Lyme Disease: The Social Construction of a New Disease and Its Social Consequences

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BECAUSE DISEASES OFTEN EVOKE AND REFLECT collective responses, their study can provide an understanding of the values and attitudes of the society in which they occur. The term "social construction of disease" has come to represent a mode of historical analysis in which nonbiological factors—beliefs, economic relationships, societal institutions—are seen as greatly influencing, if not defining, our understanding of particular ills. Historians of science and medicine most often choose to study diseases that elicit strong responses because of stigma attached either to the affected population or to the mode of disease transmission, as with syphilis or AIDS; a controversial somatic basis (which invites debate over personal responsibility), as in the case of alcoholism or psychiatric diagnoses like anorexia nervosa; or fear of acquiring a deadly disease, for example, during epidemics of a disease like cholera.

Diseases that are not deeply stigmatized, that have unassailable biological foundations, and that are not deadly are less frequently studied using the social constructionist approach, but they are no less resonant with social meaning. Lyme disease is one such case. It is a contemporary, somatic, treatable ailment that is typically contracted during wholesome outdoor activity. The history of Lyme disease exemplifies

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how social factors interact with biological ones in virtually every aspect of a disease's discovery and progress: its etiological investigation, epidemiology, clinical presentation, diagnosis, treatment, and prognosis.

Some observers have called the investigation of Lyme disease a biomedical success story. American researchers recognized a cluster of cases of arthritis in and around Lyme, Connecticut in the mid-1970s, discovered that the vector was the deer tick *Ixodes dammini*, and subsequently identified the pathogenic agent as a spirochete, which we now call *Borrelia burgdorferi*. Yet these accomplishments cannot be understood without some insight into the historical context—both within and outside the clinic and laboratory—in which this new understanding emerged.

In order to emphasize the interaction of biological and social factors in the early biomedical investigation, I focus on the construction of Lyme disease as a new disease in the first section of this article. Early on in their work, researchers recognized that it was closely related to a disease known since the beginning of the century—erythema chronicum migrans (ECM). I examine what was known about ECM and related conditions in the pre-Lyme-disease era and how investigators understood and presented the relation of Lyme disease to ECM from among the available options.

In the second section, I focus on the social consequences of Lyme disease, emphasizing how the general meaning and significance of Lyme disease has been contested by biomedical scientists, clinicians, patients, advocacy groups, and the media. Five features merit particular attention: public and professional responses to the new disease; problems of diagnostic testing; the social negotiation of the definition of, and treatment for, chronic Lyme disease; the commercialization of Lyme disease; and the nature of Lyme disease as a public-health problem. Although biomedical knowledge has conditioned the progress of this debate, it has by no means resolved the conflicts among different parties.

I argue that the construction of a categorically new disease was built implicitly and incrementally from a number of interacting factors, not as a self-evident reflection of the biological and epidemiological facts. These factors include the nationality of investigators (Americans vs. Europeans), disciplinary background (rheumatology vs. dermatology), methodological approach (prospective studies vs. case reports), inter-

pretation of biological evidence (possible differences between European and American spirochetes and ticks), intellectual or attitudinal features (skepticism toward research in past generations), ecological relations (divergent interactions among vectors, hosts, and demographic changes), and professional concerns (potential self-interest in promoting a new disease). I believe not that it was wrong to have conceptualized Lyme disease as a new disease, but rather that other conceptions were possible and plausible. Although this approach necessarily involves some selective hindsight, my goal is to demonstrate the range of potential scientific and social responses that could have appropriately been brought to bear on biological processes, not to prescribe the correct or necessary one. This demonstration might be criticized for merely illustrating a tautology—disease, a culturally defined concept, is socially constructed (Rosenberg 1989). However, the details, not the mere existence, of Lyme disease's social construction, provide the most useful insight into contemporary biomedical and lay practices and ideas about disease.

I have chosen to emphasize nonbiological explanations for historical and geographic differences in the identification and definition of Lyme disease and ECM. Biological factors may play an important role in these differences, but they are as yet poorly understood. The choice to build a plausible case for more readily observable social factors runs counter to the usual assumptions of biomedical investigators, who, for example, tend to attribute clinical differences in European and American Lyme disease to differences in ticks or spirochetes. It also conflicts with the approach of some historians who have assumed that biological factors must have played a major role in the emergence of new diseases, because astute clinical observers would have noticed important clinical features had they been present previously (e.g., English 1989).

Even if biological differences in ticks or spirochetes are linked in the future to the way the different manifestations of *B. burgdorferi* infection have been perceived, a social-constructionist approach would still be valid. Such an approach would allow a more subtle analysis of how the meaning of disease changes as biological constraints change. To use Charles Rosenberg's "frame" metaphor for analyzing the history of disease, we might make better sense of the "interactive negotiation over time, this framing of pathophysiologic reality in which the tools of the framer and the picture to be framed may well have both been changing" (1989, 7).

The Emergence of a New Disease

A good number of review articles have celebrated the recognition of Lyme disease and the rapid elucidation of its epidemiology, etiology, and appropriate therapy. According to one editorial:

> There is something very satisfying about the progress that has been made since the summer of 1975, when the Lyme mothers recognized a pattern of disease in their town's children. The triumph belongs to the inquisitiveness and determination of clinical and laboratory investigators in medicine. The efforts of unfettered investigators, who had time to plan careful epidemiological and etiological studies, and a spirit of collaboration among scientists of many disciplines have led to the discovery of the probable cause and cure of Lyme disease. (Harris 1983, 774–75)

Other reviewers cite Lyme disease as a classic example of how effective and rational therapy follows from good basic science; for example, "knowledge of the trigger in this case has led to rational treatment—early antibiotics shorten the disease process" (Bacon and Tunn 1986, 898).

These accounts of Lyme disease's history, however, obscure a more complex reality. To say that the discovery of the Lyme spirochete led to rational treatment, for example, is to put the cart before the horse; the suspicion of a bacterial/spirochetal etiology followed from the responsiveness of ECM and early Lyme cases to antibiotics. This account owes more to an idealization of the relationship between basic science and therapeutics than to the actual chronology of investigation. More problematic is the fact that many aspects of the "discovery of the probable cause and cure of Lyme disease" were previously described in the ECM literature, such as the rash, the tick vector, neurological complications, the responsiveness to penicillin, and even a suspicion of the spirochetal etiology.

