Nosology, Mortality, and Disease Theory in the Eighteenth Century

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THE way causes of death are categorized depends on the way that causes of disease are understood. In this essay I argue that our current system for classifying febrile diseases draws on an effort that was first consciously undertaken in the eighteenth century. I then describe the development of eighteenth-century disease theory and discuss the role of contagionism in the creation of separate disease entities.

Table 1, based on the London Bills of Mortality, represents the early modern lay approach to disease classification.[1] The bills were begun in 1519 in the hope of obtaining an early warning of the appearance of the plague.[2] In plague years, every London parish was ordered to appoint "searchers" to investigate every death and report any that appeared to be due to the plague. At the beginning of the seventeenth century the bills became continuous, and by 1626 other causes of death were added to the records, together with the sex of the decedent.[3] The searchers assigned causes one by one as they viewed each victim's body, drawing on their own experience and information from the decedents' families or medical attendants. They did not work from any official classification and probably received no training. The parish clerk summarized their reports and sent the

1. William Black, "A chart of all the fatal disease and causalities in London, during 75 years . . . [1701–1776]," in Black, *An Arithmetical and Medical Analysis of the Diseases and Mortality of the Human Species*, 2nd ed., (London: C. Dilly, 1789), pp. 42–43.

2. Charles Mullett, *The Bubonic Plague and England* (Lexington: University of Kentucky Press, 1956), p. 47.

3. William Black, *An Arithmetical and Medical Analysis of the Diseases and Mortality of the Human Species*, 2nd ed. (London: C. Dilly, 1789), p. 253.

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TABLE I

Chart of all the Fatal Diseases and Casualties in London, 1701–1777

Disease	*1701–1732*	*1747–1777*	*Total*
Ague	278	208	574
Fevers: malignant, spotted, scarlet, and purple	104,285	94,215	256–495[a]
Small pox	566,667	65,441	151,570
Measles	4,590	6,418	13,866
Quinsy, sore throat	395	615	1,297
Pleurisy	986	728	3,525
Rheumatism	815	303	1,468
Gout	958	1,813	3,236
Consumption	92,221	130,698	288,928
Chin cough, hooping cough, cough	748	7,007	9,573
Asthma and tissick	13,028	11,853	34,341
Apoplexy and suddenly	5,241	6,624	15,152
Palsy	882	2,041	3,544
Lethargy	231	179	526
Meagrims	23	—	23
Headach	53	18	77
Lunatick	1,025	2,174	3,876
Spleen and vapours	105	—	125
Rising of the lights	2,458	49	2,704[a]
Stoppage of the stomach	6,696	483	9,465
Vomiting and looseness	1,502	254	2,004
Cholic, gripes & twisting of the guts	24,700	2,271	29,710
Flux	378	493	971
Bloody flux	381	187	745
Worms	1,359	171	1,691
Jaundice	3,059	3,818	8,909
Gravel, stone, & strangury	1,657	850	3,205
Diabetes	85	21	125
Dropsy and tympany	27,056	13,410	70,506

TABLE I

Continued

Disease	*1701–1732*	*1747–1777*	*Total*
Livergrown	171	23	269
French pox	2,289	2,013	5,965
Scurvy	91	101	226
Evil	1,539	395	2,360
Leprosy	72	54	195
Rash	205	83	341
Itch	—	42	84
Childbed	7,454	6,191	17,057
Abortive and stillborn	Illegible	19,061	46,831
Chrisoms and infants	1,165	—	1,771
Miscarriages	—	105	152
Convulsions	206,378	174,417	492,761
Headmold-shot, and water in the head	2,983	1,359	6,355
Teeth	43,677	25,896	89,847
Thrush	2,030	2,492	6,034
Scald head	24	22	75
Rickets	5,299	216	6,569
Inflammation	75	2,288	3,061
Imposthume	1,484	265	2,130
St. Anthony's fire	73	132	241
Gangrene and mortification	3,928	6,016	13,438
Canker	319	138	580
Cancer	2,100	1,401	4,275[a]
Sores and ulcers	1,180	579	2,071
Fistula	562	253	1,025
Birsten and ruptures	619	303	1,226
Swelling and wen	6	86	139
Killed by falls, bruises, fractures, and other accidents	1,745	2,149	4,820
Self-murder	1,112	1,064	2,869
Executed	—	1,515	—[b]

TABLE I

Continued

Disease	*1701–1732*	*1747–1777*	*Total*
Drowned	2,093	3,499	7,043
Burnt	144	259	493
Scalded	51	91	191
Stifled, suffocated, and smothered	50	158	276
Overlaid	1994	509	3,799
Found dead	943	469	2,082
Grief	267	164	421
Frightened	14	15	45
Surfeits	815	58	933
Starved	17	110	223
Excessive drinking	286	258	1,222
Bleeding	149	184	397
Poisoned	7	34	48[a]
Bit by mad dogs and cats	3	21	38
Bedridden	104	161	265
Aged	62,041	47,141	139,248

[a]Addition mistake in original has been corrected.
[b]Total not given in original.

results to the clerks' hall, which issued a weekly return for all the fatalities that occurred in the city. These were compiled into an annual list.

Table 1 is based on a further compilation published by William Black in 1789. Black renamed, grouped, and often summarized the categories that he found in the annual bills, before compiling them into fifteen-year totals. Nevertheless, certain comments can be made about the original bills that he used. First, the bills were a direct result of a particular theory of the nature of a particular epidemic disease; they were initiated by lay government authorities who were attempting to uncover outbreaks of what they believed to be a contagious disease in order to isolate carriers. Thus, it is not surprising that the bills continued to reflect a lay view that certain readily identifiable, individual febrile diseases existed, although the categories

used for describing these diseases were often different from those in use today.

