History and Health Science: Medical Advances across the Disciplines


Historians and bench scientists have viewed each other’s work with a mixture of envy and suspicion. Historians have longed for the precision and replicability of scientific experimentation. Medical scientists and microbiologists have, in turn, admired history’s ability to capture the broad sweep of human society and the motivations of individuals. Most scholars have despaired of bridging the gap between the highly technical and incremental enterprise of science and the careful evaluation of sources and syntheses.


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Nonetheless, the process has begun. As Rotberg put it in the introduction to a collection of essays which appeared in *The Journal of Interdisciplinary History* between 1975 and 1996, scholars are exploring “the deployment of medical information and insight to solve historical questions previously presumed to be solely political, social, or economic.”

This essay surveys a number of books by life scientists who have written historical narratives and historians whose work has incorporated insights from the life sciences. Part of this discussion examines the distortions and anachronisms that inevitably arise when practitioners of one discipline arrogate the methods and findings of another. Indeed, differences of emphasis even arise in citation conventions: Both historians and bench scientists list authors for short citations, but the former often stress a publication’s title and the latter its date. Notwithstanding such formal discrepancies, however, historians and scientists—who, at first glance, would appear to have widely different orientations—ultimately represent the interactions between people and other inhabitants of the biosphere in surprisingly similar ways.

Unfortunately, some of the pioneering monographs in medical history, by historians and scientists alike, stand in need of re-evaluation in the light of modern microbiology. These works fall into two categories: those that rely on symptoms and clinical signs, rather than molecular structure or properties of the organisms, to identify pathogens and those that use terminology and explanations based on outmoded notions of immunology. The hypotheses founded on their questionable assumptions and methodologies can no longer be considered the state of the art in medical writing.

**Archaisms in the Classics** During the last 130 years, bench scientists have made enormous progress in understanding the causes of disease. Previously, diseases could be understood only through their symptoms and clinical signs. As William Coleman, *Yellow Fever in the North* (Madison, 1987), and James C. Riley, *The Eighteenth-Century Campaign to Avoid Disease* (New York, 1987), have shown, clinicians gave their utmost attention to external conditions—physical changes and the links between these pathological phenomena and particular environments and behaviors.

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Of the microbiologists, Robert Koch, in particular, developed relatively rigid criteria for proving that a specific organism caused a specific disease. Not all subsequent identifications, however, stood the test of time. Before the 1930s, microscopes were not strong enough to detect individual viruses, and even closely related bacteria could be confounded. At the turn of the twentieth century, typhoid and paratyphoid infections, for example, were lumped together as enteric fever.

In the twentieth century, however, microbiologists and clinicians increasingly confirmed their diagnoses through microscopic analysis and microbiological methods, including the ability to culture and characterize organisms serologically. By the 1950s, led by the work of the geneticists James Dewey Watson and Francis Harry Compton Crick, and parallel advances by immunologists, the bench scientists achieved a higher level of precision—the identification of the molecules and their structure within cells, and of the specific genomes that contained the unique instructions for the genetic structure of all organisms—from viruses to mammals. In the 1990s, microbiologists developed tools that allowed an even higher level of precision: distinguishing the molecular structure of entire genomes and examining the interactions among the various genes and proteins that organisms form according to instructions from their genomes.²

Contemporary science has advanced well beyond the study of symptoms and clinical findings, at least so far as currently living organisms are concerned. Much of our information about diseases of the past, however, still depends largely on historical accounts of symptoms and signs. Analogously, when a physician first sees a sick patient, clinical findings are critical in determining the direction of the diagnostic pathway. Although scientists are able to identify certain past microorganisms by using, for example, a polymerase chain reaction to amplify material from dead organisms, the body of evidence thus recovered is neither as large nor as temporally extensive as the descriptions available in historical sources.³ The temptation is to comb the verbal evidence of symptoms for want of microbiological data. Paradoxically, such genetic evidence is important almost exclusively in a historical context, since

the new methodologies are so sensitive that they detect trivial organisms or contaminants irrelevant to the problem being researched.