European Research on ECM in the pre-Lyme Disease Era

Reviewers attribute the first descriptions of ECM to the Swedish dermatologist Afzelius (1910) and to the Austrian dermato-venereologist Lipshutz (1913), who described an expanding, ring-shaped rash that

developed at the site of a tick bite (*Ixodes ricinus*). Thyresson (1990) notes that Balban (1910) described a rash similar to ECM, but did not report antecedent tick bite. Dermatologists' interest in ECM, and perhaps as a consequence, its diagnosis, was largely confined to northern Europe. Diagnosis was based on the characteristic rash. The incidence and prevalence of ECM were not carefully studied, but individual dermatologists reported having seen as many as 45 cases in private practice, indicating that ECM was not an uncommon condition (Sonck 1965).

European analysts offering clinical descriptions of the rash pointed out its migrating and recurrent features. Hellerstrom (1930) reported a case of ECM complicated by meningitis. Other systemic manifestations of ECM included nausea, lymph-node involvement, itching, and pain (Hellerstrom 1951; Hollstrom 1951). No mention was specifically made of problems with joints, a finding later associated with Lyme disease. Pain when present was attributed either to nerve involvement or to the rash itself. ECM, even when complicated by meningitis, was considered to be self-limited.

Etiological speculation focused on an infectious agent carried by ticks, although allergy was also a recurrent theme (Dalsgaard-Nielsen and Kierkegaard 1948). General support for the infectious etiology of ECM came from the systemic nature of the disease and its transmission by tick bite. Binder, Doepfmer, and Hornstein (1955) provided dramatic evidence for an infectious agent by injecting portions of ECM rash into volunteers, who developed ECM within three weeks. In 1957, Sonck (1965) injected himself with a patient's rash and demonstrated his own subsequent ECM to an international dermatological conference.

The clinical response to antibiotics also prompted scientists to focus attention on bacteria as causal agents, although the tick-vector and neurological symptoms suggested a viral etiology. In the decade before Lyme disease was identified as such in the United States, French investigators tried to demonstrate that ECM resulted from rickettsial infection (Degos, Touraine, and Arouete 1962), a bacterialike organism that causes Rocky Mountain spotted fever, another tickborne infection resulting in neurological symptoms. Among midcentury dermatologists, the prominent hypothesis was that a spirochete was responsible for the disease (Burgdorfer 1984, 515), a hypothesis that was plausible to practitioners of the specialty. Dermatology and "venereology" formerly were closely linked, and ECM shared many features with other spiro-

chetal diseases, among them rash and neurological symptoms (similar to syphilis) and transmission by insect bite (similar to relapsing fever).

Most observers attribute the spirochetal hypothesis to Lennhoff, who published his findings from memory after World War II, having lost his laboratory records fleeing Norway (1948). Lennhoff claimed to have identified spirochetes in ECM lesions and in 20 other disorders as early as 1930. He later collaborated with the Scandinavian dermatologists Hollstrom and Hellerstrom and identified spirochetal bodies in their ECM cases (Hollstrom 1951). With the recognition of spirochetes and description of neurological symptoms, antisyphilitic drugs were tried in cases of ECM. Lennhoff (1948) reported two responses to one of these drugs, Bismuth. When penicillin arrived in Northern Europe after World War II, Hollstrom (1951, 242) demonstrated its greater efficacy over other spirocheticides, arguing that this made it probable "that a spirochete is the infective agent."

European ECM investigators, nevertheless, did not convincingly demonstrate the spirochetal etiology for ECM. Thyresson (1990) states that Lennhoff's spirochetes were later proven to be artifacts. Despite the implausibility of Lennhoff's larger claims and the absence of studies that replicated his results, frequent citation of spirochetal findings in ECM in dermatology texts extended into the period when Lyme disease was initially investigated (e.g., Domonkos 1971, 511). Burgdorfer, who eventually discovered the spirochete bearing his name, attributed the failure to demonstrate the spirochetal etiology of ECM to a lack of interest:

> Thus, by 1955, clinical and epidemiological evidence was fully provided that ECM is caused by a penicillin-susceptible bacterial agent transmitted by the ixodid tick, I. ricinus. Unfortunately, no one was interested in looking for spirochetes, and the puzzle about the etiology of ECM remained unsolved. (1987, 8)

Although the spirochetal hypothesis was one of many, with the failure to prove it stemming from more complex reasons than lack of interest, Burgdorfer's assessment does correctly emphasize that ECM was understood by investigators to be a systemic condition and that some etiological and clinical investigations were remarkably prescient. Other data on ECM articulated in a summary review in 1951 correspond to our present view of Lyme disease: in many cases the tick bite is not recalled; it is not responsive to sulphonamides; and, even when accom-

panied by neurological symptoms, it is often a mild disease that usually resolves spontaneously (Hellerstrom 1951). In the decade preceding the description of Lyme disease, only a few North American dermatology texts even mentioned ECM, presenting it as an infectious process responsive to antibiotics and giving greater credence to the more recently articulated rickettsial hypothesis (Domonkos 1971; Moschella, Pillsbury, and Hurley 1975; Rook, Wilkinson, and Ebling 1972).

In order to understand the knowledge potentially available to early Lyme researchers in the United States, it is necessary to consider two other entities that we now recognize as manifestations of the same infectious process as Lyme disease and that were linked, in different degrees, to ECM. One such syndrome was acrodermatitis chronica atrophicans (ACA), a chronic skin condition first described in the late nineteenth century. Before recent investigations established the common etiology of ACA and ECM, some investigators suspected that they were both infectious diseases caused by the same organism (N. Thyresson, personal communication, September 1990). ACA had been reported to follow ECM in a few case reports (Åsbrink, Hovmark, and Olsson 1986). Reviewers have noted that the history of ACA paralleled ECM in a number of ways: demonstration of its infectious etiology by injecting bits of lesion into human volunteers; its response to antibiotics; suspicion by some of a tick-vector and by others of a spirochetal etiology (Lavoie, Wilson, and Tuffanelli 1986). ACA was nevertheless not conclusively linked to ECM until after the elucidation of Lyme disease.

ECM was more definitively linked in the pre-Lyme-disease era to a second tickborne neurological disease, variously called lymphocytic meningoradiculitis, Bannwarth's syndrome, and other names (Bannwarth 1941; Garin and Bujadoux 1922). Interest was much greater in Europe than in the United States, as reflected by its virtual absence from American neurology texts, whereas European texts devoted whole chapters to the syndrome (e.g., Meyer-Reinecker and Hitzchke 1978). At the time Lyme disease was first reported, one textbook noted that "the anamnesis often mentions an insect prick (arthropoda), especially a tick bite (*Ixodes ricinus*), which is followed by erythema migrans" (Meyer-Reinecker and Hitzchke 1978, 573). Despite the connection to ECM, the disease was generally held to be caused by a virus and thus not responsive to antibiotics. In many cases, the disease was mild and resolved spontaneously.

