The Conceptualization of Influenza in Eighteenth-Century Britain: Specificity and Contagion

MARGARET DELACY

The relationships between the definition of a disease, its presumed etiology, and the broader development of epidemiology are constantly evolving as each element both affects and is affected by the other two. In the case of influenza, the refinement of these concepts to a point where they could begin to yield useful results was an eighteenth-century achievement, and it was one that was deliberately sought through collective investigations. These defined influenza as a distinct disease for the first time, charted its incidence and spread, and raised the possibility that the disease was contagious. This article will describe

*This paper was presented at the sixty-third annual meeting of the American Association for the History of Medicine, Baltimore, Maryland, 12 May 1990. The research for this paper was aided by a grant for 1989-92 from the National Endowment for the Humanities, an independent federal agency. I thank Patricia Mercer, Good Samaritan Hospital, Portland, Ore.; Susan Goode, Influenza Unit, Centers for Disease Control, Atlanta, Ga.; and Stephen Modesitt, H.I.V. Program, Oregon State Health Division, Portland, Ore. I also thank John DeLacy for technical assistance. For providing material, I thank Geoffrey Davenport, Royal College of Physicians; Vivienne Orr, Nithsdale District Council, Dumfries; Barbara Cantwell and the Multnomah County (Oregon) Library Interlibrary Loan Department; Gary Sampson, Portland State University Library; the Oregon Health Sciences University Library; the Reed College Library; Nancy Austin, Lane Medical Library, Stanford University Medical School; John Symons, Wellcome Institute for the History of Medicine; the Welch Library of Johns Hopkins University; and the National Library of Medicine. For advice on the argument, I thank Sir Christopher Booth, Harold Cook, Anne Digby, Elizabeth Eisenstein, Norma Landau, Irvine Loudon, Guil Pat Parsons, K. David Patterson, and the reviewers for the Bulletin of the History of Medicine.


2. Beveridge, Influenza (n. 1), pp. 27-28, and Patterson, Pandemic Influenza (n. 1), p. 1. See also K. David Patterson and Gerald F. Pyle, "The Geography and Mortality of the 1918 Influenza Pandemic," Bull. Hist. Med., 1991, 65: 4–51. Modern use of the words pandemic and epidemic is regulated with some precision. Resistance to influenza strains is affected by the nature of the hemagglutinin (H) and neuraminidase (N) glycoproteins that form the shell of the virus. When mutations take place within the genes for these proteins (genetic drift), new "epidemics" may occur. When the genes for these proteins recombine (genetic shift), host immunity is lower, the disease is more severe and widespread, and pandemics occur. Influenza epidemics are declared to exist when the health authorities who maintain surveillance throughout the world determine that in a sample of communities the deaths from pneumonia and influenza combined have surpassed a set "threshold level" that is based on a seasonally adjusted composite of previous years. These epidemics are only considered pandemics when the virus itself reveals genetic shift in the H and/or N components. Obviously, this cannot be determined for outbreaks in the past, but historians have attempted to determine whether earlier epidemics were true "pandemics" by studying the nature, timing, and spread...
ertheless, some were quite fatal. In Edinburgh, according to a contemporary account, the number of burials doubled during the pandemic of 1732–33. In London, the death rate doubled during the worst weeks of the epidemic of 1729–30, tripled in 1732–33 and 1743, and quadrupled in 1830–33. The influenza epidemic of 1847–48 caused more deaths than the great cholera epidemic of 1832.3

Like malaria, which literally means “bad air,” the word influenza suggests an etiology. The disease was thought to result from the “influence” of the stars or of the heavens—an idea that has enjoyed frequent revivals.4 Although the term influenza was current in early modern Europe, the disease itself was confused with many others, especially with colds, agues, and other fevers, and it was called by many other names. According to F. G. Crookshank, influenza was introduced into English as the name for a specific disease in 1743, but Creighton dates the term to 1730.5

It would appear that John Huxham introduced the term into professional English in his Essay on Fevers of 1750, which referred to “the catarhal Fever, which spread through all Europe under the Name of Influenza in the Spring, 1743.”6 By the epidemic of 1775 the term was becoming common, and in the epidemic of 1782 it became the usual name for the disease.7 As we shall see, its adoption shows that the concept of influenza had become a useful diagnostic tool for isolating a distinctive grouping of symptoms for analysis. Once doctors agreed that they were seeing the same disease, they became more interested in comparing observations of it and treatments for it.

The investigation of influenza rested on changing conceptions of the etiology of all acute diseases, which in turn affected the way that diseases were classified and conceptualized. Traditional (“Galenic”) disease theory did not encourage the grouping of cases of disease for analysis, because this theory depicted illness as the result of an internal derangement of one of the four humors that circulated through the body. A Galenic physician saw each case of disease as different because it was in a different patient: disease was a process, not an object.8 As the medical historian Walter Pagel noted, “The humoral theory was not concerned with diseases as ‘objects,’ but with disease as the ex-

5. F. G. Crookshank, “The Name and Names of Influenza,” in Crookshank, ed., Influenza (n. 1), p. 67. Crookshank claims that the note in which Creighton gives this citation, in History of Epidemics (n. 1), p. 545, was a misprint that left a mistaken impression. However, Creighton repeats the same information on p. 362.

6. John Huxham, An Essay on Fevers, 3d ed. (London: J. Hinton, 1757; reprint, Canton, Mass.: Science History Publications, 1988), p. 11. Influenza also appears as a term in the index. The first edition was published in 1750. See also British Library (BL), Dr. Andrew DuCarel to the Rev. [Philip] Morant, 8 May 1762, Stowe MS. 796 f. 61: “[I have been much out of order with this fashionable cold . . . This distemper is not only epidemic here, but almost universally so in Europe. We have certain accounts that it is got into all parts of Germany, France, Spain, Portugal and Italy, where it is called La Influenza.”


pression of a certain relationship between the objects, namely the
humors and their qualities.” Another historian has commented,
“Within this way of seeing patients and conceptualizing fever, there
was no room for a long-term programme of ‘research’ for . . . every
case was by definition different from any other case.”

“Galenic” physicians believed that fevers were caused by a prior
dysfunction within the body. Indeed, during the Restoration period
many physicians felt that this view justified their claim to provide
superior care: they based their treatment on a thorough knowledge
of each patient and of his or her manner of living and “constitution.”
Such extended personal attention from highly trained professionals
was extremely expensive. Physicians’ claim to preeminence was threat-
ened by the encroachments of surgeons, apothecaries, and quacks.
Surgeons were only allowed to offer external treatment, but the
boundary between internal and external was contested; for example,
surgeons treated venereal disease. They also claimed that their treat-
ments (such as clysters, ointments, and venesection) sufficed for many
internal ailments. Apothecaries and quacks sold “specifics” supposedly
tailored to individual diseases. These remedies required no knowledge
of the patient. Physicians, however, argued that only they could attain
an intimate understanding of the peculiarities of each individual con-
stitution and thus amend the true—humoral—roots of disease through
the administration of internal remedies and the monitoring of the
patient’s regimen. Disease theory was always intertwined with med-
ical politics.

Sydenham and the Development of Neo-Hippocratic
Disease Theory

By the middle of the seventeenth century, however, many physicians
were seeking new ways to approach the problem of obtaining medical
knowledge. The methods they tried included studying diseases as they
occurred among groups of patients. Gradually, during the next cen-
tury, a new sort of medical report replaced the extremely detailed
description of a single case. The new reports contained systematic
summaries of selected salient points in several cases—for example,
the duration of the disease, the various therapies used, the date of
onset and termination of the epidemic in a particular area, the ages
of the patients, and the occurrence of complications. This sort of
report filled an intermediate position between the discussion of a single
case and the use of Bills of Mortality, censuses, military records, and
other such statistical sources of health information in which only one
or two facts were recorded for each individual.

Several scholars have traced the inspiration for such research to
the work of the medical reformer Thomas Sydenham, who became
interested in the problem of epidemic diseases. Seeking to explain
how many people could simultaneously exhibit identical symptoms,
Sydenham developed an etiology that depended on external factors:
he studied the disease and not the patient. This approach goes back
to classical times and to the Hippocratic treatise *Airs, Waters, and Places*,
but Sydenham gave it a new emphasis. He had developed what medical
historians call an “ontological” approach to disease: he hoped to write
the “natural history of diseases” and to classify them in the same way
that botanists classified plants.

Together with the chemist Robert Boyle, Sydenham developed the
theory that epidemics appeared when invisible emanations from the
bowels of the earth polluted the atmosphere. These imperceptible
particles created each year’s “epidemic constitution,” which governed
all the epidemics that occurred in that year, such as the appearance
of “epidemic coughs” (influenza) in 1675 and 1679. These emanations,
however, also interacted with the perceptible physical qualities of the
atmosphere and season. These qualities were heat, cold, moisture, and
dryness. According to this “neo-Hippocratic” theory that Sydenham
helped popularize, atmospheric qualities affected the humors of the
body, which were also hot, cold, moist, or dry.