Historians have tended toward evidentiary conservatism. Although some historians of health have used physical, visual, and statistical evidence to reconstruct the past, many depend almost exclusively on verbal evidence—both written and oral. The call for new evidence generally means verbal evidence. For example, McNeill, in *Plagues and Peoples*, solicits “epidemiologically informed researches . . . undertaken in Chinese and other ancient records.”

This appetite for description of clinical findings, alas, is not restricted to historians. Bench scientists have also fallen into this trap. In the absence of microbiological evidence regarding ancient and medieval *Yersinia pestis* (bubonic plague), Wills cites the writings of Procopius and Bocaccio, sources also employed in Diamond’s immensely popular *Guns, Germs, and Steel*. For the present, the earliest direct evidence of the *Y. pestis* organism dates from fourteenth-century France. It derived from laboratory analysis of *DNA* taken from unerupted wisdom teeth.

In the matter of the body’s defenses against disease, historians and bench scientists have evinced a similar proclivity to employ practices no longer current. Immunology has made enormous strides within the past thirty years in understanding the complexity of mammalian defenses against invading microorganisms. Some of these defenses are genetic and specific to certain members of a human population. In any human population, individuals possess varying combinations of human leukocyte antigens (HLA), known more generally as major histocompatibility complexes. These complexes are associated with individuals’ resistance or susceptibility to particular diseases, including arthritis, ankylosing spondylitis, vivax malaria, and tuberculosis.

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White blood cells themselves play a significant role. In a summary intended for non-specialists, Hall describes some of the soldiers of the immune system. . . . The body’s six kinds of white blood cells that all come from a single stem cell in the bone marrow . . . neutrophils, which engulf bacteria upon contact and send out early-warning signals; monocytes, which turn into macrophages that swallow up invaders; eosinophils, which attack parasites; and basophils, whose granules contain histamine and other compounds related to allergies. The lymphocytes—B cells and T cells, which are tailored to the invader—arrive later at the field of battle. The B cells turn into plasma cells that manufacture thousands of highly specific antibodies, which are released into the bloodstream. The T cells coordinate the entire immune response and eliminate viruses that hide inside infected cells.7

Given the complexity of the subject, especially in bench scientists’ own terms, it is not surprising that discussions of human resistance to infectious disease in the historical literature do not reflect current immunological thinking. McNeill, citing books published in 1934 and 1956, refers to “the white corpuscles, which constitute a principal element in defenses against infection [and] actually digest intruders,” a conflation of macrophages and neutrophils. Curtin and McNeill stress the importance of acquired immunity, which subsumes the function of lymphocytes, and Curtin’s later assertion that “an individual’s pattern of immunity is largely established in childhood” must now be tempered by current thinking on the body’s continuous encounter with potentially harmful microorganisms.8

Nor are a number of what used to be “biologic truths” about individual diseases valid any longer. In 1935, Zinsser attributed the gradual disappearance of bubonic plague from Europe to “the increased domesticity of rats. . . . which remain contentedly at home, and as a consequence of this, plague foci among them remain restricted to individual families and colonies.” In a recent survey of the literature relating to Yersinia pestis, however,

8 McNeill, Plagues and Peoples, 7, 3; Curtin, Death by Migration, xiv.; idem, Disease and Empire, 4; Levin and Antia, “Why We Don’t Get Sick.”
Hinnebusch stated that the bacillus can be transmitted by 80 species of flea to no fewer than 200 species of rodent. Zinsser also linked the decline of leprosy in medieval Europe to bubonic plague: "(Most) of the lepers of Europe were wiped out by the plague, and . . . the few who survived were too scattered and represented too meager a spark to revive the disease." But McNeill’s suggestion that exposure to tuberculosis most likely produces immunity to leprosy because the two are closely related mycobacterially is more in accord with contemporary research.9

Another source of error for historical medicine is malaria. McNeill’s statement that “malaria plasmodia do not trouble the mosquitoes that carry them from human host to human host” is far from reality. Individual female anopheles take only one blood meal from their human hosts during their lifetime. Uninfected mosquitoes easily digest the blood and reproduce. Infected ones are incapable of digesting this blood, because their digestive systems are clogged with plasmodia; they bite repeatedly in a vain attempt to get nourishment, infecting humans in the process.10

