

# ANIMALS, DISEASE, AND MAN

*making connections*

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**ABSTRACT** The intricate causal relationships between disease in man and disease in animals first began to be elucidated in the mid-19th century. Although the connections between animal and human disease are now generally understood, individuals as well as societies remain slow to act on this knowledge. This paper examines the gradual recognition of these disease connections and explores the parallel theme of man's reluctance to appreciate the implications of these connections. It identifies factors that have inhibited the realization of the links between disease in man and animals, and discusses several milestones in the scientific elucidation of these links. Beginning with emerging concerns over the relationship between bovine and human tuberculosis in the 1860s, it follows the discovery of insect vectors, animal reservoirs, and the links between animals, influenza, and man. Despite warnings of the potential significance for human disease of patterns of changes in the relationship with animals and the natural world, scientists have continued to treat human and animal health as largely independent disciplines, while historians too have neglected this important aspect of human disease.

ON A NOVEMBER EVENING in the early 1930s, a woman named Hanna Belin entered a reputable cafe in the French city of Tours in search of refreshment. She was still young, vigorous, active; medically qualified and newly mar-

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ried. She was in a hurry. Her attention was caught by the counter display of oysters. They were so beautiful, green against the translucent ice, and she was tempted to a treat she had often indulged in as a student, a plate of oysters and a glass of white wine. A few days later she began to ail. Her condition deteriorated and typhoid was diagnosed. Five agonizing weeks later, she was dead.

By one of those strokes of ironic fate, Hanna's husband was director of the local bacteriological institute. Deeply traumatized by the experience of his wife's illness and premature death, Marcel Belin set out to study the connections between shellfish consumption and typhoid in humans. He published the result of those researches in 1934, in a book that condemned the French government for political expediency in not ensuring that the shellfish industry be subjected to hygienic regulation (Belin 1934). In a remarkable introductory section, Belin set out the cultural issues surrounding shellfish consumption, social and gastronomic pleasure, and the assessment of personal risk. In a passage redolent of memory and experience, he described a hypothetical dinner at which shellfish had been consumed, and a sea trip during which raw mussels were eaten, bought from a retailer at the quayside. How could it be, he asked, that death derived from such enjoyable activities, activities that had given their participants such a heightened sense of well-being and of life's bounty, activities that had been enjoyed many times before (pp. 7–8)?

When Hanna Belin died, it had been known for nearly 40 years that typhoid could be contracted by eating shellfish taken from waters polluted by human sewage. This was just one byway in the pathways of disease transmission discovered to run between animals and humans at the end of the 19th century. Despite the historic and intimate relationship between disease in man and disease in animals (Diamond 1997), the existence and nature of the relationship only began gradually to be elucidated in the years after 1850. Intellectually, socially, and politically, many factors delayed popular and scientific recognition of the importance of these connections. These factors remain influential today. As her husband acknowledged, Hanna's case demonstrates three elements of human behavior: first, that social and gastronomic memory and experience favor the assumption of a benign and harmonious interchange with the natural world; second, that knowledge and education do not prevent the decisive operation of that assumption in the personal assessment of risk; and third, that governments do not necessarily take steps to preserve their peoples from known health risks if it means interfering with the interests of agriculture and industry.

A further irony in the Belins' tragedy lies in the fact that it took place in France. France was the country in which the science of comparing human and animal disease had its birthplace, and where the dangers to man of infection transmitted in foods of animal origin was first recognized. It was in France, in the later years of the 18th century, that the interest in veterinary science and comparative medicine emerged, an interest that was, eventually, to elucidate the multiple pathways of disease transmission between animals and man. The four-

dation of veterinary schools in France in the 1760s—an example briskly followed across Continental Europe, less briskly in Britain—led to the development of an ideal of comparative medicine as distinct from comparative anatomy. By the late 20th century, “comparative medicine” had come to be understood as the comparison of the clinical, epidemiological, and laboratory aspects of diseases in animals with analogous diseases in humans. In earlier times, the term was used more loosely to describe a medicine that sought to elucidate general principles of disease processes through the comparative study of human and animal epidemics, and of human and animal pathology (Wilkinson 1992).

