1808-1809 showing fever mortality peaks. Separate figures for scarlet fever are available for the 1806-1810 period but show no correlation with the fever mortality peaks. It is difficult to judge the significance of fevers for the overall picture, as we do not know what was included other than scarlet fever. It appears that the proportion of deaths due to fevers was fairly stable and generally evenly distributed over time among the provinces.

Population density and epidemic disease

As can be seen, Sweden was not a particularly densely populated country. Two patterns emerge. First, the most densely populated provinces, with population density between 10 and 24 per square kilometre in 1750, are the same as those in group 3, that is, provinces with high crude death rates and no significant declines in mortality during the period. Here we find the classic correlation between densely populated regions with numerous urban areas and a high incidence of infectious disease. The other three groups classified by level and change in CDR contain a mix of densely populated and less densely populated provinces. In the sparsely populated provinces in northern Sweden, with population density less than five per square kilometre in 1750, measles played an insignificant role in the epidemic panorama. This is only to be expected, since to sustain an outbreak of measles at epidemic proportions requires concentrations of people, and there were only a few small towns in the north during this period. No other specific disease shows any correlation with population density.

Conclusion

During the initial phase of receding epidemics in Sweden's epidemiologic transition we find a variety of transitions at a regional level. The interpretation of these results is far from self-evident. This is in part due to how one chooses to look at the role of epidemic disease. If we consider crude death rates, we find that in the most densely populated and highly urbanized provinces of the country crude death rates were high with a significant proportion of all deaths caused by infectious diseases. Epidemics appeared regularly but in relative terms did not cause the dramatic mortality tops seen in many of the other provinces.

Some of the provinces with declining crude death rates clearly were passing through the stage of receding epidemics, as peaks in epidemic mortality disappeared in the later part of the period resulting in lower death rates. Several provinces appear to have declining crude death rates which were not related to a decrease in epidemic diseases, as the proportion of deaths due to infectious diseases remained high, although towards the end of the period mortality tops disappeared, indicating that they were entering the transition. Others appear to be taking their first steps in the epidemiologic transition.

Transitions can also be observed on the basis of the disease panorama. Several provinces in central Sweden had high rates of malaria-related deaths and for this disease improvements were yet to come. In the northern provinces, because of the sparse population,

measles was not the killer it was in the rest of the country. Dysentery affected most of the provinces but those which were involved in Sweden's war with Russia, sending troops or receiving prisoners, were harder hit. The transitions related to smallpox were related to a large degree to the implementation of a successful vaccination campaign, although the earlier decline in the north is puzzling. Only whooping cough continued to affect all areas. Of course, even those provinces which were clearly entering the stage of receding epidemics had new unknown dangers ahead of them. Cholera was waiting down the line, as was the less dramatic, but lethal, diphtheria.

If one wishes to understand the mechanisms behind the disappearance of epidemic disease, it is necessary to look beyond aggregate national data which hide more than they reveal. During the period covered here several patterns emerged affecting local populations differently. Interestingly, however, the various diseases rarely overlapped and, given the short time period we are considering, this meant that the population was suffering from one disease or another nearly every year, although not necessarily in the same order.

Should these variations in patterns be interpreted as evidence of the existence of several epidemiologic transitions, or of one? While at first glance it might be tempting to conclude that there were several transitions, the results point in the other direction. The transition did not occur in all areas simultaneously. What we see here are different stages in the phase of receding epidemics. Some provinces were just entering the receding phase, while others were well on their way. The timing of the decreases in infectious diseases thus influenced the disease mix in each particular area. Sweden as a whole was experiencing the epidemiologic transition, but different regions were experiencing this in different ways.

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