9. Walter Pagel, *Joan Baptista Van Helmont, Reformer of Science and Medicine* (Cam-
11. See the works by Cook cited in n. 8, above; and Philip K. Wilson, *The Art of
Surgery in Early Eighteenth-Century London: Textual Analysis and Professional Con-
cerns* (Paper presented at the meeting of the American Association for the History
of Medicine, Cleveland, Ohio, 1994). I would like to thank Philip Wilson for providing
me with a copy of this paper. In this article, “physicians” refers to medical practitioners
holding an M.D. or M.B. degree, or to an audience that is presumed to consist primarily
of M.D. or M.B. holders. The term doctors, however, is used to apply to any regularly
trained medical practitioner and does not imply possession of an advanced degree.
Most surgeons and apothecaries learned their trade through apprenticeship, although
many had attended universities.

13. Ibid., pp. 180–83. On Sydenham’s disease theory, see also R. R. Trail, *Syden-
*Dr. Thomas Sydenham, 1624–1689: His Life and Original Writings* (Berkeley: University
Conquest of Epidemic Disease: A Chapter in the History of Ideas* (1943; reprint, Madison:
University of Wisconsin Press, 1980), chap. 9, pp. 161–75.
The idea that external atmospheric factors caused disease inspired Sydenham's friends in the Royal Society to gather information on the climate and characteristic diseases of widely separated geographic areas. In 1666 Henry Oldenburg, secretary to the society, published "General heads for a natural history of a country" in the society's Philosophical Transactions and obtained responses from correspondents addressing these subjects. In the same year, Boyle published a paper on meteorology arguing for the same connection. In 1692 the physician and philosopher John Locke, a great proponent of Sydenham's work, sent out questionnaires to physicians overseas, inquiring about death rates, weather, and morbidity patterns in their localities. The encouragement of the society elicited from travelers many accounts of the effect of climate on health.

The Royal Society's interest in this subject continued into the next century. In 1723 James Jurin, secretary to the society, appealed to readers for precise meteorological observations that could be collated and compared with the prevalence of disease. This appeal led to the publication of a number of works that sought to study the relationship between climate and epidemics by systematically recording both the weather and the illnesses that occurred in one place over many years. Most of our information about influenza in the first half of the eighteenth century comes from these works. In accord with their general approach, most of these authors attributed episodes of influenza to the weather. For example, in his Essay concerning the Effects of Air on Human Bodies, the Scottish mathematician John Arbuthnot attributed the epidemic of 1732-33 to "the perspiration of the ground." Similarly, the neo-Hippocratic physician John Huxham conducted a prolonged investigation of the interaction between the air and the epidemic diseases of Plymouth. He blamed "a thick, damp, chilly disposition" during the winter for the influenza epidemic of 1737.

Sydenham's theory of the "epidemic constitution" focused interest on the external causes of disease, and therefore the aspects of illness that remained the same from patient to patient, but by the same token it discouraged efforts to distinguish between diseases. The focus of the early investigations was more on how much disease there was altogether in a certain place and time than on what different diseases were present. If the atmosphere itself caused disease, then everyone who breathed the same air must have the same disease, its manifestations affected only by individual differences in constitution. Conversely, if the weather or the wind changed, so would the disease. Huxham, for example, believed that influenza could transform itself into malaria, "the Difference of the Constitutions of the Patients, &c. thus altering the Face and Nature of the Disease."

According to this theory, although one disease could become another, each "disease" remained specific to its time, place, and patient; it was the result of a particular concatenation of causes. A similar combination at another time or place might induce similar symptoms, but there was no actual physical link between one episode and another. Huxham's classification was based on constitution and regimen, but he considered any classification to be of secondary importance. What really counted was the way that disease manifested itself in a given individual: "a Disease is a Disorder in the animal Economy, distinguished indeed by such and such particular Symptoms, and called by such or such a Name; but each particular Disease, in every individual Patient, is to be considered . . . not according to the Nomenclature, but according to the Nature, Causes and Symptoms of the particular Disease in that particular Person."

The Rise of Contagionism

During the second half of the eighteenth century physicians continued to pursue epidemiologic investigations, but the focus of their efforts gradually shifted from long-term investigations of the climate and diseases of a single area, to studies of episodic epidemics of individual diseases. Certain investigators began to relegate climate and atmosphere to a secondary role and focused their arguments on the hypothesis of direct infection from specific disease-causing particles.

This change in focus stemmed from the introduction of inoculation in the 1720s, which had led practitioners to see smallpox as a distinct
disease: smallpox inoculation induced smallpox, not chicken pox or measles, and a case of smallpox conferred immunity to further infections of the same disease but not to other illnesses. Each new case of smallpox resembled other cases in its symptoms, although there was some variation in severity and complications. Smallpox matter could easily be carried in a box, and when it was introduced into a new area it continued to cause the same symptoms regardless of local conditions. Inoculation, therefore, helped doctors to sort out the different eruptive diseases, and eventually many of them came to see fever as the symptom of a number of different diseases, rather than as a disease or "dis-order" in itself.22

Increasingly, contagionism began to compete with theories that had depended either on the state of the weather or on an imperceptible corruption of the air.23 Like atmospheric explanations, contagionist theories laid the primary blame for disease on causes external to the body: in the early eighteenth century, some medical writers considered contagion to be merely another form of atmospheric pollution. According to this formulation, disease-causing particles that floated in the air might come either from the air itself or from exhalations from the bodies of those already ill.24

By the second half of the century, many medical writers adopted this argument but changed its emphasis. They depicted direct infection from specific disease-causing particles as the primary factor in the generation of some diseases, relegating air and constitution to a secondary role.25 As new "fevers" were identified, doctors began to ask whether these were "pestilential" (spread only by specific contagious particles from case to case) or "common" (the result of a more generalized environmental contamination or atmospheric change).

Instead of investigating the interaction of climate and disease in a single locality, doctors became interested in discovering how diseases spread from one place to another and whether they exhibited the same symptoms in each place. Epidemiologic investigation tried to settle this question. By noting the pattern of epidemics' spread, investigators hoped to determine whether disease was passed only from person to person or whether it appeared "spontaneously" in many individuals in a community at once because of a diffuse poisoning of the air, of food, or of the water supply. Such investigation could only be done, however, after there was some measure of agreement that a particular group of symptoms did, in fact, constitute a recognizable species of "disease."

One turning point in the development of contagionism in the second half of the century was the publication of An Account of the sore Throat Attended with Ulcers by the Quaker physician John Fothergill in 1748. This essay described an epidemic of what was probably scarlet fever and attributed the epidemic to a specific contagion, a "miasma sui generis."26 In 1775 Fothergill would sponsor an important study of influenza which included the first letter claiming that the disease was contagious. However, the work on the transmission of smallpox carried out in the final quarter of the century by Fothergill's friend John Haygarth, of Chester, was probably the single most important factor in inducing a significant number of physicians to entertain the hypothesis of strict contagion for a wide range of fevers.27

In 1778 Haygarth published a paper arguing that typhus was contagious and recommending the removal of the disease's victims to separate fever wards. In 1777 he had begun a systematic investigation of smallpox, which became the subject of his major work, published in 1784. In that book he argued that smallpox spread solely by contagion; air transmitted the disease over only a very short distance. He claimed that the transmission of smallpox could be interrupted by isolating patients and by maintaining strict cleanliness to prevent at-


25. Ibid.


tendants from conveying any contagious matter out of the house.\textsuperscript{28}

The importance of Haygarth’s work on smallpox was not that it established that smallpox was contagious, for that had been known for many years. It was his insistence on contagion as a necessary, not merely a sufficient, cause.\textsuperscript{29} He used probability theory and patients’ reports to show that anyone who contracted smallpox must have encountered a carrier. Control the carriers, he argued, and one could control the disease. Whereas medical authors at the middle of the century depicted contagion as a part of atmospheric transmission, Haygarth eliminated the atmosphere as a general cause, contending that new cases could only arise from preexisting cases. By 1780 he had begun to apply this expertise to other fevers, including influenza, and to argue that they, too, were contagious and could be controlled by the isolation of patients, the control of contacts, and careful hygiene.\textsuperscript{30} Haygarth’s work, however, remained controversial, and in the case of influenza his contentions were never accepted by a clear majority of the profession. This was partly because the nature of the disease itself made it difficult to obtain unambiguous evidence of contagion.

Difficulties Caused by the Nature of Influenza

Research into influenza thus formed part of the larger debate about the nature and causes of epidemic fevers. In the seventeenth century, influenza was still confused with so many other diseases and was called by so many different names that it was nearly impossible for doctors to compare notes, or to develop a useful etiology. The nature of the disease itself also hindered efforts to uncover a clear pattern of incidence. The symptoms of influenza in individuals were more subtle than those of a disease such as smallpox, and thus the incidence of influenza was more difficult to track and analyze. Patients with mild cases hardly ever consulted practitioners. On the other hand, when an influenza patient died, the parish register often attributed the death to an ensuing disease, such as pneumonia. This made it very difficult to use the usual mortality figures as a guide to the overall incidence of influenza. Unsure of both the total number of cases of influenza and the total number of deaths it caused, physicians were often unable to establish even approximate case-fatality rates. They estimated the severity of epidemics by discussing the overall change in death rates during epidemic weeks, supplemented by the impressions they drew from their own practices.