The developments in France took place against a mixed cultural background. In the past, man and domestic animals lived in close association, and their medical treatments often overlapped. Both humans and animals were often observed to suffer in epidemics, even if their symptoms were dissimilar (Bynum 1990; Porter 1993). However, direct connections were not necessarily drawn. Meteorological conditions, for example, were considered a potent force for ill-health, and would naturally affect both man and beast (Watson 1843). Among ordinary working people, the possibilities of some disease transfers between humans and animals were certainly recognized. Eighteenth-century English farm workers knew that cowpox could harmlessly infect humans, and that the infection protected against smallpox. It was this folk tradition that led Edward Jenner to vaccination. By the 1880s, indeed, many British medical men believed that human and bovine variola were “one and the same disease” (Fleming 1881, p. 1). Meanwhile, the term zoonosis, which had originated in Germany, entered English medical and veterinary dictionaries. In the 1860s, it carried a double meaning, as an animal disease or an animal disease in humans. Despite the dictionary entries, this term did not become commonly used until after World War II (Fiennes 1978). It was not, for example, used by the English veterinarian George Fleming in his publications on veterinary public health in the 1870s and 1880s.

The local understandings of the possibility of disease transmission between animals and man existed within a cultural context that inhibited a wider intellectual realization that disease phenomena could be similar and therefore comparable in humans and animals, and that there might also be a direct, causal relationship between them. Increasing urbanization from the mid-18th century had begun to place a greater distance between man and animals (Porter 1993). Moreover, the long cultural dominance of Christian theology in Western Europe had engendered a highly anthropocentric view of the natural world. Man was seen as a being apart, unique in possession of mind and spirit, and of the power of reason. Man was God’s chosen instrument for the regulation of other forms of life. The anthropocentric tradition of the Christian Church drew an “insuperable line” between man and the animal kingdom (Thomas 1983). Although Jeremy Bentham had breached the line as early as 1789, when he defined the criterion for the treatment of animals to be whether they could suffer, rather than whether they could reason (Maehle and Trohler 1987), the long-established belief in an

absolute distinction between human and animals remained well entrenched. Popular and religious reaction to Charles Darwin's *On the Origin of Species* (1859) testified to the widespread, deep-rooted survival of the concept of human uniqueness. And it had ramifications. As the Edinburgh physician Lauder Lindsay noted in 1874, there remained an inveterate tendency to differentiate man from all other animals. Man was held to occupy a uniquely different zoological platform in respect of his anatomical structure, functions, mind, and soul, and also of his diseases (Hardy 2002).

For many scientists, as well as for ordinary people, accepting the idea that the diseases of humans and animals might be causally related required considerable cultural and intellectual readjustment. Although some diseases, including rabies, glanders, and anthrax, had long been known to be transmitted from animals to man, these diseases were transmitted through the "artificial" process of inoculation—through the bite of a rabid dog, or the entry of glandered pus or anthrax dust through abrasions in the skin. The same could be argued for cowpox. What has been called the "biological exchange" of contagion between animals and man had not seemed to exist. None of the known major human infections—smallpox, measles, whooping cough, scarlet fever, typhoid—had been observed to transmit to animals; nor had rinderpest, foot-and-mouth disease, or distemper been seen to transmit to man. Influenza, it was true, had been observed in both humans and horses, but since the epidemics did not necessarily coincide in time, it was long thought that both occurrences were due to some mysterious meteorological condition (Sisley 1891). As for worms, tapeworms, flukes, and trichina, whose transmissibility between species had been recognized in the 1860s, these were viewed as parasites rather than diseases. Like leeches, they could be seen as independent entities, accidentally parasitic on a range of creatures.

Moreover, the use of animals to study the problems of human disease was well established on the continent of Europe by the middle of the 19th century (Bynum 1990). Animal experiments became the crucial technique for the study of the life processes, including the study of pathology. In a rapidly expanding medical research culture, the major diseases of humankind naturally attracted attention. Among these diseases, tuberculosis occupied a special place as the disease that above all others brought suffering and premature death in contemporary society. In England and Wales, for which moderately reliable cause-of-death statistics are available for this period, tuberculosis in all its forms was the leading cause of death for most of the 19th century (Smith 1988). Tuberculosis was the first disease to break the conceptual barrier distinguishing the diseases of man from those of animals.