Second, the bills reflected an unsystematic view of what kinds of causes of death should be listed and of how different causes were related to each other. This is less evident in Table 1 because Black attempted to organize all the causes listed into categories, but it is still evident that some "causes" were actual ailments, such as "rickets," some were symptoms posing as diseases, such as "palsy," "lethargy," and "vomiting," and others were simply descriptions of certain characteristics of the patient, such as "lunatic," "bedridden," and "aged."

Although searchers might use the diagnosis offered by a medical attendant, they were not trained to recognize what sorts of causes the authorities were interested in, with the exceptions of plague and murder. Nor did they attempt to separate immediate from predisposing causes of death. The result was that two life-stage categories ("aged," for the elderly and "teeth" for the very young) covered many ailments that might have been listed differently under other circumstances, and many ailments were recorded that would not have been permitted by a more rigorous system. On the other hand, it is clear that the searchers did routinely identify certain individual febrile diseases, such as smallpox and measles, as causes of death. The preoccupation with uncovering potential cases of the plague led to an emphasis on identifying acute fevers, if only to eliminate plague as a cause in a given death.

Third, some disease categories disappeared during the eighteenth century. In the years between 1701 and 1732 there were 2,458 reports of "rising of the lights," whereas between 1747 and 1777 there were just 49. Similarly, "spleen and vapours" declined from 105 reports in the first period to none in the second; "cholic, gripes and twisting of the guts" declined from 24,700 reports in the first period to just 2,271 in the second; and "stoppage of the stomach" fell from 6,696 cases to 483. Clearly, ideas about the dangers of wandering organs, malignant humors, and mechanical obstructions were becoming less popular by the second half of the century. "Rickets" declined from 5,299 reports in the first period to 216 in the second; this probably also reflected a redefinition of the disease. Altogether, the reports of these diseases fell from nearly 40,000 in the first period to about 3,000 in the second. Reports of "worms" also fell tenfold; this may

possibly reflect more effective prevention or treatment, although a change in definition seems to be the more likely explanation.

Although there was a large numerical increase in deaths from consumption, from 92,221 in the first period to 130,698 in the last, the size of the change does not suggest a fundamental change in the concept of the disease. On the other hand, the ten-fold rise in reports of whooping cough, from 748 reports in the first period to 7,007 in the second, suggests that many searchers recognized this affliction as a separate disease only after the turn of the century. The only other important "new" disease revealed in these tables is "inflammation," which rose from 73 reports in the first period to 2,288 in the second. This may reflect a late-century substitution for part of such terms as "stoppage," that were earlier used to explain otherwise mysterious deaths.

It is evident that the categories used by lay viewers to describe the causes of death changed over the century, reflecting underlying changes in medical philosophy. It is also evident the terms used are so variable that bills are unreliable guides to the incidence of many diseases. These difficulties were noted at the time by frustrated medical arithmeticians. It is also clear that febrile diseases formed a large proportion of all the reported causes of death. Black noted that his group of 256,000 reported "fevers" alone amounted to one-seventh of all assigned causes of death.[4] If we add to the reports of "fevers" those for "small pox," "measles," "sore throat," and "whooping cough," the total reaches 418,525: nearly one-quarter of the 1.8 million London deaths in this period. In addition, contemporaries noted that many unacknowledged cases of fever were hidden in such catch-all categories as "aged" and "teeth."

Even when fevers were cited as causes of death, confusion prevailed. Black stated that he "was anxious to determine with some probability, the ratio of desolation in London, by each of the different febrile genera; because it would be an important guide to the prevention and cure." However, he complained that "In the London bills of mortality, many different genera of fevers are crammed into one indiscriminate heap, from which it is impossible to extricate the specific nature or genus of febrile carnage."[5]

4. Black (n. 1), p. 43.
5. *Ibid.*, p. 44.

It is thus clear that Black believed there were different "genera" of fevers, each of which had its own "specific nature." He hoped that disentangling these different fevers and analyzing them separately to determine the proportion of total mortality caused by each kind of fever would improve measures for both prevention and cure. In his emphasis on the need to distinguish and reclassify diseases such as "influenza" or "chicken pox," Black was drawing on a medical tradition that originated in the late Renaissance but was only beginning to find widespread acceptance among university-trained physicians at the time he was writing. It was the gradual adoption of this conceptualization of fevers as specific entities, each directly related to a particular pathogenic substance, that created the framework for our current methods of classifying and understanding diseases.

CONTAGIONISM AS A SOURCE FOR THEORIES OF SPECIFICITY

Although lay opinion in early modern Europe had often distinguished certain individual, readily identifiable diseases, academically trained physicians favored theories that discouraged such differentiation. These theories depreciated the possibility that specific febrile diseases might be contagious. Even in the eighteenth century, academic theories of fever still drew on a composite of classical medical writing known as "Galenism." This was the system of four elements, fire, water, earth, and air, four qualities, heat, cold, wet, and dry, and four humors, blood, bile, phlegm, and choler or black bile. Galenism depicted disease as the result of individual differences in constitution and manner of living. "Fever" was due to an excess of hot humors in the body. Epidemics were thought to result from a complete corruption of the atmosphere, perhaps caused by celestial influences, but individual constitutional differences determined who was susceptible to the polluted air. Thus, disease was not a "thing" but a matter of degree or a process: there were really no diseases as such, but rather a succession of diseased patients.

Galenic fever theory had often been rejected or greatly modified in the early modern period. During the Renaissance, the rediscovery of many classical medical texts and the appearance of two terrifying diseases, plague and syphilis, set off a landslide of new medical works. Some of these maintained that disease came not from an internal imbalance but from an invasion of the body by particular morbific matter that came from outside, a "contagium" that could be transmit-

ted from person to person in the same way that mold could be spread by contact from one fruit to another. This could explain why a disease such as the plague could cause the same set of symptoms in individuals with different humoral balances.