Eighteenth-century doctors were well aware that most of the deaths that occurred during an influenza epidemic were due not to the disease itself but to other diseases that followed in its wake. Huxham commented that in the epidemic of 1733 influenza was rarely fatal unless patients “[made] too slight of it, either on account of its being so common, or not thinking it very dangerous, [and] often found asthmas, hectic, or even consumptions themselves, the forfeitures of their inconsiderate rashness.”\textsuperscript{31} The Royal College of Physicians noted in 1782 that although “it has been observed . . . that this disease was not in itself fatal, and that few could be said to have died but those who were old, asthmatic, or . . . debilitated by some previous indisposition. . . . the great increase in the burials after the disease had appeared about three weeks . . . is very striking.” The College reported that London burials rose from 299 for the week ending 7 May, to 560 for the week ending 11 June.\textsuperscript{32}

Influenza often appeared without apparent warning; we may now presume that it was introduced by animals or asymptomatic carriers.\textsuperscript{33} Sometimes it exploded in a community, with many cases appearing at once rather than in succession. Moreover, in cases when many people living in a household or an institution were known to have

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32. “An Account of the Epidemic Disease, called the Influenza, of the Year 1782, collected from the Observations of several Physicians in London and in the Country, by a Committee of the Fellows of the Royal College of Physicians in London,” reprinted from Medical Transactions, 1785, in Thompson, p. 163.
33. Interspecies transmission is known to occur in nature but is poorly documented. Pigs, horses, and birds, especially feral ducks, are all potential carriers. See Beveridge, Influenza (n. 1), for a comprehensive discussion of avian transmission, and the essays in Beare, Basic and Applied Influenza Research (n. 1), especially G. R. Noble, “Epidemiological and Clinical Aspects of Influenza,” pp. 11–50; and V. S. Hinshaw and R. G. Webster, “The Natural History of Influenza Viruses,” pp. 80–104, for a summary of current “mainstream” views of the epidemiology of outbreaks.
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been exposed to a patient with the illness, a significant proportion seemed to escape the disease entirely.

It was evident that one attack of influenza did not confer lifelong immunity to further attacks of the disease, and thus it was difficult to explain why some of those who were exposed became ill and others did not. At the same time, many of those who did become ill could not trace their disease back to a previous case. Finally, it was difficult to distinguish mild influenza from a number of other diseases that might also be circulating at the same time. Even at the end of the century, many practitioners refused to distinguish between influenza and severe colds.

Influenza Research in the Early Years of the Eighteenth Century

As noted above, most early discussions of influenza occurred in the course of more general, "neo-Hippocratic" discussions of the climate and diseases of a given geographic area. Examples of this genre are the works of Arbuthnot on the effects of air on human bodies, and the works of the Quaker physician William Hillary, who referred to influenza in his analysis of the variations of the weather and epidemic diseases in Ripon between 1726 and 1734. There were also, however, three separate treatments of the disease before the middle of the century: a book entitled *De Febre Britannica* (1713), by an otherwise unknown London apothecary named John Turner, describing an epidemic in the previous year; a book by an anonymous author on the epidemic of 1729; and a work that has apparently not been consulted by other historians of influenza, a manuscript account entitled "Histories of the Epidemic[sic] Colds which happened in the Years 1729 and 1732/3 . . ." by a London apothecary and a Fellow of the Royal Society named John Chandler. Chandler's work is of interest to the history of statistics as well as that of epidemiology because of his innovative efforts to glean information from the recalcitrant Bills of Mortality.

Like the authors who followed him, Chandler was fully aware that the causes of death given in the Bills of Mortality were unreliable and that in any case deaths that had been precipitated by influenza were usually ascribed to some intervening disease such as pneumonia, or to some catch-all diagnosis such as "old age," if the victim was elderly, or "fits" or "convulsions," if the victim was a young child. Nevertheless, he noted that the number of deaths rose from 531 for the week ending 16 January 1782, to 1588 on 30 January, before declining to 628 on 13 February. Chandler then compared the increases in deaths ascribed to particular causes with the increased number of deaths in particular age groups. For example, the increase in deaths from "age" and "asthma and tisick" (398) closely corresponded to the increase in the number of deaths of people more than sixty years of age (374). The increase in deaths from "consumption and fever" (311) corresponded to the increased number of victims aged between thirty and sixty (339), and the increased number of deaths from "convulsion" and "teeth" (242) corresponded to the increase in the number of deaths of children under five (222). Not only did these categories show the greatest proportional increase during the epidemic, but they also showed the greatest rate of diminution as the epidemic diminished. Chandler also pointed out that those who were between five and thirty years of age were least likely to die.

Chandler's etiology was typical of the period in which he wrote. Without differentiating between influenza and the common cold, he ascribed the epidemic to cold weather: "The common cause of Colds, is, in general, an accidental, or sudden exposure, of the Persons taking them, to a colder, or damper air, than that to which they are usually accustomed, or were in immediately before such Exposure." The result of this exposure was that the "fibres crisped up, the pores clos[e]d, the humours condens'd on the Superficies, bound up in the Body . . . and . . . the body loaded with them. . . . the Juice[s] thick[e]ned and stiffened."

Chandler explained the age distribution of mortality by the fact that very young children were naturally flabby and weak, and subject to slow circulation, thickened blood, and obstructions, and hence to convulsions. Old people, on the other hand, were especially susceptible because they had little strength and warmth of constitution. Their solids were rigid and inelastic, and their humors cold and viscid, and they were liable to "sudden-stops, suffocation and total stagnation." Thus, the incidence of mortality due to influenza was due to the interaction of weather and constitution.


36. Ibid., p. 18.
Chandler had already written a treatise on smallpox in 1729, and in 1748 he would publish a book on the need for pharmaceutical reform. He sent his manuscript on influenza to the Royal Society, but despite the paper's innovative approach to statistical epidemiology, it was not printed in the *Philosophical Transactions*. Later research on influenza did not pursue Chandler's method of comparing the assigned causes of death with the ages of the decedents in order to estimate an unassigned but significant precipitating factor. Nor did later investigators discuss the rate of decrease in mortality due to particular causes in addition to the rate of increase.

At this point something of a vacuum was developing in the London medical world. The College of Physicians did not encourage medical research, and the Royal Society was becoming more specialized in areas of science that were not strictly medical.

The Development of Collective Investigation at Edinburgh in the 1730s

During the 1730s, however, a group of acquaintances at Edinburgh University were becoming interested in developing collective epidemiologic research. The impetus may have come from the professor of anatomy, Alexander Monro (primus). Monro encouraged both the development of aggregate case histories and the pooling of experience by several physicians to increase the number of cases observed. He published the results of his own cases, both successful and unsuccessful, and solicited information from colleagues concerning the outcome of their cases.37

When the Edinburgh Infirmary opened in 1729, Monro designed the registers in such a way that the information could be used for clinical research: the complications of each disease were to be cross-referenced.38 Such thorough registers were unusual, and the system of indexing shows that already some Scottish physicians were thinking in terms of individual diseases characterized by regularly occurring symptoms and less frequent "complications" from other, superimposed diseases. This is in contrast to Huxham's scheme, in which one disease simply turned into another.

Monro was a founder of the (Scottish) Society for the Improvement of Medical Knowledge and edited the society's journal, *Medical Essays and Observations*. One of the purposes of the journal was to make a systematic collection of meteorological and epidemiologic data. The authors hoped that this could settle the conflict between Hippocrates and Sydenham over whether perceptible or imperceptible atmospheric changes were responsible for the incidence of epidemics. As part of this project, the next volume included a description of the influenza epidemic of 1732–33 in Edinburgh. This account referred to influenza as "fevers of cold" and "this epidemic disease," discussing its incidence, mortality, symptoms, and geographic progression.40

Monro had been a student of the surgeon William Cheselden in London and had joined a student medical society with John Rutty's relation, Dr. William Rutty. It may have been this friendship that led Rutty to urge his fellow Quaker John Fothergill to enter Edinburgh Medical School and study with Monro.41 There Fothergill met another student, William Cullen. Fothergill and Cullen were among the first members of the Edinburgh Medical Society, a student organization founded in 1735 with Monro's aid and encouragement.42 At least thirty-five medical writers on influenza had been members of the society (among them were one honorary and one extraordinary member), and nine had been president.43

John Pringle (later Sir John Pringle) was then the professor of moral philosophy at Edinburgh. Pringle began to study medicine after hearing Boerhaave lecture on the application of the scientific method

37. "Preface," *Medical Essays and Observations*, 1752, 1 (4th ed., revised and enlarged): xvi. The full title of the journal was *Medical Essays and Observations, Published by a Society in Edinburgh*. This journal was issued in several editions.
39. This claim is made by Alexander Bower, *The History of the University of Edinburgh* (London: Oliphant, Waugh & Innes; Edinburgh: John Murray, 1817), pp. 173–74. Sir Christopher Booth, however, believes that it is more likely that the connection that influenced Fothergill's choice of university was with the Hillary family. See also R. Hingston Fox, *Dr. John Fothergill and His Friends: Chapters in Eighteenth-Century Life* (London: Macmillan, 1919).
to medicine, and obtained an M.D. degree from Leyden in 1730. He was to become an especially enthusiastic practitioner of the "survey" method of medical research. In 1738 he questioned five colleagues on the effect of antimony, and between 1756 and 1758 he corresponded with six military surgeons concerning the use of sublimate of mercury. After he settled in London in 1748, Pringle maintained his friendship with Fothergill. Fothergill, Pringle, and Cullen were all interested in improving the differential diagnosis of fevers, and all eventually came to entertain a contagionist theory of transmission for some fevers. Fothergill's treatise on the sore throat epidemic of 1745 was noted above. Cullen devoted much of his life to the development of a new system for classifying diseases. When he became a professor at Edinburgh in 1755, he scandalized the university by rejecting the neo-Hippocratic disease theory of Boerhaave and introducing his own synthesis. His new theory gave a prominent role to contagion. Pringle's work on dysentery and typhus are well known; he was the first to argue, in 1746, that hospital fever and jail fever were the same disease. Pringle never took a position on the transmission of influenza, but in 1764 he became interested in the idea of contagium vivum after he read a thesis in favor of that theory by a student of Linnaeus. He did not explicitly accept the theory; but he quoted it at length, arguing that all hypotheses should be suspended until it had been investigated carefully.