McKeown makes a more specific error, arguing that “malaria was never an important cause of death in Britain.” Dobson, however, has shown that although the most fatal form of the disease, *Plasmodium falciparum*, was rare in England, *Plasmodium vivax*, which has a case mortality of about 5 percent, was common in the southeastern part of the country until the early twentieth century. Historians now tend to believe that malaria is limited to the tropics and sub-tropics; indeed some 65 percent of malaria cases in India today are *P. vivax*. In the nineteenth century, however, it could be found in southern Norway and England.11


Smallpox also presents problems for historians. Curtin correctly pointed out that smallpox “victims acquire a non-infective lifetime immunity to another attack” and that \textit{Variola major} was endemic among children in Europe. Yet, he errs in accounting for the relative rarity of smallpox in Africa by the continent’s sparse population. The immunity of Africans came from the incidence of \textit{Variola minor}, a milder form of the disease that provided cross-immunity \textit{V. major}. Indeed, Africans in the Sahel protected their children from smallpox by deliberately exposing them to a scab from someone actively infected. Alden and Miller’s observation that “the pox may have been less devastating in Brazil than in Europe” may well reflect the African connection.\textsuperscript{12}

disease across the disciplines

Monographs relating to the history of health care are by no means unique in their propensity to fall out of date. What remains are their theses and the strategies employed to prove them. The work of bench scientists, which usually appears in articles rather than books, is subject to a similar fate. Articles that were once on the cutting edge can still receive attention if their theses, evidentiary bases, and organizing strategies have endured despite changes in the state of knowledge. But do differences of approach between historians and health scientists matter?

\textit{Scientists Writing History} Zinsser characterized \textit{Rats, Lice and History} as a biography of typhus fever. Fundamental to his account is the notion of parasitism—a smaller creature living at the expense of a larger one. Zinsser’s parasitic relationships were the \textit{Rickettsia typhi} to lice and lice to rats and to humans. Zinsser wrote during the heroic age of bacteriology. He believed that if scientists could understand the connection between parasites and vectors and be-

between vectors and organisms, they would be able to eliminate deadly diseases from human populations, as they had eliminated malaria and yellow fever. In fact, typhus has been largely eradicated since World War II.\textsuperscript{13}

Unfortunately, sixty-five years of subsequent investigations have complicated our understanding of the relationship between parasites and hosts. In a manner that totally reverses Zinsser’s notion of parasitism, Levin and Antia suggest that elements of our immune systems act as predators to bacteria attempting to invade them.\textsuperscript{14} But this line of thinking does not destroy the attractiveness of Zinsser’s text. The quality of his writing and the number of literary works that he cited are enough to keep his work attractive after two-thirds of a century.

The same applies to McKeown’s \textit{The Modern Rise of Population}, which attempts to explain the growth of the population of England and Wales between the 1840s and the 1970s. McKeown first eliminates fertility and migration as potential causes by showing that during this period, England underwent net out-migration and that fertility levels declined nearly continuously. He bolsters his argument for the unimportance of fertility by reasoning that in an environment of high infant and child mortality, the number of births would have been offset by the number of deaths at an early age. After only a brief discussion of Thomas Malthus’ doctrine that “the tendency of all animated life is to increase beyond the nourishment provided it,” he concludes “that the modern rise of population was due to a decline of mortality rather than to a removal of restraints on fertility.”\textsuperscript{15}

McKeown approaches the history of mortality decline by establishing four possible causes: medical advances, reduced exposure to infection, improved nutrition, and noninfective conditions. McKeown took his guiding principle from Arthur Conan Doyle, as enunciated by his fictional character Sherlock Holmes, “When we have eliminated the impossible whatever remains, however improbable, must be the truth.” This kind of approach, which does not depend on the verification of a putative cause, tends to raise historians’ hackles. But McKeown employs it to ar-