#### **TUBERCULOSIS IN ANIMALS AND MAN**

In the mid-1860s, research undertaken in France, and subsequently elsewhere, into the causation of tuberculosis introduced a deep uncertainty into European

ideas about the relationship between man, animals, and disease. In Northern Europe, tuberculosis was popularly considered to be an hereditary disease. In Southern Europe, however, it was considered to be contagious, and suspicions had long been current in the northern medical community that this might indeed be the case (Guillaume 1986). In the 1860s, two French scientists confirmed that tuberculosis was a contagious disease. The military physician Jean-Antoine Villemin demonstrated the transmissibility of tuberculosis, reproducing the disease in rabbits and guinea pigs by inoculating them with tuberculous material taken from the human lung, and then further inoculating healthy rabbits with material from his diseased ones. The pathological results of similar experiments with material from a tuberculous cow convinced him that bovine and human tuberculosis were identical. Villemin's results, which were made public in 1865, were received doubtfully by the French Academy of Medicine. As one observer wryly noted: "a certain degree of hesitation, and even of unwillingness, appears to have been evinced then, and for some time subsequently to accept what Villemin had no doubts whatever was substantially correct; and when his experiments were repeated by others, it was sought to ascribe their success to anything rather than infection" (Fleming 1874, p. 472). Four years later, experiments published by the veterinary surgeon Jean-Baptiste Chauveau, who had been investigating the communicability of various infections via the digestive system, confirmed and extended Villemin's hypothesis. In a series of scrupulously designed feeding experiments on carefully selected calves, Chauveau was able to demonstrate that the consumption of tubercular meat, and of milk from tuberculous cows, did indeed cause tuberculosis. Chauveau was the first to suggest that bovine tuberculosis was a danger to humans and to indicate that milk consumption was especially dangerous. His work was subsequently endorsed by other scientists. It was reinforced by the discovery of human cases of tuberculosis linked to the consumption of known tuberculous milk in families otherwise free of the disease (Fleming 1874, p. 481).

The link between human and bovine tuberculosis remained a matter of scientific dispute for the next 25 years, and cast long shadows into the 20th century. The subject was first introduced into Britain in 1874, when George Fleming published a paper on "The Transmissibility of Tuberculosis" in the respected *British and Foreign Medico-Chirurgical Review*. It was, he noted, "one of the most interesting and important and . . . urgent, questions of recent times" (Fleming 1874, p. 461). Fleming had studied the European literature thoroughly. He described the researches of Villemin and Chauveau and their followers in detail, setting out the evidence linking tuberculosis in humans with tuberculosis in animals, and especially in cattle. In Britain, as elsewhere, the subject generated bitter debate, in which veterinarians were often pitted against medical men, and beset by political pressures. As Fleming later noted:

Since Villemin's discovery, veterinary surgeons have found themselves in a very difficult position, they being, as it were, between Scylla and Charybdis: urged, on the one hand, to completely exclude from public consumption the flesh of tuberculous cattle, which science has shown to be infective; and solicited, on the other hand, not to sanction so radical a measure, as the instances in which human health has suffered from its use must necessarily be very rare, and the danger proposed to be averted being yet but little appreciated by the great majority of the public, such a vigorous measure would not be approved. (Fleming 1883, p. 130)

### PATHWAYS OF INFECTION

Despite the arguments surrounding bovine tuberculosis, the possibility of a pathway of infection via foodstuffs derived from diseased animals to man had, for some, at least, been demonstrated. In 1876, Robert Koch published proof of a specific causal organism for anthrax, and hence of the existence of specific causal organisms for specific diseases. By 1900, the transmission of disease through infected foodstuffs was well on the way to being established. Already in 1880, the British microbiologist Edward Klein suspected that an outbreak of severe gastroenteritis was due to an organism found in a ham consumed by the victims. In 1888, August Gaertner isolated a bacterium which he called *b. enteritidis* from the meat and blood of a cow, emergency slaughtered because of persistent diarrhea. Fifty-nine people fell ill after eating her meat, and one died. The bacterium was also isolated from the organs of the fatal case (Dewberry 1959). Gaertner's discovery marked the beginning of a long trail of scientific research. By 1990, more than 2,300 related organisms, associated with a wide range of animal species, had been identified; some 150 of these have proved pathogenic to man (Cliver 1990). In the 1920s, this family of organisms became known as the salmonellas. By this time their pathogenic potential was well recognized, and they were known also to be transmitted through the accidental ingestion of infected human and animal feces (Savage 1920).