The Influenza Surveys of the Second Half of the Century

In 1758 there was an influenza epidemic in Scotland, and Pringle again decided to survey his colleagues. He obtained at least four responses from the physicians Thomas Simson, John Stedman, John Millar, and Robert Whytt. The last-named also enclosed a letter from his colleague John Alves. Pringle had begun an escalating process. Thereafter, the influenza surveys were to prove by far the most successful of all the exercises in coordinated group observation, both in terms of the volume of responses and in terms of the quality of the information collected (see Appendix). In the epidemic of 1767, Fothergill's colleague and close friend William Heberden appealed for information in the Medical Transactions of the Royal College of Physicians. This apparently obtained few responses, but Fothergill himself was more successful in 1775. He wrote a description of his own observations, had it printed, and sent it to colleagues throughout Britain with a request for further comments. He received at least fifteen extensive responses, including letters from Pringle and Heberden. Even more successful were two competing efforts in 1782: one, run by the Society for Promoting Medical Knowledge, a London organization, elicited thirty-four replies from British and Irish correspondents, and several from other parts of Europe. The other, the result of a public advertisement by the Royal College of Physicians, obtained seventeen responses. Among the respondents was John Haygarth, who had carried out his own local survey.


51. "An Account of the Epidemic Disease" (n. 32), pp. 155–64. See also Clark, History (n. 42), 2: 581–82. The College appointed a committee consisting of the officers of the College and William Heberden, John Monro, George Baker, and Richard Brocklesby, to hear the responses.

52. John Haygarth, "Of the manner in which the Influenza of 1775 and 1782 spread by Contagion in Chester and Its Neighbourhood" in Thompson, pp. 191–98. I have not been able to identify Thompson's source, but internal evidence suggests a publication...
In 1788, the physician Samuel Foart Simmons, editor of the *London Medical Journal*, requested observations and published an account of the results. Finally, during the epidemic of 1803 there were two even larger surveys. The first was conducted by the Medical Society of London and elicited fifty-eight letters. The second was the work of the physician Thomas Beddoes, who sent out a circular with five questions. Beddoes received 124 responses, which he published in the *Medical and Physical Journal*. Several of the respondents had also polled colleagues before writing. The journal also published several separate accounts. During the eighteenth century there were also many separate treatises published on influenza epidemics, some by very distinguished physicians, such as Sir George Baker; and William Falconer, of Chester and later of Bath (see Appendix).

The number of investigations undertaken, the number of doctors polled in a given investigation, and the number of cases each doctor collected for a "typical" report all increased steadily throughout the eighteenth century. This method of investigation was especially appropriate in the case of influenza. Overall, the disease was dramatic because of the sudden burst of cases, but individual cases were generally unremarkable in their course and did not merit extensive separate reports.

Underlying the development of these investigations was a rapid improvement in media of communication, particularly the development of specialized medical journals. Indeed, several of these were established explicitly for the purpose of furthering the collective exchange of medical information. The rise of the provincial press, the enormous expansion of secular publishing, and "infrastructure" improvements such as mail service also played a vital role.

These improvements made it easier to survey the increasing number of provincial doctors about an epidemic before it had disappeared. Thus, epidemics could now be studied prospectively rather than retrospectively. In the case of influenza, etiological arguments often centered on evidence gained from tracking the disease from place to place and noting its date of onset in each location. By the end of the century, the press, along with a well-established network of correspondents, played an especially important role at the beginning of influenza epidemics by alerting country doctors to expect cases of the disease and note the date they appeared. Without this early warning, the first few cases would have slipped past without being recognized, making it difficult to recreate a picture of the way the disease spread through the country.

For example, Thomas Glass, an Exeter physician, noted in 1775 that "from the 8th of November the number of people who were continually coughing increased so fast, that it was soon evident the epidemic colds, which began in London, as we were informed by the public papers, more than a week before, had reached us. . . . Its appearance in this city was the same as in London." The information from the London papers enabled Glass to perceive the Exeter epidemic not merely as a continuation of the "coughs" he was already seeing but as a separate phenomenon, an "epidemic cold" that had traveled across the country.

The evidence uncovered by these surveys closely reflects the more general development of eighteenth-century disease theory. By 1782, most doctors agreed about the typical signs of influenza (e.g., chills, headache, muscle aches, watery eyes, cough, prostration, and sweating) and were confident that they were analyzing the same phenomenon. Most, but not all, agreed that influenza constituted a distinct species of "fever." This was part of a process that involved the gradual separation of the disease category "fevers" into different constituent diseases such as "typhus," "scarlet fever," "puerperal fever," "pneumonia," and "hepatitis." Cullen's teaching and nosology played a central part in this process.


57. Thompson, pp. 96–97. See also John Ash, in Thompson, p. 103.
58. In addition to the sources cited above (n. 46), see Lester S. King, "Boisier de Sauvages and Eighteenth-Century Nosology," *Bull. Hist. Med.*, 1966, 40: 43–51; and
Medical investigators could not effectively pool their experience until they had defined the disease itself, but as they compared notes they also modified their definitions. For example, they were able to agree that certain complications, even though not seen in every case, resulted from the same disease. By 1782, most medical authors agreed that influenza could induce pneumonia and aggravate cases of tuberculosis. Several writers had also identified less common complications, such as encephalitis, ear abscesses, prolonged fatigue, and loss of the sense of taste. Once they had a clearer picture of the manifestations of the disease they were better able to trace its pattern of onset and thus to study its etiology.

Despite the fact that many influenza sufferers did not consult a doctor, writers in the second half of the century were able to offer at least a rough impression of the impact of the disease on the population by drawing on their own practices and comparing notes with others. Some practitioners saw large numbers of cases during epidemics: a surgeon in Bury Saint Edmunds reported that he had seen more than 500 cases during the epidemic of 1782, and a physician in Bath was said to have treated 120 people in two days.

Case notes enabled doctors to gain an impression of the incidence of the disease, which varied from epidemic to epidemic. Some reported on the different ways in which the disease had affected different groups of people. In his summary of reports sent to the Society for Promoting Medical Knowledge in 1782, Edward Gray noted that the elderly were less likely to contract influenza but had more severe cases when they were attacked. He also concluded that very young infants were generally immune. He added that many who had escaped in 1775 became ill in 1782, and many who had become ill in 1775 escaped in 1782. Samuel Argent Bardsley, physician to the Manchester Infirmary, reported to the London Medical Society in 1803 that the case-fatality rate was not more than one in two hundred, that female servants were especially susceptible, that infants and the heads of families were relatively immune, and that those who suffered the most severe cases were “old, asthmatic or otherwise debilitated.” He noted influenza’s disastrous effect on pregnant women and recommended that they observe strict seclusion to avoid contagion.

The Issue of Contagion between 1758 and 1782

It seems evident that from the time of Pringle’s first circular, doctors considered the possibility of contagion an important one for investigation, even though Pringle’s correspondents preferred to blame some unknown atmospheric phenomenon. For example, Robert Whytt noted that influenza was probably not caused by the weather, because the spring season had been remarkably mild and dry. He rejected contagion because attendants of the sick did not seem to be especially likely to contract the disease. He concluded, “Our epidemic did not spread by contagion, from one person to another, like the plague,

60. William Norford to Henry Reyell Reynolds, Bury St. Edmunds, 13 August 1782, Royal College of Physicians, MS. 1045/18. I would like to thank the Royal College of Physicians for supplying this material and granting permission to publish it.
smallpox, or measles, but seemed to be owing to some particular quality of the air.\textsuperscript{64} Another of Pringle’s correspondents, John Millar, also argued that influenza “did not seem to be produced by any other contagion than that of the air, because all in the same family that were seized with it generally fell down at once . . . nor did it spread, as might have been expected, were it infectious.”\textsuperscript{65}

The collective effort of eighteenth-century epidemiologists gradually called into question this contention that epidemics were generated in the atmosphere, either by the sensible qualities of the air, such as cold, or by imperceptible airborne emanations. First, it became increasingly evident that epidemics of many diseases could take place during a variety of weather conditions. The effort by the Society for Improving Medical Knowledge to analyze the interaction of epidemic diseases and weather patterns by comparing the seasons and diseases in four cities had failed to uncover any comprehensible pattern.\textsuperscript{66} Far from settling the conflict between Hippocrates and Sydenham, it demonstrated the futility of this approach to epidemiology.