The best early summary of the case for the contagiousness of the plague was *On Contagion and Contagious Diseases and their Cure*, published by the Italian physician Girolamo Fracastoro in 1546. Fracastoro was by no means the first to propose that the plague was transmitted physically from victim to victim: European cities had been taking public health measures predicated on the idea of contagion for a century before his work appeared. Fratcastoro offered, however, the clearest discussion of the implications of contagion for an understanding of the nature of disease, and he expressed views that would be echoed and ultimately adopted in the eighteenth century. In a classic article on Fracastoro, Charles and Dorothea Singer wrote that he deserved credit for "finally and clearly distinguishing the three categories of infection, by contact, by fomites and at a distance."[6] Even more important, however, was the fact that "he enunciated clearly, perhaps for the first time, the modern doctrine of the specific characters of fevers."[7]

The doctrine of disease specificity grew directly out of Fracastoro's theory that diseases were transmitted by "seminaria," or seeds. If epidemic diseases were caused by a change in the atmosphere, then everyone breathing that air (e.g., everyone living in a given city at a given time) should be afflicted with the same disease. Those who accepted this view searched for common characteristics underlying all diseases occurring in a single area at a single time and explained any divergence in symptoms by invoking individual constitutional differences. Although Fracastoro agreed that the cause of disease might be airborne in the case of epidemics, he argued that in most cases the air only served as a medium for carrying specific seeds of disease from a particular source. The air itself had not become corrupted. Fracastoro argued that a disease was defined by its cause, and in contagious diseases that cause was the seed: "For the humours in some individual may be in a normal condition . . . and yet the plague

6. Charles Singer and Dorothea Singer, "The scientific position of Girolamo Fracastoro [1478?–1553]," *Ann. Med. Hist.*, 1917, *1*, 1–34, p. 10.

7. *Ibid.* See also Vivian Nutton, "The reception of Fracastoro's theory of contagion: the seed that fell among thorns?" *Osiris*, ser. 2, 1990, *6*, 196–234, p. 210.

may be contracted from another person. Therefore there must be present some other principle per se of that contagion. . . . The principles of contagions per se are the germs themselves."[8] The contagium *was* the disease; neither the symptoms, nor "obstruction or plethora or a malignant condition, be it quantitative or qualitative of the humours," nor the process of the disease, was sufficient to constitute it.[9]

Fracastoro's use of the word "seed" does not imply that he believed that the disease-causing particle was alive. His conception of the nature of disease-causing matter was closer to that of an enzyme than a *contagium vivum*, and his depiction of its nature eschewed vitalism. However, the idea of "seed" does imply specificity: a seed of a particular plant can bring forth only that plant itself. Moreover, it is clear that for Fracastoro these seeds were no mere metaphor: he insisted that disease particles reproduced themselves.[10] Like his contemporaries, however, Fracastoro also believed in spontaneous generation. Although the seeds of diseases were specific, they could be generated anew in both the air and the body.

Because each contagious disease was the result of a specific kind of seed, Fracastoro believed that each required "its own special and peculiar treatment."[11] Thus, it was important that a physician recognize individual diseases, and Fracastoro devoted considerable space to describing in detail those individual diseases that he regarded as contagious. Among these he included plague, poxes, measles/scarlet fever, "sweating sickness," "lenticulae" or typhus, phthisis, rabies, syphilis, scabies, certain forms of dysentery, certain scrofulous tumors, elephantiasis and leprosy, herpes, and some other skin infections.[12]

Fracastoro did not create these disease groupings but borrowed them either from the medical literature or from lay ideas. For example, he speaks of the fevers "which the translators of Arab books call 'variolae,' [poxes] and 'morbilli' [measles and scarlet fever]. By the term 'variolae,' they mean fevers which people commonly call 'varolae' . . . and they mean by 'morbilli' what people call 'fersae'. . . . The Greeks, however . . . used only the term *exanthemata*."[13] Thus, it is

8. Girolamo Fracastoro, *De Contagione et Contagiosis Morbis et Eorum Curatione*, trans. Wilmer Cave Wright (New York: G. P. Putnam's Sons, 1930), pp. 83–85.
9. Ibid., p. 83.
10. Ibid., p. 35.
11. Ibid., p. 185.
12. Ibid., pp. 71–183.
13. Ibid., p. 73.

evident that "the people" had a separate concept and term for "morbilli." "Measles," which included scarlet fever, first gained separate mention in the London Bills of Mortality in 1629.[14]

COMPETING CONCEPTUALIZATIONS OF DISEASE IN THE SEVENTEENTH AND EARLY EIGHTEENTH CENTURIES

Fracastorian theories of the plague were widely shared by lay governments, but the plague became less frequent in Europe after the beginning of the seventeenth century, disappearing from Britain after the epidemic of 1665. With its disappearance, Fracastorian contagionism drifted into the eddies of medical theory. The mainstream of the profession became preoccupied with entirely different ways of conceptualizing illness. Many professional physicians, including the members of the College of Physicians, adopted a theory known as "iatromechanism," which depicted the body as a machine filled with tiny moving particles and which attributed disease to mechanical dysfunctions caused by blockages and collisions of these particles. Such factors as "overheating" caused the particles to boil up too rapidly, raising the blood pressure and jamming the particles too tightly in capillaries. Like Galenism, iatromechanism depicted fevers as matters of degree caused by internal dysfunctions. Thus, by the late seventeenth century, physicians viewed fever as a single process with different degrees of severity and did not envision a large number of separate and distinct febrile diseases. By the time of the Marseilles epidemic of 1720, many medical authorities even doubted that the plague was contagious.[15]

Some historians view the work of Thomas Sydenham, and particularly the preface (1676) to his *Medical Observations* (1666) as an exception to this tradition.[16] Sydenham did oppose the theory that disease was "but a . . . disordered effort of Nature thrown down from her proper state, and defending herself in vain," and called instead for a natural history of diseases drawn "with the same care which we

14. E.W. Goodall, *A Short History of the Epidemic Infectious Diseases* (London: John Bale, Sons and Danielsson, 1934), p. 61.