\textsuperscript{13} Zinsser, \textit{Rats, Lice and History}, 60–62.
\textsuperscript{14} Levin and Antia, “Why We Don’t Get Sick.”
\textsuperscript{15} McKeown, \textit{Modern Rise of Population}, 18–41, 44.
gue that before the introduction of broad spectrum antibiotics in 1935, biomedicine did little to reduce mortality, that the reduction of exposure to infectious disease “came about as a secondary consequence of other influences which lowered the prevalence of diseases in the community,” and that “a substantial part of the [decline in noninfectious disease] resulted from other influences, particularly improvement in nutrition of mothers and children.” McKeown’s conclusion is, “We owe the reduction of mortality and growth of population basically to improved nutrition which resulted from the increase in food and to the change in reproductive behaviour which ensured that the advantage was not reversed.”

Demographers have long been critical of both McKeown’s statistics and his methods. Perhaps the final word comes from Woods, a practicing geographer, who divided the elements of historical demographic regimes into five categories: marriage, children, effective replacement, mortality, and migration. His demographic analysis is more subtle than McKeown’s, and it reaches different conclusions. Taking Malthus much more seriously than McKeown does, Woods begins with a discussion of the two restraints on population increase found in the 1798 and 1803 editions of his Essay on the Principle of Population: the positive check, by which mortality increases (through famine, disease, and war) when food is in short supply; and the preventive check, by which marriage is deferred, thereby reducing fertility, during food shortages. According to Woods, the positive check did not affect England after the sixteenth century, and the preventive check became largely inoperative during the early nineteenth century. Reduced fertility (regarded by McKeown as irrelevant to population growth) interacted with child mortality to create the low mortality characteristic of modern demographic regimes.

Analysis of the causes of mortality decline based on Woods’ indicators produce a different explanation for mortality decline in McKeown’s period. The decline in the late nineteenth century was due, in descending order of importance, to changes in disease patterns, sanitary reforms initiated by the local govern-

16 Ibid., 128, 91-104, 127, 151, 159.
ment, and improvements in diet and medicine. The decline in the twentieth century was due to medical improvements and diet alone.\textsuperscript{18}

Woods’ critique does not deprive McKeown’s book of its status. McKeown’s series of publications extending from 1955 to 1976 reflected the spirit of his times. His work represents not so much the microbiological triumphalism of the pre-World War II era as the developmentalism of the postwar world. First and foremost a physician (epidemiologist), in addition to being a demographer, McKeown was constantly looking for ways to apply his findings about the demographic transformation of Victorian Britain to the problems of the Third World, though he was conscious that conditions were not identical.\textsuperscript{19}

In his last book, \textit{The Origins of Human Disease}, McKeown clings to the generalizations of \textit{The Modern Rise of Population}, holding that “the remarkable growth of population . . . was due essentially to increased food production which led to a reduction of mortality.” He closes with an optimistic picture of health: “Except when determined at or soon after fertilization disease is not an inescapable attribute of the human condition; it results from unhealthy ways of life and can be prevented if those ways can be changed. The deleterious influences are of two kinds: deficiencies of basic resources, of which food is by far the most important; and exposure to hazards . . . natural, mainly from parasites, or man-made from conditions that man created for himself.” His reformist stand continued to permeate his writing. “Poverty is not a direct cause of disease, but it is the main determinant of influences that lead to disease. . . . [Lack of food and environmental hazards] are still the major causes of ill health in the Third World and in the poor of technologically advanced countries.”\textsuperscript{20}

The two more recent works by Wills and Diamond also have their literary charm and political engagement, but their manner of presenting evidence is altogether different. In \textit{Yellow Fever, Black Goddess}, Wills addresses the question of how human activities over the past 3.2 million years have led to disturbances in the ecological balance, thus giving rise to great epidemics, or in his terms, “plagues” (the British version of the book is titled \textit{Plagues: Their

\textsuperscript{18} Ibid., 27, 359.

\textsuperscript{19} McKeown, \textit{Modern Rise of Population}, 42, 117, 135, 158.