Almost in parallel to this realization of disease transmission through foodstuffs and animal feces, came the recognition that diseases could be transmitted through insect vectors. The possibility that disease might be conveyed from animals to man in insect bites was one of the most important of the connections made in the years around 1900. It came about through the attempt to solve the conundrum of the origin of bubonic plague.<sup>1</sup>

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<sup>1</sup>Researchers since 1900 have uncovered a range of means by which various diseases may be transferred from animals broadly defined to man. Besides ingestion and animal and insect bites, these include the handling of diseased animals and their products, direct or indirect exposure to infected animal urine and/or feces through breaks in the skin or through the mucous membranes, passage through intact skin, inhalation of contaminated dust from infected animals, and laboratory accidents.

Plague had broken out of China in 1894, reaching India by way of the trade routes through Hong Kong in mid-1896. The initial focus of the outbreak was in Bombay, where large numbers of rats were noted to be dying that summer. But whereas several Indian hill tribes traditionally quitted their homes immediately on seeing a dead rat, in urban Bombay no attention was initially paid to the phenomenon of dead and dying rodents (Creighton 1905). During the outbreak in Hong Kong, however, the French microbiologist Alexandre Yersin had isolated the plague bacillus, and in October 1896 the Bombay epidemic was bacteriologically confirmed to be bubonic plague. By 1898 all the great provinces of India were affected, and by 1903 the human death toll was running at a little under a million a year. An estimated 12 million people lost their lives in that plague epidemic (Greenwood 1935).

From the time that plague emerged in Hong Kong, it aroused excited interest in medical communities. Opinions on its causation were sharply divergent, ranging from the miasmatic ("soil-bred") through the ingestion of rotten grain, to contagion from rats (Thomson and Thomson 1901). In 1871, Emile Rocher had noted the death of rats in association with the outbreak of plague. Travelling through the Yunnan province of China, Rocher noted the presence of endemic plague, and that rats were invariably the first victims (Cantlie 1896; Rocher 1879). In the early 1890s, another Frenchman, Pierre-Louis Simond, began studying plague at firsthand in China and later in India. In 1898, he published his contention that plague was transmitted to man from rats through the bite of a bug or flea (Simond 1898). The idea received a very mixed reception. When E. Calmette declared that Simond had proved that fleas were the chief agent of plague infection, the distinguished parasitologist George Nuttall riposted that he had proved nothing of the kind (Thomson and Thomson 1901). Patrick Manson, however, developing his argument from the examples of several tropical diseases, was convinced that rats played a crucial role. Further, Manson flagged the importance of animals in the transmission of disease to man:

that the lower animals, especially . . . those that are intimately associated with man, play an important part in the transmission of human disease is now only becoming to be appreciated . . . for once in a way, science is vastly in advance of practice. Our sanitarians and the public do not fully recognise all that the community of interest, as regards disease germs, of man and beast means in the spread of disease. At all events if they do understand it they certainly do not act as if they appreciated it. (Manson 1899, p. 924)

Nonetheless, the hypothesis that a biting insect was the vector of plague was contemptuously dismissed by the first official investigation into the Indian epidemic (Greenwood 1935). Not until 1905 did the India Office get a research team into the field. In three years of painstaking work, the commission collected conclusive evidence of the transmission of plague from rats to man by flea-bites (Wilkinson 1992). A year later, in 1909, Charles Nicolle of the Pasteur Institute



in Tunis demonstrated that the human body louse was the vector of typhus in man, and the evidence for the transmission of bacterial diseases from insects to humans, and from animals via insects to humans, was finally fully established.