15. Mullett, (n. 2) *Bubonic Plague*, pp. 277–81.

16. See, for example, Richard Harrison Shryock, *The Development of Modern Medicine* (New York: 1936; rpt. Madison: University of Wisconsin Press, 1979), pp. 14–15; Charles Singer and E. Ashworth Underwood, *A Short History of Medicine*, 2nd. ed. (Oxford: Clarendon Press, 1962), pp. 110–11; L.J. Rather, "Pathology at mid-century: a reassessment of Thomas Willis and Thomas Sydenham," in Alan G. Debus, ed., *Medicine in Seventeenth-Century England* (Berkeley and Los Angeles: University of California Press, 1974), pp. 85–87.

see exhibited by botanists in their phytologies," because identifiable diseases, like plants, belonged to distinct species.[17]

However, Sydenham's idea of how a "species" of disease should be constructed was different from ours. The use of a familiar word has misled modern readers into assuming that Sydenham had adopted a "modern" conceptualization of disease specificity. In fact, Sydenham's understanding of disease species depended on his own "neo-Hippocratic" theories of causation. He believed that epidemic fevers resulted from imperceptible subterranean emanations whose diffusion in turn depended on the "epidemic constitution" of the air in a particular season. This constitution influenced the symptoms in all patients who fell ill at a particular time. Among epidemic diseases were "pestilential fever" or plague; inflammatory fever, and dysentery. Initially, Sydenham claimed that smallpox, measles, and erysipelas were nonepidemic "intercurrent" diseases, but later in the same book he treated smallpox and measles as epidemic.

Each constitution engendered different "species" of disease whose individual nature was determined by the disturbance of a particular, specific fluid, or humor, in the body.[18] "Whereas all species, both of plants and of animals, . . . subsist by themselves, the species of disease depend on the humours that engender them."[19] Sydenham added that "where any particular constitution engenders any species of epidemic . . . this species is generically different from the species of any other constitution, however much one name may be current for all; whilst all the peculiar species which set in under one and the same constitution . . . [are] referable to one common and general producing cause."[20] The prevailing epidemic constitution altered all humors; a person exposed to a disease that attacks a particular humor becomes ill with a particular species of disease, but one that also manifests the tendency of the prevailing epidemic. Even the continued and intermittent fevers of the same constitution were different manifestations of the same class of disease.

17. Thomas Sydenham, preface to "Medical observations concerning the history and the cure of acute diseases," in *The Works of Thomas Sydenham M.D.*, trans. R.G. Latham (London: Sydenham Society, 1848, facs. 1979), *1*, 13, 16. Sydenham entitled this section "Preface to the Third Edition," because be considered this work to be a new edition of his *Methodus Curandi Febres* (1666), but it contained extensive additions and was published under the new title of *Observationes Medicae*, the title that is used by Latham.

18. Ibid., 19.

19. Ibid., 20.

20. Ibid., 38.

Sydenham used the term "specific" in a sense that varies from its present, loose meaning of "particular." In Aristotelian logic, each category of objects was determined both by its membership in a larger group, the genus, and by the specific characteristics—the "differentia" that separated the members of that group into discrete subgroupings. The classic example was the definition of man as a rational animal. The species "man" cannot be understood except as a combination of both the genus "animal" and the differentia "rational." Until the time of Linnaeus, plants were often named by combining a generic name with several different adjectives, the differentiae.[21] "Rational" is thus a specific term; one that divides men from other animals, but it is not a "species" in itself. To use an example from botany, there are *Rosa rugosa*, *Rosa damascena* and *Rosa centifolia*, the "wrinkled rose," "the Damascene" or "damask" rose, and the "hundred-leaved," or "cabbage" rose. These are species of the genus "Rose," but "rugosa" and "centifolia" are not species, they are simply adjectives that refer to particular kinds of plants only when used with the generic name. A "cabbage" is an entirely different plant from a "cabbage rose," which only resembles a cabbage.

Similarly, for Sydenham, "smallpox" was not a separate "species" of disease but a certain attribute of disease when a particular humor was altered. It would have been better understood as a "poxy" or even "rashy" symptom of the disease of a given year. The actual species was formed by a combination of that kind of humoral response with a particular epidemic constitution, the smallpox of the years 1667, 1668, and part of 1669. Both the constitution (the genus) and the affected humor (the cause of differentiation) together constituted the actual "specific disease." It is misleading, then, to state that Sydenham believed that disease came from outside the body, in contrast to Galenic physicians who believed it represented an internal humoral imbalance. For Sydenham the *constitution* came from outside the body, but the specific disease came from a combination of a given genus of constitution with a particular humoral modification.

The smallpoxes of two different constitutions resembled each other just as plants whose leaves have been eaten by different caterpillars may look the same, but the only connection between the two diseases

21. A.J. Cain, "Logic and memory in Linnaeus's system of taxonomy," *Proc. Linn. Soc. Lond.*, 1958, *169*, 144–62; Lester King, *The Medical World of the Eighteenth Century* (Chicago: University of Chicago Press, 1958), pp. 194–96.

was that in each case the epidemic constitution happened to attack the same humour, just as two different caterpillars might both prefer to eat a particular part of a plant. During the same year, the constitution would also attack other humors, resulting, for example, in the continued fever of the years 1667, 1668, and part of 1669, which was of the same genus as the smallpox of the same years. Each epidemic constitution created unique diseases that were not the same as similar-appearing diseases encountered in subsequent years, just as a hot year might produce one kind of leaf-eating caterpillars, and a cold year might produce another kind. The first sort might attack only the leaves and roots, whereas the second sort might eat the leaves and bark. In both cases, the plants would have chewed leaves, but the chewed leaves of 1667 would have an entirely different cause from the chewed leaves of 1675 and would require different methods of prevention. The plants would not have the same disease: chewed leaf disease would not exist as a separate entity in itself. In a particular year, both the plants with chewed leaves and those with chewed bark could be rescued by destroying a single sort of caterpillar.