ECM and Related Disease Manifestations in the United States: Tracking a New Phenomenon

The puzzle about the etiology of ECM and related conditions persisted while cases started to be reported in the United States. The first American case was a report of a Wisconsin physician who developed a chronic rash on his right torso, which was accompanied by low-grade fever, headache, malaise, and hip pain (Scrimenti 1970). He gave a history of a tick bite at the site of the rash three months prior to presentation. After two days of taking antibiotics, the rash and symptoms cleared. In 1975, another case of ECM with systemic symptoms was found, but infection was attributed to a recent trip to Northern Europe (Wagner et al. 1976).

The first American case cluster was identified by dermatologists working at the Naval Submarine Medical Center in Groton, Connecticut during the summer of 1975 (Mast and Burrows 1976a). The authors concluded that the "occurrence of multiple cases of erythema chronicum migrans within a limited geographical area within a one-month period lends further support to the concept of an infectious and insect-borne etiology" (Mast and Burrows 1976a, 860). With hindsight, Mast and Burrows's report, "Erythema Chronicum Migrans in the United States," described an earlier sample of the same disease process as Lyme cases, which were to be studied in November 1975. One can speculate that this report failed to capture much interest because the authors believed they were observing a known, obscure disease and because they did not approach the case cluster as an epidemiological problem.

The first cases of what would eventually be called Lyme disease attracted medical attention because of the actions of two women from the area. Polly Murray had been sick since the 1960s with intermittent symptoms such as rashes, swollen knees, stiff joints, and sore throat. By her own account, she had consulted over 24 doctors without getting adequate explanation or relief (Lang 1989). Alarmed by a similar illness in her sons and neighbors, Murray called a state public-health official in the fall of 1975 and was referred to Allen Steere, a Yale rheumatologist-in-training. Steere was known to the state official because he had previously been an epidemic intelligence officer for the Centers for Disease Control (CDC). During this same period, Judith Mensch contacted state health authorities and the CDC seeking an explanation for why

her daughter and other children in the area were being diagnosed as having juvenile rheumatoid arthritis, a rare and sporadic affliction. She also was referred to Steere.

Steere, other Yale workers, and the Connecticut public-health authorities then identified children exhibiting inflammatory joint disease and a few similarly affected adults from the area in and around Lyme by surveying local parents, physicians, and school nurses (Steere, Snydman et al. 1986). Although it was difficult to differentiate these cases from juvenile rheumatoid arthritis on clinical grounds, the high prevalence and the geographic, temporal, and familial clustering pointed to an infectious disease. The preliminary findings were reported at a national arthritis meeting (*JAMA* 1976).

About a quarter of these initial Lyme subjects gave a history of a rash. The Yale investigators did not see the rash in their original group of 51 cases, probably because their rheumatological case definition captured only late cases and their initial work took place after the tick-bite "high season" in summer (it is nevertheless surprising, given the sometimes prolonged and recurrent nature of the rash of ECM, that none of these cases had an observable rash). Steere (personal communication, September 1989) discussed the Lyme subjects' description of the rash with a Yale dermatologist who had attended a conference the previous summer in which the Groton cases were diagnosed as ECM, partly with the aid of a visiting Danish doctor. Steere was struck by the similarities between ECM and the rash described by Lyme cases and by the spatial proximity of the Lyme and Groton cases. The connection between the Lyme cluster and ECM was thus strongly suspected before the rash was ever directly observed by Yale investigators.

In the summer of 1976, Yale investigators were able to study cases prospectively and confirmed the rash's identity with the ECM rash. The following year, a patient with an ECM rash presented with the tick that bit him, reconfirming the connection between ECM and tick bite. Because of an explosion of ticks in the Lyme area over the previous decade, Yale entomologists started a tick survey (A.C. Steere, personal communication, September 1989). They found a dramatically greater abundance of what was initially identified as *Ixodes scapularis* in the area of the cases, compared with a nonendemic area (Wallis et al. 1978). Entomological investigations of babeosis, a malarialike disease, led to a reclassification of *Ixodes scapularis* into two new species. The vector of both babeosis and Lyme disease in the Northeast was named

Ixodes dammini after Gustave Dammin, Harvard pathologist (Spielman, Clifford et al. 1979). Further epidemiological investigations associated Lyme-disease cases with *I. dammini* in different areas of the United States (Steere and Malawista 1979). A related tick, *I. pacificus*, appeared to be the vector of Lyme disease in Oregon and California (Naversen and Gardner 1978).

The identification of the Lyme spirochete was made by Willy Burgdorfer and collaborators (Burgdorfer et al. 1982), who were working on a tick/rickettsia survey in eastern Long Island. They were studying the ecology of *Rickettsia rickettsii*, the etiological agent of Rocky Mountain spotted fever. Because of the failure to find dog ticks that harbored *R. rickettsia*, the Burgdorfer group tested other ticks, including *I. dammini*. Burgdorfer noticed what he thought were microfilaria in the hemolymph of two *I. dammini* ticks and decided to dissect their digestive tracts looking for earlier developmental stages. No evidence for these worms were found, but he did discover spirochetes.

Later, spirochetes were found in other *I. dammini* ticks, the West Coast ticks implicated in Lyme disease, and the European ticks, which were the putative vector of ECM. Antibodies to this new spirochete were found to cross-react with serum of Lyme-disease patients, and later the spirochete itself was directly identified in patients with Lyme disease (Benach et al. 1983; Steere, Grodzicki et al. 1983). It is noteworthy, however, that the ultimately successful approach to elucidating the cause of ECM had been anticipated many times in ECM's history. Lipshutz in 1923, for example, cited the need to study the saliva and intestinal tract of the tick vector of ECM (Burgdorfer 1987). Later on there were calls for other potentially rewarding approaches, such as microscopic examination of skin, spinal fluid, and lymph nodes in order to isolate the hypothesized pathogen (Hellerstrom 1951).

The long interval between these suggestions and Burgdorfer's identification of the Lyme spirochete makes apparent the technical intricacies of identifying and culturing the spirochete responsible for ECM/Lyme disease (see Barbour 1987), as well as the lack of concerted effort to find its cause in the pre-Lyme-disease era. The spirochete's location in the tick's midgut, rather than the salivary glands, where other arthropod-born infectious agents are usually isolated, probably contributed as well to the difficulty in identifying it (A.C. Steere, personal communication, September 1989). Finally, Burgdorfer's identification of the spirochete, which he described as an "encounter [with] . . . poorly stained, rather

long, irregularly coiled spirochetes," was a complex "discovery" that had its genesis not only in Burgdorfer's long experience dissecting ticks and looking for microorganisms inside them, but also in his knowledge of the European literature on ECM. Reflecting on his "chance" discovery, Burgdorfer (1987) recounted: "The microfilaria led me to the discovery of the long-sought cause of ECM and Lyme disease."