\textsuperscript{20} McKeown, \textit{Origins of Human Disease}, 61, 216, 128.
Origins, History and Future). His approach is both evolutionary and demographic, in that he uses population density to explain speciation among microorganisms. Most biological parasites that manifest themselves as diseases (in plants and animals as well as humans) adapt to a particular species. “Because each pathogen must concentrate its efforts on overcoming the defenses of a particular type of host, pathogens inevitably become more and more specialized and thus they, too, become more and more different from each other. Plagues emerge “when a population of any species . . . becomes too large for its resources. . . . [W]hen the population of hosts happens to explode in numbers and to modify its environment in the process, the most virulent pathogens can suddenly gain a brief advantage. . . . After the plague, the most virulent strains disappear or fall to low levels, and the host species and its pathogens soon revert to their previous uneasy balance.”21

The triggering mechanism for a plague occurs in the “penumbra” of the normally harmless organisms that surround plants or animals; under conditions of crowding, a previously innocuous microbe can develop into a plague. This phenomenon cannot be explained by the common cladistic view of bacterial evolution according to which bacteria, like larger organisms, mutate one gene at a time. During the late 1990s, however, microbiologists discovered lateral transfer, which enables viruses and bacteria to acquire genetic materials immediately and pass them on to their descendants. This amounts to Lamarckian, as opposed to Darwinian, evolution. As Wills puts it, “In acquiring these genes, our pathogens have been handed tools that they are able to make deadly use of in emergency short-term situations.”22

This microbiological discovery accords well with the ecological concerns of the late twentieth century: the dangers of overpopulation and the constant threat of unforeseen disaster. The problem is to identify the limits that any given human environment can support. Although the threat of overpopulation through an increase in the number of births is now less likely, since fertility has declined in every region of the world except rural sub-Saharan

Wills, Yellow Fever, Black Goddess, 9, 23, 27.
Africa and southwest Asia, Wills’ good-hearted account stands as a worthy effort to communicate scientific concerns across disciplines.23

By contrast, Diamond’s best-selling *Guns, Germs, and Steel* aims at explaining the macropolitical status quo. Diamond asks, “Why did wealth and power become distributed as they now are, rather than in some other way?” His explanation involves “the immediate factors that enabled Europeans to kill or conquer other peoples—especially European guns, infectious diseases, steel tools, and manufactured products.” These factors contributing to European superiority turned on “continental differences in domesticable plants and animals, germs, times of settlement, orientation of continental axes, and ecological barriers.” Diamond’s point is that western Eurasia benefited from a unique configuration of geographical conditions that conferred on its inhabitants plentiful wild plants and animals for food, early exposure to pathogens specializing in human hosts, and effective long-distance trade routes the likes of which were unavailable in other parts of the world.24

Diamond, whose research interests lie in molecular physiology, evolutionary biology, and biogeography, justifies his explanation as the result of “a natural experiment,” achieved “by comparing systems differing in the presence or absence (or in the strong or weak effect) of some putative causative factor.” He grants that “natural experiments in any field . . . are inherently open to potential methodological criticisms. Those include confounding effects of natural variation in additional variables beside the one of interest, as well as problems in inferring chains of causation from observed correlations between variables.”25

At first glance, Diamond’s work seems cautiously quantitative, but he paints the huge canvas of human history with a broad brush. Early in his study, he identifies “one of the key factors in world history: diseases transmitted to peoples lacking immunity by invading peoples with considerable immunity.” An immunologist

25 Ibid., 27, 424, 425.
might call this factor leukocytic and lymphocytic determinism. The case of the American Indians peppers his narrative. His strongest statement is, “The main killers were Old World germs to which Indians had never been exposed, and against which they had neither immune nor genetic resistance. Smallpox, measles, influenza, and typhus competed for top rank among the killers.”

Diamond’s position receives at least partial support from Black, an epidemiologist, who has shown that American Indians possess only half of the HLA antigens of Old World Peoples and that their resistance to disease is further impaired by genetic homogeneity. Nonetheless, comparing the reaction of Native Americans and Old World peoples to vaccines, Black also maintains, “In no instance was the level of induced antibody [in Native Americans] inferior to that usually observed elsewhere.”