Although injured bark and injured leaves in the same year might share the same cause, they would be different sorts of ailments, provoking a different defensive reaction from the plant itself. It was actually the defensive reaction, not the initial provocation, that produced the disease seen by the physician. The closest modern medical comparison would be to cancer: Kaposi's sarcoma and melanoma are separate diseases, yet both are called "cancer." In each, an event causes a particular sort of alteration in a particular seat, but the two precipitating events are unrelated to each other. The "cancer of 1995" might be caused by an increased amount of sunshine, which might also cause an epidemic of sunburn, whereas the "cancer of 1996" might result from an increased level of HIV infection. Both the cancer and the sunburn of 1995 are alterations of the victim's body in response to the same source of irritation; neither could exist as objects outside the body.

Sydenham also identified what he called "intercurrent" and chronic diseases. The intercurrent diseases were fevers that sometimes became epidemic, but normally occurred at a relatively constant rate unrelated to the epidemic constitution. These were usually due to individual acts, especially a premature change of dress and exposure to cold after exercise. However, they could also be caused by an extreme change

of air temperature, and because this could affect many people at once, these diseases sometimes appeared as epidemics. The same symptoms might also appear as complications of the epidemic diseases. Among these Sydenham listed scarlet fever, pleurisy, "bastard peripneumony," rheumatism (rheumatic fever), erysipelas, and "quinsy." Because the most common cause for an intercurrent disease was an individual error, there was no physical connection between two victims of these diseases.[22]

During the early part of the eighteenth century, three competing ways to describe fever gradually emerged. The first, an evolution of iatromechanism, viewed fever as a process that involved an imbalance within the body. This increasingly became a simple polarity rather than the complex quartile degrees of Galenism. The body was either too hot or too cold; its fluids were either too rapid or too torpid; later in the century it was thought that its nerves and fibers were too tense or too relaxed. This scheme lumped many fevers together into two or three fundamental categories such as "putrid" and "inflammatory" fevers.

The second scheme was a neo-Hippocratic approach, depicting fever as the result of the interaction between an individual and the predominant atmosphere he or she lived in. Different sorts of environment, combined with different humors, produced different fevers, so diseases were split into "marsh fever," "ship fever," "camp fever," "jail fever," "hospital fever," "Hungarian fever," "Uppsala fever," "sea-scurvy," "land-scurvy," "bilious fever" (disordered bile), or "cholera" (disordered choler). The third scheme was based strictly on symptoms, particularly the period of the fever and the appearance of any rash; this produced the various intermittent fevers (quartan, tertian, and semitertian), continued and relapsing fever, spotted fever, petechial fever, miliary fever, scarlet fever, yellow fever, whooping cough, the poxes, and "sore throat."

Most physicians adopted an idiosyncratic mixture of all three approaches. The potential for diagnostic chaos is obvious. The confusion in describing fevers was even worse than it was in botany because it was possible for botanists who wished to compare notes on a given plant to exchange seeds and specimens. Patients with acute diseases were not so easily pinned to a page. Moreover, physicians saw these

22. Sydenham, (n. 17), pp. 239–40.

categories as mere intellectual constructs, as names used for convenience but not representing real underlying entities. Under the right conditions, such as a change in the weather, one disease could become another. Whereas most botanists agreed that there were fixed species of plants, many physicians believed that there were as many kinds of diseases as there were patients.

THE EIGHTEENTH-CENTURY REVIVAL OF CONTAGIONISM AND SPECIFICITY

In the early eighteenth century, a series of events that included epidemics of smallpox and plague led to a gradual transformation in the way diseases were viewed. The reappearance of plague at Marseilles in 1720 evoked a torrent of works, including several that revived traditional contagionism. Among these was Richard Mead's *A Short Discourse Concerning Pestilential Contagion, and the Methods to be Used to Prevent It* (1720). In 1721, there was an epidemic of smallpox, and for the first time the British experimented with inoculation, championed by Hans Sloane (later President of the Royal Society). As Genevieve Miller has shown, the introduction of inoculation gave a great impetus to contagionism.[23] For the first time, it was possible to demonstrate that a particular physical substance taken from one patient caused a disease process in a second patient that was nearly identical to the first. The idea that smallpox matter caused only smallpox, and its converse, that smallpox resulted only from contact with smallpox matter, gained increasing acceptance.

Yet another event passed almost unmarked: the publication in London in 1720 of Benjamin Marten's *A New Theory of Consumptions: More Especially of a Phthisis or Consumption of the Lungs*. Drawing on the work of Anthony van Leeuwenhoek and the parasitologist Nicholas Andry de Boisregard, Marten developed the first fully articulated theory of *contagium vivum* as a cause for human diseases. Marten noted that this theory accounted for the fact that there were regular variations among different diseases such as the plague, pestilential and other fevers, small pox, measles, and several other diseases. "How can we better account for the regular Types [of acute disease] . . . than by concluding they are severally caus'd by innumerable Animalcula

23. Genevieve Miller, *The Adoption of Inoculation for Smallpox in England and France* (Philadelphia: University of Pennsylvania Press, 1957), pp. 258–60.