Lyme Disease and Its Relationship to ECM

In their first report, the Yale investigators named the disease they observed "Lyme arthritis," stating that it was a "previously unrecognized clinical entity." They noted the similarities of their subjects' description of an expanding rash to ECM, and briefly reviewed what was known about ECM. Yale investigators believed so strongly that arthritis was the defining feature of the disease that they cited the Groton case report as part of the ECM literature, not as a different sample of the same disease they were studying (Steere, Malawista, Snydman et al. 1977).

After prospective studies confirmed the relationship between ECM and arthritis, Yale investigators acknowledged that "ECM and the subsequent neurological abnormalities are manifestations of the same illness" (Steere, Malawista, Hardin et al. 1977, 695). With this awareness and the subsequent discovery that skin, joint, and other manifestations were all related to *B. burgdorferi* infection, the disease might have been duly renamed, the label either fashioned from the cause (e.g., *B. burgdorferi* disease or *Lyme borreliosis* as it is sometimes called), or from ECM, whose prior investigators had in good measure described or predicted what was now being systematically confirmed. Instead, "Lyme arthritis" gave rise to "Lyme disease" as an enduring label, fixing the salience of the Yale investigators' contribution and emphasizing the newness of the disease. Thus, a typical publication on Lyme disease begins: "Lyme disease, first described in 1976, typically begins in summer with a characteristic skin lesion, erythema chronicum migrans, often accompanied by headache, stiff neck, fever, malaise, and fatigue" (Steere, Malawista, Newman et al. 1980, 1).

Lyme disease is not only presented as new, but its relation to ECM is also specifically limited in scope; ECM is now the name of the ailment's characteristic rash. Such usage obscures the fact that ECM was used to describe an infectious dermatological condition with systemic

features. European cases of *B. burgdorferi* infection also are subsumed under Lyme disease, as another report begins: "Lyme disease, first recognized in Lyme, CT. in 1975, is now known to occur in at least 14 states, in Europe, and in Australia" (Craft, Grodzicki, and Steere 1984, 789). Such usage implies that recognition of this disease in Europe came after the investigation of the Yale cluster—an accurate observation only if one assumes an abrupt discontinuity between knowledge of ECM and Lyme disease. Some Europeans continue to refer to "erythema migrans disease" or, more clumsily, "the infectious disease caused by *B. burgdorferi*." These investigators imply that the major accomplishment of the Lyme investigators was to bring modern epidemiological techniques, like prospective studies, to bear on a previously known clinical entity, confirming features that were suspected but never proven and creating a more complex and accurate clinical picture (Weber 1986).

Lyme Disease's Construction as a New Rheumatological Entity

The factors leading to the construction of Lyme disease as new can be grouped into three categories: those related to the rheumatological identity of the disease, such as the patterns of symptom recognition and physician referral and the structure of epidemiological investigations of new outbreaks of disease; those related to the conceptual schema and social position of investigators—their attitudes about what constitutes a new disease, assumptions about the priority of biological explanations for the appearance of new clinical features, and potential self-interest in articulating a new disease; and those that created the right conditions for the Lyme outbreak, such as the particular ecological and demographic changes that preceded it. I will consider each of these categories in turn, arguing that the perception of a new disease resulted from a complex and implicit weighting of these factors.

The construction of Lyme disease as a new ailment was justified, first, by its striking rheumatological character. "Lyme arthritis" was a new disease because the Lyme cases did not resemble any preexisting arthritic condition (Steere, Malawista, Snydman et al. 1977). The recognition of ECM preceding the condition added to its uniqueness. At the same time, arthritis was what made the Lyme cluster novel in the context of the prior history of ECM and related conditions.

Although researchers presented Lyme disease's rheumatological identity as a self-evident objective fact, it can more profitably be viewed as having been constructed from interacting biological and social factors. Given our current knowledge of Lyme disease's epidemiology, it is probable that children formed the initial case cluster because of social factors such as the greater attention given to sick children rather than because of an increased incidence among them. Once children were the object of medical attention, arthritis would be tagged as a more noticeable medical symptom, in contrast to adults in whom inflammatory arthritis is much less unusual and can be attributed to many different disorders.

The patterns of symptom recognition and patient referral that led concerned Lyme residents to Allen Steere played an important part in Lyme disease's rheumatological identity, as evidenced by the fact that patients who sought medical care from the Groton dermatologists were diagnosed as suffering from ECM. As if to emphasize from the outset the importance of these factors in defining this disease, a medical news report on the Yale investigation of the Lyme epidemic appeared a few weeks earlier in the same journal in which the Groton cases were published (*JAMA* 1976), but neither article mentions the other. Only later did the authors of the Groton report publish a letter stating that they had exchanged information with the Yale investigators and agreed that they were observing "the same process" (Mast and Burrows, 1976b).

The Yale rheumatologists' decision to make arthritis prominent in the case definition they used to collect the initial pool of cases followed the common epidemiological practice of constructing a case definition that is most likely to distinguish people who have the disease from those who do not. As in many investigations of case clusters, however, one necessarily ends up with a disease that fits one's preconceived definition, akin to a Texas bull's eye: after the bullet hits the barn, the bull's eye is drawn around it.

Drawing attention to those aspects of the initial case definition contingent on nonbiological factors is not meant to dispute the fact that arthritis was common in *B. burgdorferi* infection in the Lyme area. In a well-designed prospective study, the Yale investigators later found that seven of twelve cases defined by initial ECM went on to develop arthritis (Steere, Malawista, Hardin, et al. 1977). Nevertheless, the pattern of symptom recognition in Lyme families, their referral to academic rheumatologists, the prominence of arthritis as a symptom among children, the interest and specialty outlook of the investigators,

and the necessary limitations of case-cluster investigations all served to highlight the rheumatological identity of Lyme disease.

As the links between arthritis, other systemic signs and symptoms, and ECM became increasingly clear owing to rigorous prospective studies and the discovery of *B. burgdorferi*, a conceptual rationale less focused on Lyme disease's rheumatological identity was offered to justify its status as a new disease. "Lyme disease" brought together various isolated strands—Lyme arthritis, ECM, acrodermatitis chronica atrophicans, Bannwarth's syndrome—into a single, heterogenous clinical entity. Steere (personal communication, September 1989) likened this accomplishment to the emergence of syphilis in the 19th century as a single, protean disease from a variety of "diseases" and symptom complexes. Investigators have also argued that Lyme disease is new in a strictly biological sense, that is, biological differences between European and American spirochetes probably explain the absence of arthritis in prior descriptions of ECM and related conditions (Steere, personal communication, September 1989). This assertion is by no means self-evident, however; nonbiological factors might explain why the earlier literature failed to make mention of arthritis. European investigators necessarily saw the disease in its early stages (when the rash typically occurs) and arthritis is usually absent. Joint pain is a common background complaint that may not have been linked by investigators to ECM, or even noticed. Early treatment of ECM with antibiotics, as was the practice in northern Europe from the 1940s onward, would have reduced the number of cases with late symptoms such as arthritis. The assumption of a biological basis for differences between ECM and Lyme disease, therefore, is as much due to the standard belief system of biomedical researchers, with their preference for biological explanations, as to any direct evidence. One cannot ignore the fact, too, that investigators were likely to gain attention by conceptualizing and naming the Lyme cluster as a new disease, although there is little to suggest this as a major motivating factor.