### ECOLOGIES OF DISEASE TRANSFER

The first decade of the 20th century, therefore, saw the confirmation of major pathways of disease transmission from animals to humans. Setting aside the great tropical diseases—sleeping sickness, yellow fever, Chagas' disease—research conducted in the years after the First World War added numbers of other animal-associated diseases—including brucellosis, tularemia, Q fever, and leptospirosis—to those already known to be endemic in the West. Many of these diseases produced different clinical manifestation in humans and animals, which rendered their relationship opaque until microbiologists identified the causal organisms. Describing endemic brucellosis in villages of the Ardeche region, for example, one French observer reported on “the no end of houses sheltering goats which abort repeatedly on the ground floor, whilst above the owners of the animals, who have been bed-ridden for weeks, are in the grip of undulant fever” (Dalrymple-Champneys 1960, p. 44). To the casual eye, however, there was no apparent connection between the two problems. As the number of diseases associated with animals accumulated, so too did evidence of the ecological complexity of these diseases, with hosts, vectors, and varied environments variously interacting. The causal organism of tularemia, for instance, has been isolated from more than 100 species of mammals, nine species of domestic animals, 25 types of bird, and 70 types of insect. In the United States, the principal vector is a tick; in Sweden, two different species of mosquito. The disease also has half a dozen pathways of transmission to humans, and manifests itself in seven different clinical forms as well as subclinically. One survey undertaken in Sweden in the 1980s showed that nearly a quarter of the population had been infected with tularemia, and of those more than a third were subclinical cases (Kiple 1993).

The concept of animal reservoirs, established through the connection of rats and their fleas with bubonic plague in man, was being widely used by epidemiologists by the 1920s. In their pioneering studies of the salmonellas, published in the middle of that decade, William Savage and Bruce White (1925) suggested wild rodent populations as a harbor for these organisms. Domestic ducks were soon implicated also (Scott 1930). In 1928, the English epidemiologist Clifford Gill, whose principal interest was in the generation of epidemic waves, selected three diseases for scrutiny: malaria, plague, and influenza. Picking up on a recent Chinese study, he noted that marmots had “since time immemorial” been associated with plague in Mongolia and Manchuria, and that rats also provide an endemic focus (Gill 1928, p. 367). Drawing again on the technique of historical epidemiology, he reported that all authorities agreed that influenza epidemics originated in the “silent spaces” of Asia, Siberia, and Western China, and that

American epidemiologists had recently claimed that Canada and the United States had long provided an endemic focus for the disease. Noting the tradition of equine influenza, Gill found the existing evidence for an animal reservoir of the disease inconclusive, although he sagely hedged his bets by concluding that "it is inexpedient to exclude from consideration the possible occurrence of a reservoir of infection apart from man" (p. 225).

Gill was unaware of work then being undertaken in America that would reinforce the suspicion that animal reservoirs had a part to play in the generation of flu pandemics. Indeed, in the 20th century the focus of research in comparative medicine shifted away from Europe to the United States (Wilkinson 1992). Here too, connections between disease in man and animals continued to emerge, as the public health services and the Bureau of Animal Industry grappled with human and animal diseases newly observed in the wake of man's ever-expanding interventions in the country's natural habitats. In the autumn of 1919, J. S. Koen, a veterinarian working for the Bureau of Animal Industry in Iowa, observed a new disease of swine with symptoms similar to those of the disastrous human epidemic of the previous year. Further study showed that although the disease had disappeared in humans, it reappeared every autumn in pigs in Iowa. Veterinarians at the Bureau worked on the problem without much success, until in 1930 it attracted the attention of Richard Shope, then working at the Rockefeller Institute of Comparative Pathology at Princeton. Described as an "extrovert and unconventional thinker receptive to novel ideas," Shope succeeded in 1931 in transmitting the disease from pig to pig with filtrates obtained from the respiratory tract of sick animals (Beveridge 1977, p. 5). This started a train of research in Britain and elsewhere that within a few years showed the ferret and the mouse to be susceptible to human influenza. By the 1950s, this research had generated a substantial scientific understanding of influenza viruses. In that decade, it was finally shown that horses were indeed susceptible to the same strains of influenza as humans, and also that the organism of fowl plague belonged to the influenza family (Easterby 1980). Not only did influenza demonstrate that airborne viral infections could indeed be common to animals and man, but it was also showing signs of being another infection with multiple animal reservoirs.