. . . that variously offend us according as their Species are different."[24] Marten's work ran to a second edition but apparently made little impact on the medical profession. However, the next year a botanist, Richard Bradley, F.R.S., and a client of Hans Sloane's, published a tract that argued that the plague was carried by minute insects. Bradley argued that swarms of tiny disease-causing insects flew through the air and that each species of insect sought its own proper "nidus," or nest to lay its eggs. These eggs were then dispersed by the victim and infected new patients.[25]

In 1730, Thomas Fuller, a country physician and expert on pharmacy, published his *Exanthematologia; or, An attempt to Give a Rational Account of Eruptive Fevers, Especially of Measles and Smallpox*, dedicated to Hans Sloane and the Royal Society. Like Marten, but in more detail, Fuller defended the thesis that many fevers were contagious and that contagion also entailed disease specificity. Unlike Marten, however, Fuller did not unambiguously ascribe these fevers to "animalcula," referring as well to "particles," and "seeds." He denied that the air alone could generate disease: this must result from small particles, and these particles must act only by contact within a "determinate Sphere of Activity."[26]

> The Chicken-Pox, and several others . . . are Distempers that have all of them a distinct material Cause, and therefore differ not only in Degree, but in Essence also from one another. . . . The Particles which constitute the material and efficient Cause of the Small-Pox, Measles, or other venomous Fevers, are of specific and peculiar Kinds; and as essentially different from one another, as Vegetables, Animals, and Minerals of different Kinds are from another.
>
> And since . . . no Effect can be produced but by its own proper Cause, I am hard to believe, that the Small-Pox or Measles can be produced by such Things as have no manner of Affinity with them; such as are Fevers of any other Sort; cadaverous Steams from Men that dy'd of other diseases; from putrefy'd Carrion; Exhalations from fermenting Minerals; . . . [or]

24. Benjamin Marten, *A New Theory of Consumptions* (London: R. Knaplock, A. Bell, J. Hooke and C. King, 1720), p. 65. See also Catherine Wilson, *The Invisible World: Early Modern Philosophy and the Invention of the Microscope* (Princeton, N.J.: Princeton University Press, 1975), pp. 164–68. A similar etiology had been suggested for rinderpest, a cattle disease, by Carlo Francesco Cogrossi in 1714.

25. Richard Bradley, *The Plague at Marseilles Considered* (London: W. Mears, 1721). See also Wilson, (n. 24) *The Invisible World*, pp. 161–64.

26. Thomas Fuller, *Exanthematologia* (London, 1730), p. 190.

Earthquakes; nor from foul ways of living, Nastiness, corrupt Meats and Drinks.

My Notion of this matter is that the variolous Seminaria are as special and cognate to that Distemper, as Seeds are to their proper Vegetables and cannot possibly by any Power of Nature, produce anything but the true Small-Pox any more than Thistles can Figs.[27]

Fuller believed, as had Marten and Fracastoro, that disease was an entity that had a separate existence as a "seed" outside the body. One individual sort of disease could not become another. Moreover, unlike Fracastoro, Fuller denied that disease-causing seeds could be spontaneously generated by combinations of matter, although he did argue that they existed in the upper atmosphere and rained down on Earth.

Fuller's adoption of contagionism was directly responsible for the fact that he was the first medical writer to clearly differentiate between chicken pox and smallpox. Moreover, his theories led him to become an early supporter of smallpox inoculation. Fuller and Marten apparently developed their theories independently, but their works, with those of Mead and Bradley, sought an audience within the Royal Society.

THE NEW CLASSIFICATIONS OF THE EIGHTEENTH CENTURY: SAUVAGES, LINNAEUS, AND CULLEN

In the first half of the eighteenth century contagionists were an embattled minority. After the middle of the century, however, physicians both expanded the list of contagious diseases and constructed new diseases out of symptoms previously noted but not grouped in this manner. In an earlier article, I traced the way that this process worked in the case of influenza. Here I consider nosologies, which attempted to classify disease on a larger scale. By comparing them, we can see how contagionism gradually redefined the confused category of fevers, even though it often remained in the background as an organizing principle.[28]

27. Ibid., p. 95, and quoted in Ludvig Hektoen, "Thomas Fuller 1654–1734, country physician and pioneer exponent of specificness in infection and immunity," *Bull. Soc. Med. Hist.*, 1917–22, *2*, 328.

28. After completing this article, I obtained a copy of Lester King's *Transformations in American Medicine: From Benjamin Rush to William Osler* (Baltimore, Md.: Johns Hopkins University Press, 1991) and found that the following argument closely parallels his. However, I differ with his conclusions, particularly concerning Cullen's contribution and the overall nature of eighteenth-century achievements. See also Margaret DeLacy, "The conceptualiza-

Two early nosologists, François Boissier de Sauvages de la Croix and Carolus Linnaeus, were botanists seeking to fulfill the demand for a "natural history" of diseases comparable to the natural histories of plants. Sauvages had a traditional, multifactoral theory of disease: he wrote that

> the febrile or disease-causing matter is of several sorts (*espèces*): often it is a corrupt chyle, which clogs the capillary vessels by its viscosity, or that irritates and contracts the blood vessels by its acrimony. Often . . . [morbific particles] come from miasms, or form themselves in the blood, by the suppression of ordinary evacuations, particularly by suppression of transpiration; prurulent essences, or corrupted fluids, or they are products of harmful qualities of the air, of food, or of drink, which alter the mass of the blood.[29]

Sauvages first published a symptomatic nosology in 1731, the *Nouvelles Classes des Maladies*. In 1763 he added to the work by subdividing each "genus" into "species." This created about 2,400 species of disease. In the meantime, Linnaeus, who corresponded with Sauvages, developed a modification of this system to use as the basis for his own teaching. In 1759 he published it in the name of one of his students and in 1763 he published it himself as *Genera Morborum*.

Although the two works are superficially quite similar, there are significant differences, particularly in the treatment of fevers. Sauvages tried to classify fevers literally in the same way that botanists classified plants, only by their appearance. In the case of diseases, this meant their symptoms. Each separate symptom, in effect, became a separate species of disease. Sauvages explicitly rejected any effort to classify diseases by cause or by "seat." Because the most important symptom was the pattern of the fever itself, he established three orders of fevers in his "class II: fevers"; these were "continued," in which the fever increased and subsided only once during the course of the disease; "remittent," in which the fever increased and decreased several times during the course of the disease; and "intermittent," in which the fever disappeared and returned several times during the course of the disease. Eruptive diseases such as plague, measles, and "purples" were

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tion of influenza in eighteenth-century Britain: specificity and contagion," *Bull. Hist. Med.*, 1993, *67*, 74–118. For a contrasting view, see Christopher Hamlin, "Predisposing causes and public health in early nineteenth-century medical thought," *Soc. Hist. Med.*, 1992, *5*, 43–70.