The third set of factors helped create the right conditions for the Lyme outbreak. Ecological and demographic relationships in particular areas, which are themselves mediated by social factors, are the most important, although they are not fully understood. For example, suburbanization has probably promoted Lyme disease's appearance and increasing incidence in the last two decades. When rural farmland is transformed into a suburban landscape, there is an increase in wood-

land and a seemingly paradoxical increase in the deer population and the ticks whose life cycles depend on them. Such changes may have resulted in a dramatic increase in *I. dammini*'s range from a few isolated offshore New England islands to its present widespread distribution. More detailed ecological speculation has focused on how summer resorts with high winter and low summer populations of deer might provide the best conditions for the transmission of Lyme disease and other tickborne diseases (Spielman, Wilson et al. 1985). Whatever specific ecological and demographic changes were entailed, the resultant clustering of *B. burgdorferi* infection in a localized area created the appropriate conditions for case recognition. The near simultaneous presentation of cases from the same geographical area presenting to Yale rheumatologists and Groton dermatologists in 1975 suggests that a threshold of biological and social circumstances had been reached, allowing recognition of a new biological process, although just what was new was open to negotiation.

Biomedical Consequences of Lyme Disease as a New Disease

However plausible and self-evident the emergence of Lyme disease as a new disease appeared to investigators at the time, this particular social construction had consequences, not only for the way its significance would be assessed in both lay and medical worlds, but also for subsequent etiological investigation and clinical care. The belief that the Lyme cluster represented a new disease, similar but not identical to ECM, may have contributed to the Yale investigators' initial suspicion that the etiological agent was a virus, rather than a penicillin-sensitive bacterium or spirochete, as the ECM literature strongly suggested. According to a news report that appeared prior to their first publication, "The investigators were particularly interested in testing the patients for the group A arthropod-borne viruses that cause the joint diseases Ross River (Australia), chikungunya (Africa and Asia), and o'nyong-nyong (Africa), in all of which mosquitoes are vectors" (*JAMA* 1976, 242). These potential etiological viruses, suspected because of their ability to cause epidemic arthritis, derive from the exotica of tropical disease rather than what was known about ECM. Steere (personal communication, September 1989) attributed the initial fixation on viruses,

and the downplaying of etiological speculation in the ECM literature, in part to his group's "rheumatological mind set."

Credence in the newness of Lyme disease also reinforced the Yale investigators' skepticism in the late 1970s toward another aspect of the evolved wisdom on ECM—that it was effectively treated by antibiotics. Lyme investigators were ambivalent about treating cases with antibiotics. They generally withheld antibiotics from patients during the first and third summer seasons, only routinely administering them in the second and fourth. When not treating with antibiotics, Lyme cases were frequently given antiarthritic medicines, including steroids, which are relatively contraindicated in many infectious conditions (Steere, Malawista, Newman et al. 1980).

Lyme investigators did not explicitly reject the lessons from the ECM literature about antibiotics, but the rationale for their early reluctance reveals attitudes that sustained the categorical boundary between ECM and Lyme disease. First, they noted that the prior literature on ECM and related conditions was divided on antibiotics. This statement does not correspond to the near unanimity in the ECM literature on the efficacy of antibiotics in treatment, a consensus indirectly acknowledged by the Yale investigators in their reference to 12 studies supporting their use, as against a single disconfirming study (Steere, Malawista, Hardin et al. 1977). As further evidence of the consensus in the ECM literature, the cases that presented to the Groton dermatologists and the other American case reports were all treated with antibiotics with apparent success. Rather than representing a fair assessment of the ECM literature, the Lyme investigators' rationale points to an underlying skepticism toward knowledge elucidated by workers from a different culture and medical specialty, using a less rigorous methodology in the distant past.

Second, the Lyme investigators explained their reluctance to use antibiotics by observing that "the large variation in the natural course of the disease makes it difficult to evaluate whether the observed improvement in the individual patient would have occurred anyway" (Steere, Malawista, Hardin et al. 1977, 696). This comment can be understood in part as an implicit criticism of the earlier ECM literature, which did not, among other limitations, include control groups, making the evaluation of "observed improvement" problematic. The comment also reflected the clinical experience of early Lyme patients, some of whom developed joint, neurological, and cardiac problems despite receiving

antibiotics. Later, this propensity for chronic symptoms to occur despite early antibiotic therapy would be viewed as a problematic but accepted biological attribute of Lyme disease.

Lyme researchers, third, were influenced by European investigators of Bannwarth's syndrome, who believed that the syndrome was caused by a virus and was not treatable by antibiotics (A.C. Steere, personal communication, September 1989). In this instance, Lyme investigators followed earlier researchers who, like themselves, discounted the relevance of the ECM literature to the systemic, nondermatological condition being studied. Investigators of Bannwarth's syndrome, despite knowledge of the link to ECM, similarly ignored the suspicion of a penicillin-sensitive bacterium or spirochete and the clinical response to antibiotics. The ambivalence toward antibiotic treatment in the early years of the Lyme epidemic was thus in large measure a consequence of the belief in a new disease, reinforced by a skeptical attitude toward the ECM literature.

Antibiotics were later reintroduced as part of routine therapy only after retrospective analysis of unmatched and nonrandomized consecutive cases demonstrated that the therapy appeared to help (Steere, Malawista, Newman et al. 1980). Lyme investigators were thus able to use their varying clinical treatment to good effect. The discovery of the spirochetal etiology of Lyme disease would later provide an additional rationale for antibiotics and a guide for their use (e.g., using larger intravenous regimens for late symptoms as in syphilis).

In sum, I have outlined how several factors—cultural, disciplinary, methodological, biological, attitudinal/intellectual, ecological, and professional—each contributed to the manner in which American scientists distinguished Lyme disease from its antecedents. These different factors can be likened to a set of partly detailed transparencies, which, when projected together, result in a coherent image of a new disease. I have stressed that Lyme disease did not have to be constructed as a new entity, not that it was unreasonable to have done so. Had the dermatologists who first reported the case cluster in Groton, Connecticut been solely responsible for the investigation, they might not have given the same prominence to either arthritis or the newness of the ailment. The construction of Lyme disease had important consequences for early etiological investigation and treatment. As we shall see, it made a timely entry into a public debate about the significance of chronic disease, personal responsibility for disease, and the authority of science.