In 1972, Rob Webster and Graeme Laver reported that the hemagglutinin protein of an avian strain of influenza isolated years previously was closely related to that of the human 1968 Hong Kong pandemic. They suggested that the new flu strain might have originated by genetic recombination from a mammalian or avian influenza virus (Webster and Laver 1972). Within months, W. I. B. Beveridge, the professor of Animal Pathology at Cambridge University, noting the "mounting evidence that animal strains are involved" in the genesis of pandemics, suggested a study of the ecology of man and animals throughout Gill's "silent spaces" (Beveridge 1972, pp. 86–87). Beveridge speculated that the intimate human-animal contacts that characterized human culture in that vast area

could provide the point of origin for flu epidemics: “new born animals,” he wrote, “are tended in the same yurts where people live with their babies. In parts of China it is also common for animals and people to occupy . . . the same room. This close association between man and animals has been going on in central Asia for seven thousand years” (p. 86). He drew attention to influenza as a global epidemic beyond human control and emphasized the potential hazards brought about by modern social conditions: “Parasites flourish when their hosts become numerous and crowded. The human race has become very numerous and crowded. Modern transport and large aggregations of people have provided ideal conditions for the spread of this airborne parasite” (p. 86). As with all influenza scientists, the specter of 1918 was not far from Beveridge’s mind. Presciently and ominously, he noted that one strain of avian flu caused 100 percent mortality in chickens and warned of the possibility of a repeat of 1918. Twenty-five years later, in the winter of 1997, influenza experts in Hong Kong were looking in horror at a similarly lethal avian influenza among domestic fowls that had demonstrably caused deaths in man (Davies 1999; Laver, Bischofberger, and Webster 2000). Within five years the circulation of related avian flu strains in mainland China was again raising concern (Henderson 2002).

Influenza, like plague, has a dramatic history as a major killer of humans, and, as with plague, the suggested animal connection has been skeptically received in some quarters (Davies 1999). The elucidation of other connections has been less contentious. While influenza researchers were realizing the potentials for transfer between human and animal viruses in the years after World War II, a more mundane, but equally complex disease ecology was being unraveled in a different scientific community. Concerns over the apparent escalation in prevalence of food poisoning surfaced in Britain after 1945, although it can be argued that this increase was in many respects artificial. Notification of human salmonellosis had begun in 1939, recording a few hundred cases a year. By 1950, the number was several thousand and rising. The number of salmonella serotypes observed was also increasing, as a global trade in basic foodstuffs developed, initially under the pressures of war. Before 1939, only 14 salmonella serotypes had been observed in Britain; by 1944, the number was 23; and by 1962, around 77. Meanwhile, the development of the new technique of bacteriophage typing was enabling microbiologists at the newly established Public Health Laboratory Service to track the reverberations of salmonella outbreaks. Their investigations reinforced recognition of the numerous links between reservoirs of infection in domestic animals, especially cattle, pigs, and poultry, and human infections. Bessie Callow, who was crucially involved in developing the phage typing of *Salmonella typhimurium*, observed that the epidemiological study of these organisms was complicated by their many indigenous sources (Callow 1959).

Phage typing also opened sobering new windows on the causation and proliferation of salmonellosis in domestic animals. Animal feedstuffs and their preparation methods were shown to be important sources of infection in livestock,

and long-standing veterinary concerns about the transport, handling and examination of animals before slaughter were reinforced. Moreover, because the organism involved in individual human cases could now be precisely identified, it was discovered that contaminated foodstuffs could be the link between scattered cases within so wide a geographical area that their common cause previously never would have been suspected (Anderson 1962). By 1960, the salmonellas were known to have one of the widest host ranges of any known disease organism—testimony, one English Ministry of Health official noted, that “the cumulative effect of the zoonoses can but have a very appreciable adverse influence on general public health” (Twohig 1960, p. 131).