29. François Boissier de Sauvages, *Nosologie Methodique . . . suivant le système de Sydenham . . . trans. from Latin*, 3 vols. (Paris: Herissant le fils, 1771), my translation, p. 341.

placed in class III, "inflammations," where they formed the first order. The second order of this class consisted of inflammations of membranes, such as pleurisy and enteritis, and the third of inflammations of organs such as hepatitis and nephritis. Thus, he regarded the exanthematic diseases as a sort of generalized inflammation different from the periodic fevers.[30]

The result of Sauvages's multicausal view of fevers is that there is no hierarchy of symptoms, no way of establishing which symptoms are definitive characteristic features of a disease and which are accidental. There are as many disease species as there are symptoms, and the same symptoms may appear in several different categories. A "continued" catarrhal fever, and a "remittent" catarrhal fever appear in different classes, as do the "fever resulting from the third stage of scurvy" and scurvy itself, or buboes and plague. Without an etiology, there was no way of distinguishing between the continued fever that characterized typhus and the fevers that were caused by exercise, cured by music, or created by a performance of Euripides. Synonyms for the disease that we now call typhus, which was then known by terms such as "Hungarian fever," "slow nervous fever," "continued fever," or "camp fever," crop up in many different headings, each as a separate species, with no suggestion that they might refer to the same disease or that they might be related to each other. In the order of remittent fevers (order II of class II: "fevers") they become hopelessly confused with terms for "tertians," a variety of malaria, and also with the complications of other diseases such as the fever that occurred as a complication of confluent smallpox.

The system developed in Linnaeus's *Genera Morborum* appears almost identical to that of Sauvages. Nevertheless, Linnaeus's classification is infused with a different spirit, for Linnaeus's categories of fever followed his views on etiology. Linnaeus began his eleven classes with three classes of fever: first, "exanthematici," which included such diseases as smallpox, plague, measles, "petechia," and syphilis; second, "critici," which included the three orders of continued, intermittent, and remittent fevers, and third, "phlogistici" or inflammations. Linnaeus believed that these three classes corresponded to three general external causes. The fluids in the body could be altered by both acid and putrid ferments. The acid ferments acted on the serum in the

30. Ibid.

body and caused the "critical" fevers (class II), whereas the putrid ferments acted on the blood and caused the "phlogistici" (class III) or inflammatory fevers. Most of the "exanthematic" fevers, however, were due to specific *animalcula.*[31]

In the thesis *Exanthemata viva* of 1757, republished in 1760, Linnaeus argued that all contagious diseases share certain features: they all "blossom out into Exanthemas internal or external," they all caused restlessness and fever, and they all responded to medications that killed insects, such as sulfur and mercury. Epidemic dysentery was an "internal itch of the intestines," caused by mites related to those that caused scabies. Other such diseases included whooping cough, smallpox, measles, plague, and venereal diseases. He did classify some exanthemata as non-contagious because he believed that some species of the causative animalcules could persist in the environment without human hosts.[32]

There were several sources for Linnaeus's contagionism. Although he spoke no English, Linnaeus had visited England and maintained close, lifelong ties with the circle of botanists surrounding Hans Sloane. The *Exanthemata viva* mentions a number of contagionist authors including Fracastoro, and it is evident that Linnaeus was familiar with much of the earlier medical discussion of this issue. However, he also drew on personal experiences, lay attitudes, developments in microscopy, and the experience of smallpox inoculation. Although Linnaeus placed contagious exanthemata first in the *Genera Morborum*, he never fully worked out the implications of contagionism for disease classification. His own disease terminology is confused and varies from work to work, perhaps because of the influence of collaborative authorship.[33]

After the middle of the century, a growing acceptance of contagionism gradually helped to sharpen the definitions of individual disease

31. Richard Pulteney, *A General View of the Writings of Linnaeus* (London: T. Payne and B. White, 1781), pp. 171–200. See also Frederik Berg, "Linnés Systema Morborum," *Uppsala Universitets Årsskrift*, 1957, *3*, 1–132; and Frederik Berg, "Linné et Sauvages: Les rapports entre leurs systèmes nosologiques," *Lychnos*, 1956, pp. 32–54.

32. Margaret DeLacy and A. J. Cain, "A Linnaean thesis concerning *Contagium Vivum*: the *Exanthemata Viva* of John Nyander and its place in contemporary thought," *Med. Hist.*, 1995, *39*, 159–85. Although it was published as a thesis by one of Linnaeus's students, John Nyander, Linnaeus should be considered the author of *Exanthemata Viva.*

33. Ibid.

complexes including typhus, which was gradually "constructed" out of the welter of jail, camp, hospital, ship, Hungarian, continued, slow nervous, petechial, spotted, putrid, and other fevers. Once they began to look for the evidence, administrators saw that newcomers often "brought" these fevers from jails to ships, or from ships to hospitals, and started to attribute outbreaks in different institutions to the same disease. Because they increasingly believed that typhus was a single contagious disease, they also hoped they could isolate victims and prevent its spread.