In that case, how important was the introduction of Old World diseases to the conquest of the New World? Diamond takes a maximalist position: “Throughout the Americas, diseases introduced with Europeans spread from tribe to tribe far in advance of Europeans themselves, killing an estimated 95 percent of the pre-Columbian Native American Population.” Henige associates this sort of historical judgment with a group of historians whom he pejoratively calls the high counters. Henige rightly contends that the high counters do not have adequate evidence to prove the occurrence of devastating epidemics, but he does not discuss other conditions that might have led to widespread death in the New World.

In the authoritative new anthology, *A Population History of North America*, Thornton adopts a more moderate position: “It cannot be concluded that significant depopulation occurred early in the sixteenth century or that it reached pan-regional proportions during the sixteenth century.” He lists a number of non-disease factors that he also deems responsible for diminishing American Indian populations: “wars and genocide, enslavements,

26 Ibid., 77, 78, 196, 197.
removals and relocations, and changes in American Indian societies, cultures, and subsistence patterns accompanying European colonialism."

According to Thornton, the devastation of the Americas proceeded not so much from impersonal geographical and biological causes as from human conquest, a consideration that does not play a large part in Diamond’s account. Might this factor not also have been dominant in other parts of the colonized world, or even in the industrial and political development of the colonizing nations of Europe? Diamond’s “natural experiment” runs the risk of excluding human initiative from the sweep of human history.

Diamond’s discussion of the possibility that tuberculosis occurred in the New World before the voyages of Columbus also raises questions: “Tuberculosis DNA has been reported from the mummy of a Peruvian Indian who died 1,000 years ago, but the identification procedure used did not distinguish between human tuberculosis from a closely related pathogen *Mycobacterium bovis*.” Diamond’s point in this passage is that since the mummy might have had *M. bovis* rather than *M. tuberculosis*, the evidence for the latter before Columbus’ appearance is still lacking. In his endnotes, however, he cites an article contradicting his own position. Furthermore, a closer look at the medical literature would have revealed that *Mycobacterium bovis* causes extrapulmonary tuberculosis, a serious disease in its own right. This apparent flaw and the one preceding it do not, of themselves, justify discounting Diamond’s entire book, but they raise doubts about the framing of Diamond’s “natural experiment.”

The work of the four natural scientists examined above demonstrates the broad variety of methodological, and even political, approaches that even a hard science framework can accommodate. How does it differ, if at all, from work on the same subjects by historians?

*Historians Writing Science* McNeill’s *Plagues and Peoples* has served as a beacon for historians of human disease for a quarter of a thousand years.


century. It is cited extensively by non-historians such as Diamond. Its framework links encounters between humans and microorganisms to encounters among humans by means of a powerful metaphor: McNeill portrays “most human lives as caught in a precarious equilibrium between the micro-parasitism of disease organisms and the macro-parasitism of large-bodied predators. . . . A conqueror could seize food from those who produced it, and by consuming it himself become a parasite of a new sort on those who did the work.”

Later in the book, his link between the two varieties of predation is even more explicit: “civilized history has characteristically exhibited sharp fluctuations up and down, as periods of peace and prosperity induced population growth in excess of macroparasitic powers of absorption (i.e., destruction); whereupon an increase in death rates asserted itself through a breakdown of public order . . . [which] could always be counted on to reduce population catastrophically whenever less drastic regulators of peasant numbers failed to maintain a satisfactory balance.”

In this passage, McNeill appears to be endorsing Malthus’ positive check (population destruction by famine, disease, and war) as well as his preventive check (less drastic regulation of population by deferred marriage). His metaphorical framework can be approached through two questions: (1) Can human domination be equated with macro-parasitism? (2) Did micro-parasitism produce major shifts in world history? As far as macro-parasitism is concerned, McNeill views empire building as a zero-sum game. His position on India is that “(m)ore going to one kind of parasite leaves less for others,” a proposition that he also applies to China and the Mediterranean. He refers to this phenomenon as “the simple polarity of older ages, whereby human societies were divided between food producers and those who preyed upon them.” This explanation harks back to Zinsser’s account of parasitism. But scientists now prefer explanations of immunology based on biological equilibrium that make parasites and hosts, let alone relative advantages, difficult to distinguish.