In 1936, statistical analysis revealed the epidemiological context for Hanna Belin’s tragedy. The distribution and incidence of typhoid in France was directly related to shellfish consumption (Dubreuil 1936). Phage typing greatly extended understanding of the distribution of salmonellas in general and their relationship with the natural world and human activity. In the last decades of the century, these understandings were furthered again through molecular biology. Phage typing was largely replaced by techniques that involve restriction enzyme analysis of DNA obtained from various potentially contagious organisms. The scientific understanding of the relationships between animals, human diseases, and their vectors has been far from static.

### **CHANGING RELATIONSHIPS**

In the years after 1945, the growing sophistication of research technologies for the detection and analysis of disease-causing organisms brought an increasing realization of the extent and complexities of the interconnections between animal and human disease. The broader implications of these connections attracted little wider attention, even though they cumulatively raised far-reaching questions about man’s relationship with and treatment of, not only animals, but the whole natural world. Some 200 years ago, in 1796, Edward Jenner elaborated the connection between man, his relations with animals, and disease:

The deviation of man from the state in which he was placed by Nature seems to have proven to him a prolific source of disease. From the love of splendour, from the indulgence of luxury, and from his fondness for amusement, he has familiarised himself with a great number of animals, which may not originally have been intended for his associates. (Hull 1930)

That recognition of the disease potential in human distortion of the relationships originally established in nature has been variously recognized by later observers. George Fleming offered a variation in his sustained advocacy of comparative medicine. In 1871, for example, he wrote that the study of animal plagues affords an introduction to

subjects of the mightiest importance in the physical and organic worlds; and the wonderful relationship which exists between life and the elements surrounding it—the reciprocal influence of these, and the connection between cause and effect—are the most interesting and engrossing of any subject the human intellect can grasp for examination. (Fleming 1871, p. xxxi)

Yet Fleming's vision met with little support. Throughout his career, Fleming was discouraged by the failure of comparative pathology to make headway in Britain. "It has not," he noted, "been looked upon with favor by the medical profession." By contrast with Europe, British governments remained impervious to its potential benefits (Fleming 1871, pp. v–vi). In 1881 he was still arguing for the inclusion of comparative pathology in the English medical curriculum:

surely a knowledge of animal diseases in their relation to those of our own species, is of far more moment to the surgeon or the physician in the practice of his profession, than an acquaintance with zoology or comparative anatomy. . . . Why are we so unwilling to adopt what is manifestly and so urgently required in order to complete the [medical] student's education and to render him a more useful and enlightened member of society? (Fleming 1881, p. 1)

For many and various reasons, comparative medicine remained outside the mainstream of British medicine, except insofar as animal models and animal experiments had established themselves as a central research tool of modernizing medical science (Bynum 1990). The search for animal models of human diseases was to be particularly important in the 1960s and 1970s, in the heyday of comparative medicine. Yet a sub-current of concern with the pathways of disease transmission between human and animals nonetheless continued through the 20th century. A trickle of publications concerned with man and his disease environment reflected this continuing interest. Relevant volumes include T. G. Hull's *Diseases Transmitted from Animals to Man* (1930), Joseph Bigger's *Man against Microbe* (1939), which contained chapters entitled "The Menace of Animals" and "Winged Death," and Richard Fiennes's *Man, Nature and Disease* (1964). Fiennes, himself a veterinary surgeon, believed that the study of disease in nature offered the key to the fundamental characteristics of disease in man. In his view, the subject had been subordinated to the overriding need to master the various acute and chronic human infections. Fiennes was concerned by what he saw as the "disequilibrium" that had grown up between human and animal communities, and by changes in natural habits. Infectious diseases, he argued, were derived from these habitats, and they might prove a source of other diseases (Fiennes 1964).

Fiennes was not entirely a voice crying in the wilderness: the World Health Organization and the Food and Agriculture Organization had set up a joint expert committee on the zoonoses in the 1950s. And in 1960, an official of the Ministry of Health, recognizing that "medical men for long overlooked the significance of animal disease as a factor in human health," called on "the general practitioner, the

public health official, the epidemiologist, the veterinarian, the laboratory worker and the hospital physician . . . by integration of effort and co-ordination of work help to ensure that these diseases do not add further to the burden of mankind” (Twohig 1960, p. 131). Among the many pressures on modern medicine, increasing exponentially after 1945, this was a pious hope. W. I. B. Beveridge summed up the position definitively in 1972: “Ever since the great work of Charles Darwin, man has been seen to be but one of the animal species, yet the major stream of scientific thinking has continued to proceed on the basis of the old premise of a fundamental division between the human and animal worlds” (Beveridge 1972, p. 19).