Second, by comparing their notes doctors were able to trace the movement of influenza epidemics not only from country to country but also from place to place within Britain. This evidence made meteorological explanations increasingly difficult to sustain. Although all of a region experienced the same weather, one town might experience an epidemic before another town nearby. A distant town might suffer an epidemic weeks after it appeared in London, even though the weather had changed. The spread of the disease was not correlated with prevailing winds, but it did follow the pattern of human migration (i.e., trade routes), and it moved down the “urban hierarchy” from large settlements to smaller ones.\textsuperscript{67}

In 1762, Sir George Baker was frankly agnostic on the question of causation, commenting:

I have chosen to premise these brief remarks on the state of the atmosphere . . . lest I should entirely depart from the custom and manner usually adopted . . . in works of this kind; but . . . I never could be persuaded that it was reasonable to attribute the origin of epidemic diseases to change-

\textsuperscript{64} Whytt, “Account” (n. 48), p. 65.
\textsuperscript{65} Millar, “Account” (n. 48), p. 67.
\textsuperscript{67} The phrase comes from Patterson, Pandemic Influenza (n. 1), p. 20. According to Patterson, movement from cities to outlying regions was first noted by Baker during the epidemic of 1761–62.

Baker’s comment reveals the extent to which doctors depended on the collective accumulation of precise information about the incidence and date of onset of the epidemic in various places to shape their theories. He concluded that physicians were ignorant of the mechanisms by which the disease was transmitted.

Baker was the first author actually to reject the hypothesis of atmospheric causes outright, but as more information accumulated he was soon echoed by others. Far from settling the matter, new findings added to the controversy and created an appetite for even more information. In 1767, Baker’s teacher William Heberden pointed out, “The season preceding this disorder was only remarkable for being unusually cold; but then, it is observable, that the similar epidemic cold of the year 1762 was preceded by weather as uncommonly warm.”\textsuperscript{68} In 1775, John Fothergill commented that he had made note of the weather and barometer, “[t]hough attempts to ascertain the causes of epidemics are, for the most part, more specious than substantial.”\textsuperscript{69} Sir John Pringle noted that “the sensible qualities of the air” had “evidently no part [in producing the epidemic] for we hear of the same distemper having been in Italy, France, and in the Low Countries . . . But it cannot be supposed that the state of the atmosphere, either as to weight, heat, or moisture, was the same everywhere . . . such epidemics do not . . . depend on any principles we are yet acquainted with, but upon some others, to be investigated . . . by the united inquiries of . . . [Fothergill’s] brethren.”\textsuperscript{70}

Only one physician who answered Fothergill’s circular letter in 1775, David Campbell, of Lancaster, was prepared to argue without qualification that influenza was contagious because it generally ran serially through whole families and, because of the way it progressed from London toward the north of England. It arrived in Lancaster nearly

\textsuperscript{68} Baker, De catarrho (n. 55), pp. 69–70.
\textsuperscript{69} Heberden, “Epidemical Cold” (n. 49), p. 85.
\textsuperscript{70} Fothergill, “Sketch” (n. 49), p. 88.
\textsuperscript{71} Sir John Pringle, letter to Fothergill, in Fothergill, “Sketch” (n. 49), pp. 89–90.
three weeks after it had prevailed in London and about three days after it had hit Liverpool. Within a few days, it had spread outward to Kirkby Lonsdale and finally to Kirkby Steven. Although few printed reports endorsed Campbell's frank contagionism, apparently by 1775 many doctors shared his views, for Thomas Glass, an Exeter physician, commented that although he himself believed that influenza was due to "something in the air, which is not the object of sense," a belief in contagion was "the more general opinion." The pandemic of 1782 aroused a great deal of interest among medical men, who tracked its progress through Russia and India to Europe and North America. In Europe alone, it probably caused several hundred thousand deaths. In Britain, this pandemic was the subject of several separate essays and two major medical surveys, one by the Society for Promoting Medical Knowledge and the other by the Royal College of Physicians. The College advertised for information and assembled a committee to hear the responses read, after which it asked its registrar, Henry Revell Reynolds, to communicate the most important letters to the College. In 1785 the College published in its Medical Transactions a report on the pandemic that was based on the information it had gathered from respondents to the advertisement.

72. David Campbell, letter to Fothergill, in Fothergill, "Sketch" (n. 49), p. 113. David Campbell (1749–1832) was physician to the Lancaster Dispensary and author of an important book on typhus, but biographical information is limited. He obtained an M.D. degree at Leyden in 1770 and another degree at Edinburgh in 1777. I have not been able to trace any connections with other physicians in the northern group, but he dedicated his Leyden thesis to William Cullen, and to William Cuming, of Dorchester, who was a close friend of Fothergill's and Whytt's. Cuming also contributed a letter to Fothergill's influenza survey. Campbell is described as "English," at Leyden, but there is some evidence that he lived in America. He served as mayor of Lancaster, England, in 1796–97. Lancaster City Library, Biographies file; R. W. Innes Smith, English-Speaking Students of Medicine at the University of Leyden (Edinburgh: Oliver & Boyd, 1952), p. 39.


74. Patterson, Pandemic Influenza (n. 1), p. 24.


Some doctors objected to the entire enterprise, arguing that epidemiologic investigation was beside the point. In a work of 1797 entitled Medical, Philosophical, and Vulgar Errors... considered and refuted, the author, John Jones, "M.B.," included as an "error" the idea "that the influenza is a very dangerous distemper, and a new one; never known in this country till a few years ago; at which time the College, by their circular letters, cried out for help from all quarters, were themselves greatly alarmed; and spread a general terror." In fact, he argued, influenza was neither new nor dangerous but an ailment known to every person who left a warm room to ride at night in a fog. "Such is our improvement in philology... that since a travelled fine gentleman has been pleased to dub a common cold... with a foreign name of influenza; all our catarrhous colds, amongst our gentry, have assumed the same name and importance."

In this comment, Jones linked his opposition to epidemiology with his opposition to the construction of influenza as a specific disease. Both stemmed from his neo-Hippocratic etiology: if influenza could be caught by anyone suddenly exposed to cold damp air, then it could not be a separate epidemic disease and was not worth investigating as such. Influenza was merely a chill with a college education. Similarly, the neo-Hippocratic physician William Stevenson argued in 1782 that influenza came from "mechanical sudden changes of the weather, to which we did not admit our mode of living; so that ourselves were the infecting cause, and not the air." As a result, Stevenson believed, all collective efforts to investigate epidemics were irrelevant, a medical infection "more CONTAGIOUS than the disorder itself." It was "quite an absurdity... to do what can only be done in private studies." Like Huxham, Stevenson was concerned that the attention given to diseases might derogate from the attention properly devoted to sick individuals.

Stevenson, a Presbyterian Jacobite, was also suspicious of any attempt by the College of Physicians to preempt the judgment more properly exercised by individual physicians acting on their knowledge of their patients. He believed that this was analogous to the efforts...
of the Church of England to subvert the priesthood of all believers. Dedicating his work to “all who are not MEMBERS of the Royal College of PHYSICIANS,” he claimed that the College had no authority to pronounce on the issue of disease transmission: “No body of men has a right to establish doctrines, or even to recommend them, beyond the authority of a single individual: nor should I be less apt to suspect the scientific decisions of a royal college of physicians, than the theologic [sic] ones of the houses of Convocation.” Stevenson’s condemnation of medical and theological authority alike was reminiscent of the rhetoric of medical radicals in the Civil War period, as was his repudiation of venesection on the grounds that “blood is the life of man.”

Despite such strictures, seventeen medical practitioners, including an anonymous “young Apothecary,” sent accounts to the Royal College of Physicians, which still holds the original copies. None of the correspondents was a Fellow of the College, although one, Martin Wall, of Oxford, was later admitted. The resulting report, although it commented that the 1782 pandemic “had more evident marks of contagion, than that of 1762,” was noncommittal on the question of transmission. It simply recited, without further comment, facts that might be interpreted either way. In some cases individuals had brought the disease into towns, but in other cases there was no known connection between outbreaks.

The letters themselves were divided on the question of disease transmission, with six making no commitment either way, five favoring some atmospheric theory, and six arguing for contagion. Nevertheless, the neutral stand of the report did not adequately convey the nature of the responses. The anticontagionist letters were in general perfunctory, whereas the contagionist letters included two reports later published separately. The first of these came from John Haygarth, of Chester. The second was sent by Robert Hamilton, of Luton, in Bedfordshire, to Fothergill’s protégé John Coakley Lettsom, in London, who forwarded it to the College; both of these reports were carefully thought out, thoroughly researched, detailed, and persuasive.

Haygarth and Fothergill were close friends; they had attended the same school, and Fothergill spent his summers in Cheshire. Haygarth had responded to Fothergill’s circular on influenza in 1775, but at that time he did not believe that the disorder was contagious. He had traced the time of onset through Cheshire and Wales, and had found that the disease attacked some Cheshire villages more than ten days after it had appeared in the western part of the county and in bordering parts of Shropshire. He wrote that “[t]hese facts, compared with the general seizure, make the theory of this epidemic very difficult. On the whole, I believe people in the country were attacked rather later than in the towns they surrounded.” He found no evidence that situation or prevailing weather conditions made any difference.

In the succeeding years, however, Haygarth had carried out the investigations that persuaded him, and many of his colleagues, that smallpox, typhus, scarlet fever, and possibly other epidemic fevers as well were contagious in the strictest sense—that they could not spread without direct contact and therefore could be controlled by quarantine. In response to the inquiry by the College of Physicians in 1782, Haygarth again turned his attention to influenza. He again surveyed his colleagues in the neighboring towns and villages to prepare his reply.