Corresponding difficulties can be found in McNeill’s account of micro-parasitism—“how varying patterns of disease circulation have affected human affairs.” After describing pre-agricultural
times and the neolithic revolution, McNeill arrives at a period in disease history that coincides with the rise of the four global ecumenes (macroregions with sufficient communications to foster biological, cultural, and trade relations, though not necessarily with political connections) in his Rise of the West: “By 500 B.C. different micro- and macroparasitic balances had established themselves in each civilized region of Eurasia, and unstable accommodations between human hosts and the new civilized diseases had begun to manifest themselves in some and probably all of the major civilized centers.” His next major landmark corresponds to the closing of the global ecumene in 1500: “The result of such systematic lightening of the microparasitic drain upon European populations (especially in northwestern Europe where both plague and malaria had about disappeared by the close of the seventeenth century) was, of course, to unleash the possibility of systematic growth. On the time scale of world history, indeed we should view the ‘domestication’ of epidemic disease that occurred between 1300 and 1700 as a fundamental breakthrough.”

McNeill’s is an unreasonably optimistic interpretation of those four centuries. Livi-Bacci, in his general history of population, refers to the fourteenth and early fifteenth century as “a devastating and long-term catastrophe” from which European population would not reach pre-crisis levels until the mid-sixteenth century. Moreover, as plague began to decline in importance, typhus, syphilis, and smallpox replaced it—hardly a domestication of epidemic disease. McNeill’s disease history of Europe for this period would appear to have been forced into his overarching view of world history. McNeill’s distinction between micro- and macroparasitism has not survived beyond the historical discipline. Anderson and May, in their massive survey of human infectious disease, define microparasites as those organisms that reproduce themselves inside their hosts and macroparasites as those that reproduce themselves outside them. That McNeill’s generalizations concerning both macroparasitism and microparasitism

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34 Ibid., 4, 69, 198
are no longer valid in a general sense does not completely nullify the utility of *Plagues and Peoples*. This cogently written and copiously documented work has introduced more than a generation of historians to the study of disease. Students in the future, however, should be advised that not all of its biological assumptions are current.

The other historical monographs under consideration constitute the first two volumes of a projected trilogy by Curtin. They serve as a social science counterpart to McNeill’s humanistic framework. Each is written to address a specific historical question with a specific thesis bolstered by a broad range of primary and secondary sources. The only problem is that Curtin encountered unanticipated results.

*Death by Migration* is Curtin’s history of nineteenth-century “relocation costs”—“the ratio between mortality levels for British and French soldiers in the tropics and those at home.” Military death rates in the tropics were roughly twice those of soldiers in Europe at the beginning of the century; this ratio was retained for the rest of the century, though mortality levels declined substantially in both regions as a result of concerted efforts to enhance survival. In his conclusion, Curtin admits that his findings surprised him: He did not expect the greatest drop in absolute death rates between the 1840s and 1860s to come from empirical rather than scientific measures, nor for relative relocation costs to remain stable until World War I.  

In the body of his monograph, Curtin discusses major regional differences in the causes of soldiers’ deaths. In Britain, most deaths came from lung disease; in the West Indies, from tropical fevers; and in South India, from diarrheal diseases. In his context, Curtin faces a paradox similar to the one that confronted McKeown: British mortality from tuberculosis declined without substantial medical intervention. Furthermore, British innovations in the tropics—“moving troops into the highlands to escape malaria, moving them under canvas away from barracks and cities to escape cholera and yellow fever, [and] improving the water supply”—strike him as nonscientific: “Each remedy fell short of its potential, because the medical officers had no way of knowing why these empirical safeguards worked.” He states that the “broad

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37 Curtin, *Death by Migration*, xiii, 159-160.
pattern of shifting relocation costs began as a fact of nature. It was only slightly influenced by human interventions, either positive or negative.” This complaint implies that, of all human initiatives, only biomedicine can reduce death rates. During this period, however, mortality levels fell precisely because of the commonsense innovations of British medical officers, even though they had no formal scientific rationale.  