### AN ENDURING DIVIDE

Science has not been alone in observing this “fundamental division.” The naturalist Gerald Durrell (1963) once noted his surprise at the number of people in different parts of the world who seem oblivious to the animal life around them. With some honorable exceptions, historians of medicine and science have also focused on human medicine, rather than studying the history of comparative medicine, or animal medicine, or disease relationships within the natural world. *The Cambridge World History of Human Disease*, one of the most monumental history of medicine compilations of recent years, describes 158 “major human diseases,” more than a quarter of which are associated with animal reservoirs or insect vectors (Kiple 1993). Yet of the 59 analytical chapters which make up the rest of the book—and which include such subjects as disease and migration, famine and disease, and occupational disease—none is devoted to animals and disease. Neither historians nor scientists are immune to the enduring power of the anthropocentric perspective on disease.

Nonetheless, a skim through the *World History* with an eye to animal-related infections makes uneasy reading. As you pass, among others, from anthrax and the arboviruses to brucellosis and bubonic plague, to Chagas disease and dengue, to giardiasis, influenza, Japanese B encephalitis, Lassa fever, Lyme disease, malaria, Q fever, rabies, toxoplasmosis, tuberculosis, and yellow fever, the extensive spectrum of animal-related diseases, their multiple pathways of transmission, complex ecologies, and the delicacy of the many of the ecological balances that regulate their incidence, generate awareness of the potential of these diseases to affect human populations: notwithstanding his prepotence in the modern world that he has made his own, man remains one small biological entity in a vast natural fabric that was woven to a design beyond his specific needs and desires.

In the 1960s, the prospects seemed good for the control, even the eradication, of infectious disease. By the 1970s, the emergence of new diseases was beginning to underscore the significance of the disease hazards still awaiting man in the animal kingdom (Garrett 1994). The appearance of Marburg virus in 1967, of Lassa fever in 1969, and Ebola in 1976, for example, focused attention on human activity in Africa as a potential threat. While Lassa has been shown to have a

reservoir in an African rat, the animal-man connections of many of these infections remain obscure. Horrifying as these infections are, it was a more subtle disease transfer, that of simian immune deficiency virus to man, that generated a new global human epidemic, that of HIV/AIDS, in the 1980s. Somewhat less obscure were the apparent connections between the new epidemic disease of cattle, bovine spongiform encephalopathy (BSE), which emerged in England in the 1980s, and the appearance of a previously undetected human encephalopathy: new variant Creutzfeldt-Jacob disease (nvCJD). The political reaction to the new animal disease resonated tellingly with that described by Marcel Belin for France in the 1930s. There were also parallels with the veterinary dilemma over bovine tuberculosis in the 1880s. The British government initially made every effort to reassure their citizens that British beef was safe to eat. It was only when nvCJD cases began to appear in the mid-1990s and the condition was linked to BSE, that public outcry obliged the government to take action against British cattle producers and the meat industry.

#### CONCLUSION

In choosing to focus primarily on how the pathways of disease from animals to man were discovered, I am conscious of having relegated other significant aspects of the relationship between human and animal disease to sub-plots. My emphasis has been on continuities in this story, but there has also been change. In the past 150 years, there has been a complete transformation in human knowledge of the nature and extent of diseases transmissible from animals to man. There have also been changes in the patterns and incidence of many of these diseases, which have been largely due, for good or ill, to the deliberate or unconscious interventions of man. Humans have sought to control many of these diseases, primarily through technical interventions such as immunization, pasteurization, and pesticides. They have proved less willing to accept the implications of environmental intervention, or to control human activities that increase the potential for disease transfers.

If I can touch once more on the tragedy of Hanna Belin, the history of the realization of the connections between disease in animals and in man demonstrates that the acquisition of knowledge does not of itself lead to joined-up thinking, or prevent unwitting acts of self-destruction.

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