This time, the same evidence which had “rendered a theory of this epidemic very difficult” in 1775 seemed to Haygarth to provide compelling proof that the disease was contagious. He entitled his dissertation “Of the Manner in Which the Influenza of 1775 and 1782 Spread by Contagion in Chester and Its Neighbourhood.” He argued that the fact that the disease had traveled along trade routes from London to Chester, from Chester to market towns, and from these towns to villages and scattered rural houses showed that it was spread by carriers, not by the air. Moreover, in most cases he had been able to identify the individual who had carried the disease into a new community.

Haygarth then asked why it was that influenza spread so much more quickly than other diseases that were considered contagious,

79. Royal College of Physicians, MSS 670, 1045/18, 3012/1–23, omitting 3012/10. This collection includes one letter in Spanish from Martin Rodon y Bell which I have not included because it was dated 1790.
81. Ibid., p. 157.
83. Haygarth, “... Influenza of 1775” (n. 52), pp. 191–98.
such as smallpox. He argued that smallpox victims were generally so ill that they remained at home, and that, since the marks of the disease were obvious, susceptible persons could avoid those with active cases of the disease. He also noted that most townspeople were already immune to smallpox, whereas many of them were susceptible to influenza. He pointed out that the man who had brought influenza from London to Chester had traveled 182 miles in twenty-seven hours, so it was certainly possible for human carriers to spread the disease throughout Britain in a few days. Finally, he commented that because influenza had a shorter incubation period than smallpox, the former disease could pass from person to person more quickly than the latter.

Haygarth’s arguments were echoed by Robert Hamilton in a long and carefully researched essay sent to John Coakley Lettsom. Lettsom forwarded it to the College of Physicians, but it was Lettsom’s London Medical Society that finally published it in 1787. After tracing the date of onset of the disease from London to the north of England and documenting its spread along trade routes throughout his practice in Bedfordshire and Hertfordshire, Hamilton concluded that the “Cause of influenza was not in the Air, but in a Specific Contagion.” Bad weather might lower a patient’s resistance and render him more susceptible to infection, but “We have many examples to prove, that the air cannot hold, nor yet convey contagion to any distance. . . . Experience shews, that contagions have always been communicated by contact with the infected, either mediately or immediately.”

The subject of contagion is an obscure one. . . . We might amuse ourselves with inquiring, wherein consists its difference, by which it can produce in the human body diseases specifically different? Why one kind seems to exert its force on the mucous membrane of the trachea, nose, etc., and produce fever as in . . . [influenza]? Why another spends its fury on the skin . . . as in the smallpox? But . . . our present limited knowledge of the various combinations of matter will not allow us soon to evolve them; yet, as the knowledge of the human mind is progressive . . . I would not too rashly conclude them inscrutable. Was this inculcated it would prove a check to industry, and become the nurse of ignorance.

Hamilton’s comment hints at the underlying relationship between contagionism and a new characterization of disease: he saw diseases as “specifically different,” that is, as being of different species. If disease came from changes in the weather, then each epidemic was only accidentally related to another, for similar atmospheric conditions induced similar symptoms. But if disease resulted from a particular “combination of matter,” then disease itself existed as a “thing” and might continue to exist over time. The same “matter” that could be passed from patient to patient, causing repeated attacks of the same disease, could also cause repeated epidemics of that disease—a disease that was the same in its essence as well as in its symptoms. Conversely, people who were sick at the same time (and who were thus breathing the same air and experiencing the same weather) might still suffer from different and unrelated “species” of disease.

Hamilton’s peroration also reveals the connection that the contagionists made between contagionism, optimism about the prospect for controlling disease, the need to encourage continued research, and a more general “Enlightenment optimism.” Hamilton believed not only that “knowledge is progressive” but also that physicians must agree that this was the case. Failure to do so might lead to apathy and inaction.

The anticontagionist Stevenson had worried that the epidemiologic investigations of the College of Physicians might lead physicians to become “slothful,” relying too much on the authority of others and not enough on their own observations. He also suggested that the invocation of collective medical authority would encourage patients to neglect the control that they themselves individually exercised over their own welfare. The contagionist Hamilton, on the other hand, was concerned that the pessimism induced by emphasizing the limits to human knowledge might discourage efforts to increase collective understanding. Just as disease itself was transmitted by a social network, he believed, so too was knowledge.

The College’s report not only disregarded the arguments in these two letters but also misinterpreted the evidence itself in describing three instances of shipboard epidemics. The first had occurred on an East India Company ship, the Atlas, as it sailed from Malacca to Canton.

85. Ibid., pp. 177–78.
86. Cf. Carter, "Pasteur's Concept" (n. 29), p. 544: "One disadvantage of relying on sufficient causes is that they contribute almost nothing to the practical control of disease . . . sufficient causes make good sense in a medical system in which the threat of disease is used to reinforce social norms, but . . . prevention and treatment require necessity."
in 1780, and the report erroneously implied that the ship had had no contact with the shore. In fact, the correspondent, a firm contagionist, had pointed out that men from two fishing boats had come on board three or four days before the epidemic. The second and third outbreaks had taken place simultaneously in the British fleet in 1782: the first in Admiral Kempenfelt's squadron and the second aboard the main fleet, under Lord Howe. In both cases, the report specifically stated that there had been no communication between the affected ships and the shore.

In discussing the latter two incidents, the report drew on evidence sent by the doctor who had been called to treat the sailors in both epidemics, but neither he nor the other medical men who supplied information about the timing of the epidemics claimed that the ships had been entirely cut off from communication with the shore. Moreover, John Lind, physician to the naval hospital at Haslar, sent a report that argued that both incidents offered strong proof in favor of contagion because the disease had been carried to individual ships by personal contacts that had been traced:

While this disorder was general at land, a squadron ... was cruising in the Channel, and entirely escaped it, until one of the ships ... put for twenty four hours into Torbay, and received the infection by a boat from the shore; in one day eighty of her people were taken ill. ... The other ships by their communication with this received the infection. ... Soon afterwards a large fleet under Lord Howe ... returned healthy to this port, and upon intercourse with the shore was immediately seized with the reigning disorder. His Lordship left a squadron upon the coast of Holland, which, ... escaped the disorder for a considerable time longer, until at last several of the ships received it. ... by means which could be traced. ...

The principal Vehicle of the infection seemed to be the breath.

The College's report did not include this version of events, perhaps because the conservative College was not enthusiastic about contagionist disease theories. As a result, this "evidence" of outbreaks on isolated ships continued for more than a century to puzzle commentators on the transmission of influenza.

Edward Gray's Report on the Epidemic in 1782

The report of the Society for Promoting Medical Knowledge appeared in the first volume of Medical Communications. There were at least thirty-two letters from England and Ireland, as well as communications from Paris, Hamburg, and Venice. These were summarized by the physician Edward Gray, a Fellow of the Royal Society. Of the British correspondents, eleven took a position on the mechanism of transmission. Of these, eight writers argued in favor of contagion; one preferred it, although he had some lingering doubts; and only two opposed it.

Gray's own comments provide a definitive summary of the contagionist case. Some respondents, he commented, thought that influenza arose from the weather, whereas others contended that it was due to a "particular and specific contagion, totally different from ... the sensible qualities of the atmosphere, yet conveyed by ... the air. But the greatest number concurred in opinion, that the influenza was contagious, in the common acceptance of that word; that is to say, that it was conveyed and propagated by the contact, or at least by the sufficiently near approach, of an infected person.

Miasmatics, Gray continued, depended on five pieces of evidence:

1st. That those most exposed to the weather were generally the first persons attacked. 2ndly. That many had the disorder without having had any communication with a diseased person. 3rdly. That several escaped, though surrounded by persons ill of the disease. 4thly. That some whole families were seized at once. 5thly. That some persons had the disorder a week or fortnight before it began to be taken notice of as a general one.

Gray believed that none of these arguments was sufficient to demonstrate airborne infection. He quickly dismissed the first, third, and fifth points. If the cause were in the air, those at home would have become ill as rapidly as those who went out, since all breathed the same air. Similarly, the escape of some from the disease was equally difficult to explain whether they were surrounded by diseased persons.
or by the air that caused the disease. Nor, finally, could the hypothesis of airborne infection explain the existence of early cases. These three pieces of evidence could not determine the issue either way.

More significant is Gray's comment on the second contention: that many were attacked without having had communication with a victim. He wrote that before this argument could be accepted it would have to be shown not only that the new patients had not encountered a previous victim but also that no one who had been near them had previously been exposed to other cases, "for as it is generally admitted, that a person who has [previously] had the smallpox, can yet convey the infection of that disease from a person ill of it to one who has not had it, so, by parity of reasoning, it will surely be allowed that a person not actually labouring under ... influenza, could ... carry the infection of it from one place to another."93

In other words, influenza's contagiousness could only be disproved if it could be demonstrated that there was no such thing as an asymptomatic carrier. Gray noted that the probable existence of asymptomatic carriers also cast doubt on the fourth argument against contagion: that whole families were affected at once. If the cause was in the air, then influenza should have affected equal numbers of persons in different families, but if the disease was conveyed by a carrier, it was easy to see why a whole family might be affected simultaneously. Gray's argument was significant for two reasons. First, Gray placed on miasmatics the burden of disproving the possibility of contagion. He was assuming that contagion was the more likely explanation, which must be shown to be erroneous before other explanations could prevail. The fact that a physician could, even tacitly, make such an assumption marked a new era in disease theory. Second, he was pointing out that the existence of a healthy carrier was "generally admitted" in cases of smallpox and should be admitted also in other diseases.