Social Consequences of Lyme Disease

Public and Professional Responses to the Emergence of a New Disease

Lyme disease's construction as a new disease probably has contributed to the very different public perception of its severity in the United States as compared to Europe; what is new is often more frightening. The resulting public and media attention may also have contributed to the dramatic increase in reported incidence in the 1980s. In this regard, Lyme disease resembles more controversial diagnoses such as anorexia nervosa, in which visibility and incidence appear to be intricately related (Brumberg, in press).

Labeled as a new disease or not, the appearance of a hitherto unfamiliar infectious disorder in epidemic proportions in the United States was likely to attract attention. Heightened concern about the severity of Lyme disease may reflect anxieties borrowed from the AIDS epidemic. The high level of public preoccupation with health and fitness in recent decades may also be an important influence.

However these social factors are weighted, researchers, clinicians, patients, lay advocacy groups, journalists, and the public at large have increasingly engaged over the last few years in a spirited, if ill-defined, debate over the significance of Lyme disease. The key issue is whether Lyme disease is an acute, self-limited, rare, treatable, minor disorder or a chronic, serious, widespread, poorly treatable threat to the public health. In medical circles it is customary to blame the media for exaggerating the risks of contracting and suffering the disease. It makes good news to report the appearance of a new, serious, and mysterious disease in suburban America. Despite this bias, much of the media coverage reflects as much as it creates underlying attitudes toward Lyme disease. Newspaper accounts stressing that Lyme disease is a growing public-health threat, for example, rely on quotes from prominent medical authorities and lay figures.

Polly Murray, one of the two Lyme women whose persistent pleas to the medical community led to the "discovery" of Lyme disease, is quoted as saying, "It's a nightmare. . . . Out here we have ticks all over, and no one knows how to stop them" (Ravo 1987, A1). Lyme disease also emerges as a threat in seemingly straightforward descriptions of the disease in newspaper accounts: "Lyme disease, a tick-transmitted

malady that can result in severe and prolonged arthritic symptoms and neurological and heart disorders, is spreading rapidly in southern New England, New York and New Jersey" (Bryant 1988, C13). This characterization is typical of the way reports leave the risk of serious symptoms unqualified. Severe symptoms could more accurately be described as occurring rarely or in a minority of patients. Rather than analyzing probabilities and medical uncertainty, scientific correspondents for newspapers and television have given extensive coverage to more worrisome features of Lyme disease: its increasing incidence, the recognition that chronic disease may develop despite early antibiotic treatment, and the potential inaccuracies in serological testing.

Some accounts, however, quote or paraphrase doctors and public-health officials who urge a more "reasoned" approach to understanding Lyme disease's significance. One newspaper reported that the CDC "has been deluged by sometimes hysterical calls about Lyme disease from the public and physicians" (Brody 1988, B8). Another noted that "doctors say a kind of 'Lyme hysteria' has taken hold" (Sobel 1988, B23). Articles quote physicians' reactions to Lyme "hysteria":

> Lots of people who are stressed out or who have chronic fatigue syndromes are picking this disease and finding physicians to be willing accomplices, willing to treat them with expensive, even experimental antibiotics, in the absence of real proof that they have Lyme disease. . . . Not surprisingly, they don't get better. (Sobel 1989a, B6)

Many physicians, moreover, use market metaphors to explain the popularity of Lyme disease. For example, one physician compares Lyme's growing incidence to a "little new company that is growing rapidly" (Voelker 1989, 11). A market for somatic labels exists in the large pool of "stressed-out" or somaticizing patients who seek to disguise an emotional complaint or to "upgrade" their diagnosis from a nebulous one to a legitimate disease. In previous years, sudden increases in diagnostic labels not otherwise justified by epidemiological evidence have included hypoglycemia, total allergy syndrome, and chronic Epstein-Barr virus infection. Today it is Lyme disease.

Other physicians, who view Lyme disease as a serious threat, have also participated in the public debate. Two doctors objected to a newspaper portrayal of Lyme disease as nonfatal, noting that "sudden cardiac death has been reported on one occasion" (Falvo and Nadelman

1987, 26). They expressed concern over the fate of patients with serological evidence of infection but no symptoms: "the long term prognosis for these people is unclear." Falvo and Nadelman also noted that "we do not yet have an adequate means of preventing tick bites in an infected area." These doctors explicitly link their view of Lyme disease as a serious threat to a call for funding research, suggesting at least one possible motivation for their position. One might argue that a single reported fatality among many cases does not make Lyme disease fatal, that there is little reason to worry about asymptomatic positives, and that it may not be feasible or cost effective to try to attack Lyme disease by preventing tick bites. But the constraints of media space, time, and outlook usually limit the debate over Lyme disease's seriousness to assertion and counterassertion.

Lyme Disease as a Chronic Disease: Problems of Diagnostic Testing

There is no question that *B. burgdorferi* infection can cause chronic symptoms. We now recognize that many of the initial cases of arthritis that launched Lyme disease represented its late stages. Nevertheless, the prevalence and diagnosis of chronic Lyme disease have been controversial. One aspect of this problem is that widespread antibiotic therapy for Lyme disease and other infections has made it very difficult to know just what late or chronic Lyme disease "really" looks like. There is no accepted natural history with which to compare cases (textbook descriptions of syphilis would be equally impoverished if they were based on the presently infected, who rarely show the hallmarks of late disease). Many physicians believe that chronic Lyme disease is overdiagnosed, resulting in a distorted clinical picture. We can identify at least six technical or conceptual problems that diagnostic testing for chronic Lyme disease entails at present.

First, there is the nonspecific symptomatology of many patients with late disease. Even before the current controversy about its chronicity, Yale investigators noted that some chronic cases could be misdiagnosed as fibromyositis, polymyalgia rheumatica, or psychiatric rheumatism. Exacerbations of late disease follow an unpredictable course, allowing psychosomatic speculation by patients and doctors: "In several instances, patients thought that emotional stress or trauma to the joint precipitated attacks. Perhaps these events altered immunoregulation in favor of the spirochete. . ." (Steere, Schoen, and Taylor 1987, 729).

The second problem is often presented as a technical one: there is no perfect test for active, chronic infection. Serological tests are neither 100 percent sensitive nor specific. As a result, their use in a population with a low prevalence of disease means that many who test positive will not be truly infected. Even if the sensitivity and specificity of Lyme tests improve, it is likely that the numbers of worried but uninfected people will increase as well, keeping the test only marginally useful.