Curtin’s second book in this series, Disease and Empire, addresses a different issue, “the relation between disease and imperialism in the European conquest of Africa [,] the disease cost of that conquest, and the way policy-makers reacted to it.” Rejecting contemporary historians who see colonial medicine as the handmaiden of imperialism, he concludes “that the conquest of Africa did far more to bring about the improvement of tropical medicine than the improvements in tropical medicine caused the conquest of Africa.” He argues that “European expeditionary armies profited . . . little from the new medical knowledge,” explaining that medical education even in the late nineteenth century stressed cure rather than prevention and that military officers, as well as politicians, subordinated health care to victory in the field.  

Curtin rightly attributes public evaluation of military medicine more to perception than to fact. The expeditions to Magdala in Ethiopia (1867–1868) and Kumasi in Asante [Ghana] (1874) “depended more on luck than on medical skill or on foresight”; the River War in the southern Sudan (1898) was praised despite heavy losses; and the South African campaign (1899–1902) was condemned despite half the casualty rate. Curtin portrays as tragic “that European leaders could sacrifice so many lives for such an insignificant national advantage. . . . plac(ing) a low value on the life of their own working class, and a lower value still on the lives of foreigners and colonial subjects.” In this book, as in Death by Migration, Curtin undervalues the empirical measures taken by European armies to improve the health of their troops: “ventilation, cleanliness, and quinine that cut the mortality rates of British and French soldiers serving at home and abroad by 50 to 75 percent between the 1840s and 1860s.” Even if such measures did not constitute the cutting edge of medicine or hygiene, they did save

39 Ibid., Disease and Empire, xi, 227, 230.
lives. Curtin’s work betrays a nostalgia for the certainties of medicine in an earlier generation, when science was science and parasites were parasites. Despite the value and the influence of his work, Curtin, like other authors of the classics in health history, must bear the mark of his times.40

The organizational strategies of the researchers discussed above, whether bench scientists or historians, range from the metaphorical to the biographical to the reformist to the social-scientific. Disciplinary background seems to have little to do with their outlook. The history of human health has excited the imagination of scholars displaying a wide range of temperament.

What distinguishes historians writing about health from health scientists writing history, however, are their evidentiary bases. Historians extract their evidence primarily from verbal, though sometimes, as in the case of Curtin, quantitative databases, whereas health scientists ultimately depend on reproducible experiments involving physical evidence or carefully documented, ideally quantitative, epidemiological observations. This difference in evidentiary base has left historians at a disadvantage when compared to their bench scientist colleagues. But how can historians tap the scientific literature without becoming scientists themselves? The short answer is that they can, and should, enlist the collaboration of colleagues in the health sciences before embarking on their research projects. But will they do it?

And for that matter, how can historians persuade health scientists to respect the canon of historical writing? Emboldened by the certainties of the laboratory and the rigor of experimental design, they are capable of ignoring the fragmentary, the verbal, and the contextual. As this analysis has shown, not even the best of them are always careful in how they construct their arguments. The study of human health requires a joint effort on the part of scientists and historians; neither side has an advantage in method or truth over the other.

But just how many medical-history researchers will incorporate new methods into their work? If the adaptation of new forms of evidence in other fields is any predictor, the answer must be that some will, but the majority will not—the clientele of this

40 Ibid., 229, 220–222, 228, 31.
journal notwithstanding. As cases in point, not all accounts of nonliterate peoples discuss oral traditions; relatively few historians of the last 2,000 years incorporate arguments based on graphic evidence from iconography or the history of cartography; and many social historians publish without recourse to statistical data. The history of medicine will undoubtedly continue to offer scholars outlets for, say, discussions of ancient healing practices, speculations about ancient plagues, or postmodern glosses of the physician’s gaze. More substantial authors, however, will frame their questions and develop their arguments in the company of bench scientists.