By the 1780s, these views were widely shared. William Cullen's nosology of 1785 listed influenza as "Catarrhus a contagio."94 John Haygarth's closest friend, William Falconer, published in 1782 a treatise on influenza which also argued that the disease was contagious, as did an essay by the London physician William Grant and a treatise by the physician Patrick Dugud Leslie, of Durham. Moreover, Leslie's work contained a long letter from the physician John Clark, of New-

93. Ibid., p. 141.
94. Cullen, Synopsis nosologicae methodicae (n. 46), pp. 289-90. Cullen listed Catarrhus a contagio in the category "Catarrhus" (colds) of the order "Hemorrhagiae" (hemorrhagic diseases; diseases causing loss of body fluids) of the class "Pyrexiae" (fevers). Other members of the order Hemorrhagiae included dysentery and phthisis.

95. Falconer, Account of the Late Epidemic Catarhhal Fever (n. 55); William Grant, Observations on the Late Influenza, the Febris Catarrhalis Epidemica of Hippocrates, as it appeared at London in 1775 and 1782 (London: Printed for author, n.d.); Patrick Dugud Leslie, An Account of the Epidemical Catarhhal Fever commonly called the Influenza; as it appeared in the city ... of Durham ... with a letter to the Author ... by John Clark, M.D. (London: S. Crowder & J. Rolson; Edinburgh: A. Gordon & C. Elliot, n.d., c. 1783). But there was also a treatise by Arthur Broughton, Observations on the Influenza ... at Bristol (London: Robinson, 1782), reviewed in London Med. J., 1782, 3: 297-98, which argued that influenza was the result of evaporation from recent heavy rains. The London Medical Journal reviewed Falconer favorably in the same volume (pp. 294-96) but commented of Broughton, "The argument is ingenious, but in the present instance we cannot adopt it. There were phenomena in the late epidemic, which we think can be explained in no other way than by supposing it to have been a contagious disease." See also Anthony Fothergill, "Account of the Epidemic Catarrh (termed Influenza) as it appeared at Northampton ... in 1775; together with a comparative View of a similar Disease ... in 1782," Mem. Med. Soc. London, 1792, 3: 30-43, which was agnostic on the question of etiology.
97. Patterson, Pandemic Influenza (n. 1), p. 29; see also p. 101 n. 2. Patterson believes that a "strong majority" of Beddoes's correspondents were anticontagionists, but my own count did not support this conclusion. Beddoes tried to classify the letters, but they soon became mixed. Needless to say, this is to some extent a subjective judgment. For example, Beddoes lists Alderson as an anticontagionist, but Alderson actually wrote that "as it began at different times in different parts of the country, there was reason to suppose it contagious; but all who got cold at that period, and for six weeks afterwards, had nearly similar symptoms." "Mr. Beddoes's Papers on Influenza" (n. 54), p. 126. Alderson may be saying that influenza was more likely to attack those whose resistance was lower, but he is certainly not saying that it is not contagious. After evaluating this response in light of the rest of Alderson's work, I have counted him as a contangist. On the other hand, I have counted Robert Bree ("I have not been able to satisfy myself on this head") as undecided, although he is also among the anticontagionists; and "Mr" Brewer ("I can do very little more than give my opinion contrary to its contagious effects") as an anticontagionist, although he is listed with the contangists. "Mr. Beddoes's Papers on Influenza" (n. 54), pp. 125, 522.

Contagionism and the Investigations of 1803

By 1803, when the next major epidemic occurred, the contagionists seem to have been increasingly on the defensive. There were again two major influenza surveys: one conducted by John Coakley Lettsom's London Medical Society, the other by Thomas Beddoes.95 One of Beddoes's correspondents wrote that most of the profession now doubted that influenza was contagious.97 The evidence of the surveys
themselves, however, suggests that opinion was almost evenly divided: of the 58 practitioners who responded to the medical society's survey, 18 thought that influenza was contagious or was probably contagious, 23 thought that it was not contagious or was probably not contagious, and 17 were undecided or returned unclassifiable responses. Of the 122 practitioners who answered Beddoes's questionnaire, there were 44 contagionists, 43 anticontagionists, and 35 who were undecided or unresponsive. In both 1782 and 1803 the proportion of contagionists was about one-third, but in the latter epidemic the number of avowed miasmatisists or anticontagionists had risen to an equal level. Some practitioners answered both surveys in 1803, and five had also responded in 1782.

It was after this epidemic that Haygarth finally decided to publish his treatise on influenza. It contained the material on the earlier epidemics of 1775 and 1782 which he had sent to the College of Physicians. Haygarth wrote that he had not previously published his comments because the contagious nature of influenza had been so thoroughly proved by many physicians, including his friend William Falconer, that it seemed unnecessary to add his support. In the intervening years, however, many physicians had again begun to ascribe influenza and many other epidemics to a "morbid constitution of the atmosphere, independent of contagion." Haygarth was afraid that if this view prevailed it would discourage efforts at prevention, because "the morbid constitution of the atmosphere cannot possibly be corrected or controlled by man."98

In the case of influenza, research had shown that certain individuals, particularly those who were aged or ill of other diseases, were especially at risk of dying from the disease, and that protective measures, such as strict cleanliness and separation from potential carriers, should be confined to such individuals. In the case of other diseases, however, Haygarth hoped that more general public health measures could be introduced, and that these measures would have a substantial impact on overall morbidity and mortality. Haygarth saw miasmatism as a political threat, not merely a competing explanation, because it discouraged public health measures, which required public investment and intervention on a very large scale if they were to be effective in interdicting disease.

In light of such comments it might be fair to conclude that one factor underlying the increasing hostility to contagionism was the more conservative political atmosphere in Britain following the French Rev-

olution. Many of the most committed contagionists were also political reformers.

Among those who continued to argue in favor of contagion were some very distinguished physicians, including Samuel Arget Bar- dley, of Manchester; John Clark, of Newcastle; Jonathan Binns, the Quaker principal of Ackworth School; and John Alderson, of Hull. Another distinguished contagionist was Andrew Duncan, Sr., professor of medicine at Edinburgh, who had been a student of Alexander Monro (primus), and who served for many years as treasurer of the Edinburgh Medical Society. William Falconer also had not changed his mind. In his account of the influenza epidemic at Bath in 1803, he commented that, although there had been debate within the profession about the nature of the disease, "I have no doubt myself that it is contagious, in the strictest sense of the word."99 Another contagionist author was Richard Pearson, an Edinburgh graduate who was physician to the Birmingham General Hospital and founder of the Birmingham Medical School. Pearson, however, did not receive much support from the eight correspondents whose comments he included in his treatise.100

More important than the numbers on either side was the fact that contagionism had become a central medical issue; Thomas Beddoes organized the answers to his survey around it. In the early eighteenth century the issue of etiology had simply not been considered in those terms. A few respondents rebelled at the way the questions had been framed. For example, "Mr. Lee," of Bristol, commented, "I do not think the prevailing disease; ... deserving of any such specific name as influenza. I think the same disease would occur on the same previous and subsequent state of the atmosphere, with respect to heat, cold, or winds occurring. ... To call diseases names, or to break or fritter their various hues into shades, may be safely abandoned to artist-godfathers and godmothers."101 As had been the case with earlier authors, Lee's atmospheric theory of disease led him to resist the classification of fevers.

98. Haygarth, "Of the manner in which the Influenza spread" (n. 52), pp. 197–98.
100. Richard Pearson, Observations on the Epidemic Catarrhal Fever, or Influenza, of 1803, To which are subjoined, Historical Abstracts concerning the Catarrhal fevers of 1762, 1775, and 1782, 2d ed. (London: C. & R. Baldwin, 1803). Pearson's own comments are reprinted in Thompson, pp. 239–46, along with a ninth response that was printed separately; but the comments of the other eight correspondents, which were included in his book, are not included in Thompson. See also John Nelson Scott, "Observations on the Influenza as It Appeared in the Isle of Man, in Spring, 1803," reprinted from Annals of Medicine, in Thompson, pp. 271–78.
Most authors, however, considered the question of influenza's etiology seriously and made their decision according to what they had observed of the incidence of the disease. England had become a nation of epidemiologists. Walter Vaughan, of Rochester, concluded that influenza was not contagious because whole families fell ill at once and because too many contacts escaped it entirely, although he was "accustomed to look upon every fever with a fear that it is contagious." The surgeon Richard Dunning feared that the disease was not contagious because of the swiftness of its spread and its apparently very short incubation period; if it were contagious, however, he thought that his friend Edward Jenner's recent discoveries (concerning vaccination) offered hope for controlling it. Dunning thought that the cause might be an atmosphere impregnated by animalcules, "animated atoms." Similarly, Edward Luscombe, an army surgeon, wrote directly to the *Medical and Physical Journal* enclosing a table that listed the numbers of people affected by influenza in each room of a cavalry barracks. He thought that the fact that a large number were exposed to a few cases in each room and yet escaped showed that the disease was not contagious.