The result of this process can be seen in the work of William Cullen, the most important eighteenth-century nosologist. Like the other nosologies discussed, Cullen's classification is ostensibly based on symptoms, not causes. Indeed, Cullen commented that Linnaeus "appears to every body else extremely ridiculous when he arranges syphilis among fevers, merely from the circumstance of their being both contagious."[34] Yet Cullen's own etiological theories influenced his system at every stage and were explicitly discussed in his "Introductory Lectures on Nosology." Cullen noted that earlier nosologists "have begun at the wrong end. Their first attempt was to form the classes and orders with which they began their system . . . [but it] is from our accuracy with respect to the species that all the rest of the divisions can be properly established."[35]

Cullen believed that the greatest difficulty in nosology was to distinguish between the species and the varieties of diseases. He compared this with the work of other natural philosophers. Botanists were able to distinguish between varieties and species by planting the seeds of plants to see if they bred the same plants again; this had reduced the number of species by nearly half. He wrote that in studying diseases, nosologists could

find something analogous to the propagation of seed in living bodies. We observe this in the case of all contagions, particularly in those we call specific contagions; and, as far as my observations goes, even in those which are not strictly specific, when we can trace a disease to its contagion, we can, in some measure, fix its species. Thus, in the case of small-pox, a great

34. William Cullen, *The Works of William Cullen, M.D.*, John Thomson, ed., *Works*, vol. 1 (Edinburgh: William Blackwood and London: T. & G. Underwood, 1827), 448.
35. Ibid., p. 449.

many varieties have been marked, but they are varieties only of one species: a proof of which is, that from the same contagion, that is, from the same seed, all the essential circumstances of the disease are produced.[36]

Thus, for Cullen, a species of acute disease was a specific group of symptoms caused by a particular species of matter, and the same matter would always cause the same disease even if the symptoms varied. He argued that his predecessors had erred by seeing different degrees of the same sort of disease as totally different, by depicting what were only varieties of certain diseases as distinct genera and in making the complications of a disease into separate diseases.[37] The separate character of the disease arose from its relationship with a particular cause. Species of diseases did not descend, so to speak, from a generic font.

For Cullen, higher classifications were always provisional, created only for the convenience of students. A disease genus was not a "thing" that was to be divided by differentia and had no "natural" preexistence. Instead, the unit of classification was the species, the bundle of symptoms generated by a single specific cause. In the case of acute diseases, individual species of matter caused individual species of disease. He noted, however, that a number of his genuses, such as "tertian fevers," also constituted species because they could not be further subdivided except into varieties.

Cullen admitted that he was unable to construct an entirely new nosology as he wished; he felt he was too dependent on the work of Sauvages. However, he reduced or eliminated many categories and diseases. In particular, he eliminated the remittent fevers, arguing that they should be considered a species of "intermittents" because they had the same cause, marsh effluvia, they appeared at the same time and in the same climates, and they were cured by the same remedy, cinchona. Of the remaining fevers, he established three genuses: "inflammatory fevers" or "synocha" which were brief, lasting less than a week, and were probably not contagious; typhus fevers, which were certainly contagious and resulted from human effluvia; and a third, uncertain genus of "synochus," probably also typhus but with less clear-cut symptoms. Cullen noted that the first account of typhus was given by Fracastoro, whose original description, quoted at length,

36. Ibid., pp. 451–52.
37. Ibid., pp. 458–61.

was identical to his own delineation.[38] He divided his genus of typhus into two species: petechial typhus and "typhus icterodes" (yellow fever). The former he divided into just two varieties, "mitior" and "gravior."

Cullen commented that the species of typhus were not "well ascertained by observation" and many of the different cases observed seemed to be merely varieties. For example, the presence of bile, a putrescent state of the fluids, or the appearance of inflammatory complications such as pleurisy, did not constitute different species of typhus. Thus nearly all the putrid, pestilential, bilious, continued, camp, and spotted fevers were combined into a single species, "typhus with petechiae."[39]

Cullen believed that there were only two general causes of fever: marsh miasma and human effluvia. Therefore, there were only two kinds of generalized fever, intermittents (malaria) and continued fevers. The exanthemata arose from specific sorts of effluvia, each one constituting a separate disease and spread by contagion. Among the exanthemata he listed smallpox, measles, chicken pox, scarlet fever, plague, erysipelas, "miliaria" (miliary fever), "urticaria" (the "febris amphimerina" of Sauvages), pemphigus (vesicular fever), and aphtha (ulcerative rash in the mouth). A few other diseases were scattered in other classes. These included syphilis, leprosy, whooping cough, influenza, elephantiasis, "trichoma," and dysentery. He believed that because the number of specific contagions was limited, the number of species of fever was likely to remain small. Fevers could not be attributed to temperaments, habits of life, food, drink, weather, or other traditional causes, although these, and particularly heat and cold, could increase susceptibility or moderate the course of the disease.[40]

Cullen distinguished between symptoms that helped to define the initial disease, which he called idiopathic, and those that appeared as a result of the disease process but were not part of the original disease itself, which he called symptomatic. For example, dysentery might appear as a characteristic, idiopathic feature of epidemic dysentery,

38. Ibid., pp. 522–23.

39. Ibid., p. 525.

40. Ibid., p. 256. For a contrasting view of Cullen, see William F. Bynum, "Cullen and the study of fevers in Britain, 1760–1820," in *Theories of Fever from Antiquity to the Enlightenment*, W. F. Bynum and V. Nutton, eds., *Med. Hist. Supp. I* (1981), pp. 135-47.

but it also appeared as a symptom in other diseases where it was not a defining characteristic, such as the dysentery that accompanied pregnancy, syphilis, or scurvy. Many of Sauvages's "species" appeared in Cullen as varieties, examples, or "symptomatic" symptoms.

It was Cullen, above all, who formed our conception of individual specific diseases, in which certain small groups of symptoms that appeared together with high frequency in a particular illness served as defining characteristics. Even when the cause could not be identified, the concept of a contagium shaped ideas about how to group symptoms into separate species of acute diseases. Even though there was always resistance to his classification from miasmatists who did not believe in individual species of fevers, Cullen's classification made it possible for physicians to correspond and compare notes using generally accepted categorizations of disease. His system became the standard for medical arithmeticians and epidemiologists at the end of the century and provided the philosophical foundation for the classification established by William Farr in the next century.

Pathological anatomy, which reached its peak in the middle years of the nineteenth century, is usually awarded the credit for establishing our current conceptualization of individual diseases, but the reordering of disease categories in the eighteenth century with the assistance of contagionist theories of transmission was an equally important step in that process.