Third, it has been demonstrated that some Lyme-disease patients who take antibiotics shortly after infection will never develop an antibody response (Dattwyler et al. 1988). Such patients could have chronic disease without serological evidence of infection. Much of the booming interest in chronic Lyme disease followed from this controversial report. Fourth, even when antibodies are present, the result only means that one has been exposed to *B. burgdorferi* at one point in the past. A positive test does not necessarily indicate active infection. Fifth, there is the problem of interlaboratory reliability. Many patients test positive in some laboratories and negative in others. Finally, there is the problematic relationship between a positive test and other diseases. For example, one report (Waisbren et al. 1987) demonstrated the presence of Lyme antibodies in four patients who had been diagnosed as having amyotrophic lateral sclerosis (ALS, or Lou Gehrig's disease). Once the question is raised, only careful epidemiological investigation can answer whether Lyme disease is associated with ALS or if, as is more probable, the association is spurious.

Because Lyme disease is socially perceived to be a fashionable diagnosis with a large market, these problems with Lyme testing are especially troublesome. Nevertheless, Steere (1989) believes that clinical diagnosis of chronic Lyme disease remains highly reliable if one limits the diagnosis to patients who have some objective signs of disease, at least intermittently, along with a compatible clinical presentation. However, such observations do not exclude the possibility that those individuals without objective findings and serological confirmation might still have chronic Lyme disease—a possibility most clinicians consider to be unlikely even as some patients aggressively pursue it.

Chronic Lyme Disease and the Social Negotiation of Its Meaning and Treatment

Despite promises of a new and better laboratory test indicative of active infection, the problem of correct diagnosis will not soon disappear. The

doctor who cares for the patient with long-standing joint pain, fatigue, and weakness will still have to make difficult judgments about the degree to which active infection adequately explains his or her patient's suffering.

Diagnosis of chronic Lyme disease can be seen as a particular instance of a more general problem in chronic disease: that of distinguishing disease from illness. In Kleinman's (1988) usage, disease refers to the biological aspects of sickness, whereas illness refers to the subjective experience. The experience of patients diagnosed with any disease can be parsed into these categories. What distinguishes chronic Lyme disease is the especially problematic negotiations between doctor and patient concerning what is disease and what is illness. "I've talked to hundreds of people (about Lyme disease) over the past four years, and this has created a sense of distrust between patients and physicians," said a prominent Connecticut health official. "The patient says, 'I have it,' and the physician says, 'No, you don't' " (Voelker 1989, 11). When one is not sure whether a patient has "disease," then both doctor and patient have room to speculate about the way in which life stress or other emotional problems may be expressed in bodily symptoms.

These difficult negotiations extend to therapy. Increasingly, patients are labeled as treatment failures because they go on to develop chronic symptoms despite early therapy, or because they present with late stages of Lyme disease that do not resolve with antibiotics. It is unclear whether the increasing amount of "treatment failure" results from specific biological factors (e.g., central-nervous-system "hideouts" of infection, antibiotic resistance, or postinfectious immunological processes) or from mistaken diagnosis and factors best thought of as resulting from illness. "To be honest," one doctor noted, "I don't know what to do with the patients who have recurrent symptoms after they've been treated, and I don't think anyone else does either. . . . The patient is frustrated and the doctor feels helpless" (Foderaro 1989, B4).

In these complex negotiations, telling a patient that there is no definitive test for chronic Lyme disease or that there is no known effective treatment after antibiotics have been tried, may leave him or her feeling rejected. That so many cases are either self-limited or respond promptly to antibiotic therapy probably encourages some doctors to suspect a patient who is a treatment failure of in fact being "ill" without being particularly "diseased." Reliance on clinical criteria for proper diagnosis makes the patient's subjective experience central. Thus, there is often tension over the patient's reliability and psychological state.

Lay advocates sometimes see physician resistance to making the diagnosis of chronic Lyme disease as resulting from their ignorance of the ailment's protean nature. Polly Murray, encouraged by her own role in translating the illness of her family and neighbors into Lyme disease, firmly offered doctors her views on the proper approach to this many-faceted disease in a medical publication:

> Some physicians may categorize some of these patients as "chronic complainers." Granted, there may be a few psychosomatics among the patients who wonder whether they have chronic Lyme disease, but it is possible that the vast majority of these "difficult to diagnose" patients, especially in highly endemic areas, may indeed have tick-related illness. It is my feeling that borrelia spirochetes may turn out to be a triggering factor in many diseases that have been described for many years, but for which a cause has not been found. I am hopeful that future research will uncover the answers to many of these enigmas. (Murray 1989, 365)

The gist of this appeal is that doctors should accept the patient's phenomenological experience of chronic illness, even if it is difficult to diagnose disease, because future knowledge might eventually clarify today's obscure pathophysiological connections. It would be an act of medical hubris, Murray implies, to label what we cannot explain today as "psychosomatic." The appeal expresses the hope that future scientific advances will translate today's illness into disease. It articulates a contradiction characteristic of many lay arguments. They depict value-neutral science as the ultimate arbiter of legitimacy while attacking the hegemony of contemporary medicine. It is ironic that those who openly articulate a legitimate subjective phenomenology of illness are more likely to be "old-time" paternalistic physicians than determined lay advocates of particular diseases.

Narrative accounts of chronic Lyme disease in newspapers are reminiscent of accounts by lay persons of chronic fatigue syndrome, whose somatic basis has been controversial, or of frankly stigmatized diseases such as syphilis. These accounts aim to evoke sympathy for the patient's suffering. The pain of the disease is presented as minor compared with the pain of not being believed or having a stigmatized disease. The overwhelming impact of disease on a patient's life is contrasted with the detached world of doctors and medical research. Doctors are portrayed as insensitive to the patient's experience of illness, which includes therapies that often do not work and practitioners who are sometimes unsympathetic.

Three cases in a *New York Times* article on chronic Lyme disease illustrate these points. "Because of Lyme disease, . . . [one victim] walks with a cane now. At the age of 39, she says she sometimes feels likes she is 82. In the last two years, she has seen 42 doctors, spent $30,000 on medical treatment, missed four and a half months of work and experienced a multitude of symptoms, from arthritis and heart palpitations to profound fatigue and depression" (Foderaro 1989). The three photographed faces suggest depression and anxiety. One person has taken Elavil, an antidepressant, which the author euphemistically refers to as a "mood-elevating drug often prescribed for those with chronic illness." None of the information in these vignettes, however, specifically links their suffering to *B. burgdorferi* infection.