Conclusion

The story of influenza research in the nineteenth century must await further analysis. Contagionist views on influenza probably became increasingly uncommon in the first half of the century, although these views were never abandoned entirely. Creighton has argued for steadily increasing anticontagionism in the 1830s, but Creighton himself was an anticontagionist, and his evidence on this point is unreliable. A study of cholera has found that many doctors continued to believe in the contagiousness of that disease, even though the evidence in that case was less persuasive than it was for influenza.

Writing in 1857, the physician Theophilus Thompson, a Fellow of the Royal Society and a Fellow of the Royal College of Physicians, offered his own conclusions, based on the materials on influenza which he had compiled. He thought that contagion could not be responsible for simultaneous appearances such as those he believed had occurred in the navy in 1782. He considered it possible, however, that contagion was one mode of diffusion among several. "The more remarkable facts relative to the question of contagion, are those recorded regarding the epidemics of 1775, 1782, and 1805. . . . If we leave out of consideration diseases communicable by inoculation, such as smallpox, the evidence for the occasional contagiousness of influenza is similar to that adduced for any disease acknowledged to be contagious."

Although it may have fallen into comparative disfavor in the nineteenth century, contagionism had made two important contributions to eighteenth-century medicine. First, it encouraged the appropriate classification of diseases. In comparison with neo-Hippocratic writers such as John Huxham, contagionists such as Robert Hamilton had a firmer hold on the ontological theory of disease—the idea that each distinct group of symptoms formed a "thing" specifically different from every other disease but persisting from time to time, from place to place, and from person to person. Although a disease might be complicated by additional symptoms, one disease could not "turn into" another, different disease because of a change in the weather or in treatment. Nor could the nature of the disease be fully determined by an individual patient's constitution. This belief that influenza was a specific disease persisting across time and place enabled physicians to accept the observations of previous generations of writers and of their own correspondents as relevant to their own experience of what they believed to be the same disease.

The value of epidemiology may now seem to be obvious, and the development of the methods used by eighteenth-century physicians may seem to have been inevitable. But the importance of the connection between disease theory, epidemiology, and methods of investigation is revealed when we turn to anticontagionist writers such as William Stevenson, in 1782; John Jones, in 1797; and "Mr. Lee,"

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102. Walter Vaughan, letter to Mem. Med. Soc. London, 1803 (n. 96). Vaughan argued that about one person in three exposed to jail fever (typhus) escaped, but that a much higher proportion of those exposed to influenza had escaped. In 1803, typhus was generally agreed to be a contagious disease.

103. Richard Dunning, letter, Med. Phys. J., 1803, 10: 129–50; quotation on p. 139. Dunning clearly had a theory of *contagium vivum* without being a contagionist in this case: he argued that animalcules caused plant diseases, and continued, "The atmosphere . . . is full of animalcules . . . There is no putrefaction where these are not most abundantly present. A drop of stagnant water is a drop of animals. Nothing forwards . . . the putrescency of animal substances so rapidly as the blow or germ of insects and animalcules: indeed, they would seem almost to be . . . the principle and pabulum of putrefaction" (pp. 139–40).


of Bristol, in 1804. These writers contested the use of a specific term to distinguish influenza from other diseases caused by similar environmental conditions. As a result, they criticized epidemiologic investigations based on the idea of "influenza" as a waste of time and effort. Not all medical theorists agreed; many miasmatists also worked on influenza epidemics. Contagionism, however, helped frame useful questions about the incidence and spread of the disease, and gave the pursuit of epidemiologic studies a new sense of importance and urgency.

In addition, contagionism gave rise to the optimism about the possibility both of understanding disease and of preventing it. No one, as Haygarth pointed out, could hope to escape invisible emanations in the atmosphere. If, however, disease was the result of poisonous particles spread by carriers, it should be possible to control its transmission and perhaps to identify the substance involved. As Haygarth and Hamilton both argued, hope is a great spur to investigation. The development of contagionist theories encouraged doctors to study the incidence of specific diseases and gave them hope that further investigation could lead to the development of measures for control. Contagionism thus encouraged further epidemiologic investigation and framed the terms of inquiry in a way that helped to elicit new sorts of evidence and to encourage new forms of collective investigation.

Accounts of Eighteenth-Century Influenza Epidemics in Britain

In the left-hand column of the listing below, asterisks preceding dates identify pandemics. Works are listed according to the date of the epidemic or pandemic they discussed, not the year of publication, which in some cases was considerably later.

Abbreviations Used Below

Thompson  Thompson, Theophilus, ed. Annals of Influenza or Epidemic Cholera and Fever in Great Britain from 1510 to 1837. London: Syndenham Society, 1852.


Authors and Works


*1729–30  Author of The Family Companion for Health (1729). An Enquiry into the Causes of the Present Epidemical Diseases... Cited in Creighton, p. 343.


*1732–33  Arbuthnot, John. An Essay concerning the Effects of Air on Human Bodies. 1751. In Thompson, pp. 35–38. Episode mentioned in course of more general work.


Baker, George. De catarrho et de dysenterio Londinensi epidemicis utriusque an. 1762, Libellus 1764. Translated by Theophilus Thompson, 1852. In Thompson, pp. 68–70.


Rutty, John. A Chronological History of the Weather and Seasons, and of the prevailing Diseases in Dublin. 1770. Excerpted in Thompson, pp. 79–84. Episode mentioned in course of more general work.


Fothergill, Anthony. “Account of the Epidemic Catarrh (termed Influenza) as it appeared at Northampton ... in 1775; together with a comparative View of a similar Disease ... in 1782.” Memoirs of the Medical Society of London (Lettoson's society), 1792, 3: 30–43.

Grant, William. Observations on the late Influenza the Febris Catarrhalis Epidemica of Hippocrates, as it appeared at London in 1775 & 1782. London, [c. 1782].


Committee of the Fellows of the Royal College of Physicians. “An Account of the Epidemic Disease, called the Influenza, of the Year 1782, collected from the Observations of several Physicians in London and in the Country, by a Committee of the Fellows of the Royal College of Physicians in London.” Reprinted from Medical Transactions (Royal College of Physicians), 1785. In Thompson, pp. 155–64. Henry Revell Reynolds received responses from "A Young Apothecary," Charles Brown, Cathbert Challoner, John Clark, Thomas Gibbons, Edward Holwell, James Lind (1716–94), James Lind (1736–1812), John Lind, Thomas Meek, William Norford, Stephen Pemberton, John Stewart, James Walker, Martin Wall, Robert White, and Peter Wright. The letters were not published. John Haygarth and Robert Hamilton also responded, but their letters were later published separately (see below). John Coakley Lettsom forwarded a copy of Hamilton's letter with a brief cover letter.


Grant, Observations. See above, 1775.


Haygarth, "Of the Manner." See above, 1775.

Leslie, Patrick Duguid. An Account of the Epidemical Catarhthal Fever commonly called the Influenza; as it appeared in the city . . . of Durham . . . with a letter to the Author . . . by John Clark, M.D. London: S. Crowder & J. Robson; Edinburgh: A. Gordon & C. Elliot, [c. 1783].

Monro, Donald. A Short Account of the Present Epidemical Disorder commonly called Influenza. London, 1782.


Stevenson, William. Candid Animadversions on Dr. Lee's narrative of a singular Gouty Case, to which are prefixed Strictures on Royal Medical Colleges, likewise a summary Opinion of the late Disorder called the Influenza. Newark, England: J. Tomlinson, 1782; microfilm, Woodbridge, Conn.: Research Publications, 1986.


Falconer, William. An Account of the Epidemical Catarhthal Fever, commonly called the Influenza, as it appeared at Bath in the Winter and Spring of the year 1803. 1803. In Thompson, pp. 253–71.


Nott, John. Influenza as it prevailed in Bristol in February-April, 1803. 1803. Cited in Creighton, p. 375.


Called by God, Led by Men: Women Face the Masculinization of American Medicine at the College of Medical Evangelists, 1909–1922*

CLARK DAVIS

The growing number of women physicians in American medicine is often considered a recent phenomenon made possible by the feminist movement of the 1960s. In fact, however, women played numerous and central roles as healers throughout the nineteenth century. They actively participated both in major sectarian movements (e.g., hydropathy) and in the allopathic profession. In the latter, for a brief period at the end of the nineteenth century, women comprised nearly half of all students at several prominent medical schools. Only in the early twentieth century, coincident with the rise of scientific medicine, did the proportion of women physicians decrease dramatically. The fact that by the 1920s women practitioners made up only a small proportion of medical professionals challenges historians to address how and why the practice of medical healing came to be the domain of men, so that a "civil revolution" was required before women were again included. This article examines southern California's College

* I am deeply grateful to Rennie B. Schoepflin for his active support and insights in the research and writing of this essay. Lois W. Banner and Steven J. Ross also offered valuable comments and criticisms. Earlier versions of this paper were presented at the American Culture Association Conference, 8 March 1990, Toronto, and the American Association for the History of Medicine meeting, Baltimore, Md., 9 May 1990, where audience members provided stimulating and challenging feedback. Grants from the University of Southern California's Department of History and from the university's Institute for the Study of Women and Men in Society supported the development of this essay.