One of these patients recalls being shunned by his family, likening his condition to leprosy. "Some members of the family think that maybe it's make believe because sometimes I look good." Such statements provide clues to how Lyme disease, whose acquisition by tick bite is a random, natural event without much potential for blame, becomes stigmatized to a degree. Stigma results from doubts about whether the illness of the person with chronic Lyme disease is caused by disease.

It is clear that Lyme disease has borrowed stigma and other features from general preconceptions about chronic disease: that there is a market for new chronic disease in the pool of would-be patients and that acquiring a medical diagnosis can give legitimacy to one's suffering even as the active search for such legitimation undermines the reality of the condition in the minds of many doctors. As a "new" entity, Lyme disease assumes a frightening visage because it allows fuller expression of the primitive experience of illness as a condition of profound uncertainty, and is thus available to the reflections and anxieties of those suffering ill-defined chronic ailments.

Commercialization of Lyme Disease

Lay concern over Lyme disease has resulted in an increasing number of office visits because Lyme disease is suspected. In endemic areas, there is a thriving market for a reliable diagnostic test. Despite their limited utility in actual practice, Lyme tests have been aggressively marketed by laboratories and promoted by those who want more attention paid to

the disease. Commercial laboratories have developed a rapid urine test for Lyme disease that can be sold over the counter and used like a home pregnancy test (Deutsch 1989). Such a test might be dysfunctional in some situations, as when a "negative" test results in the person not seeing a doctor for treatment of acute Lyme disease.

Factors other than clinical utility fuel development of these tests. Home testing allows patients to diagnose themselves. There need be no uncomfortable negotiations over whether one is sick or stressed, no waiting for appointments or doctor bills. Doctorless disease detection is already common in screening programs, for example, hypertension and fingerstick cholesterol measurements at supermarkets and shopping malls. There is a general trend to market health products and services directly to the consumer, as found in mass-media advertisements for prescription drugs. Reactions to Lyme disease reflect and incorporate these larger trends and at the same time stimulate them.

Commercial interest in Lyme disease extends from diagnostics to prevention. A variety of anti-tick products are offered for sale to the general public. These contain well-known insecticides packaged in new ways for Lyme disease. One of the most popular products is Damminix—tubes of cotton balls soaked in permethrin that are to be strewn about one's property. The development of this product is an example of the complex links between the biomedical investigation of the disease and societal response. In this case, medical entomologists devised the product for research purposes and then marketed it, using their own studies as evidence of its efficacy (Sobel 1989c). There is no convincing proof that this product will be effective in actually preventing Lyme disease, although research suggests that it decreases *I. dammini* attachment to mice (Spielman 1988). The risk, for example, of acquiring Lyme disease on one's property might be so low that the expensive added protection might be of negligible utility. One retailer of commercial anti-tick products lamented, "I hate the idea of making money off an illness, but everyone seems to be profiting from Lyme disease these days" (Deutsch 1989, D8).

Monetary profit from Lyme disease is merely another example of its success in providing value to the different actors who have figured in its development. Investigators, clinicians, patients, and, most recently, politicians who gain publicity for new public-health measures profit less tangibly, but no less substantially, than the makers of home tests and insecticides.

Lyme Disease and the Public Health

Just as Lyme disease has been constructed as a chronic disease, so it has also become a public-health problem. Areas where ticks abound are places of higher risk. Measures to prevent tick bites, to kill ticks, to protect oneself against infection, and to intervene in the tick or spirochete life cycle all have theoretical appeal. Because these approaches are possible, however, does not mean they need to be studied or implemented. That is a question of risks, costs, and benefits, each of which is open to interpretation and negotiation.

There are at least five dimensions to the American public-health response to Lyme disease, which demonstrate the role of social factors in interpreting and negotiating risks, costs, and benefits. First, social factors affect the assessment of proposals to alter the ecology of ticks and spirochetes. Deer-eradication programs have been suggested, a measure tempered for some only by the awareness that there are other hosts for *Ixodes dammini* besides deer (these animals are, however, a crucial link in the tick life cycle). By way of comparison, no one has apparently suggested deer-eradication programs for the elimination of babeosis—which, although less prevalent, can be fatal to individuals without spleens. European scientists and public health officials sometimes express amazement at the draconian public-health measures proposed:

> I recently attended a meeting about Lyme disease held in Bethesda, Md. I heard doctors advocating the wide use of pesticides and the burning of grassy areas to control ticks. If I were to suggest an approach with such drastic environmental consequences in my country it would not be seen as justifiable. (Sobel 1989a, B6)

A second area of negotiation is the degree and type of action individuals might take to prevent Lyme disease. Newspaper reports discuss steps summer campers in endemic areas should take to prevent infection (Sobel 1989b). The pitch of such appeals is often high enough to generate hysteria. Camp owners and others with an economic interest in outdoor activities have to assure clients that everything possible will be done to prevent infection, while not emphasizing Lyme disease to the point of frightening them away. Invitations for an outdoor wedding in the summer of 1989 in an endemic area were accompanied by Lyme-disease literature (C. Rosenberg, personal communication, Au-

gust 1989). Guests attended "on the mutual promise of constant tick checks."

A related third controversy is whether physicians should treat people who have been bitten by a tick in the period before they might develop symptoms of Lyme disease. Some clinical studies suggest that the risk of suffering a side effect of antibiotics is equal to the risk of acquiring Lyme disease from a tick bite, even in endemic areas (Costello et al. 1989). Other investigators have applied elegant decision-analysis techniques to this problem, concluding that persons who are bitten by a tick in an endemic area should probably be treated with antibiotics and not be tested (Schwartz et al. 1989). These analysts do not, however, take into account substantial, if hidden, costs related to the problematic chronic status of Lyme disease. Empiric treatment without testing of asymptomatic people could create a group of people who later might suspect that they have chronic Lyme disease and who could not be "ruled out" by a negative antibody test because early treatment has been reported to abort antibody response to infection (Dattwyler et al. 1988). In general terms, this aspect of Lyme disease embodies tensions in modern clinical and public-health strategies that sidestep the diagnosis of disease.

A fourth aspect of the public-health paradigm, mass screening of asymptomatic people, has been proposed for Lyme disease. For example, newspapers report that some doctors in endemic areas await a more accurate Lyme test, which they will order "as part of their patients' annual check-ups" (Sobel 1988, B23). Despite such plans, there is no definite indication for screening Lyme disease even in hard-hit areas. It is difficult to imagine that the basic criterion for a good screening program would be met by Lyme disease: that is, the ability to prevent serious morbidity or mortality by detecting early, asymptomatic cases. It has not been demonstrated that asymptomatic individuals with positive serologies would benefit from treatment.

Finally, controversy has arisen over whether steps should be taken to protect the blood supply from Lyme disease (Altman 1989). Public-health officials have been criticized for not acting to prevent such transmission. Although there have not been any reports of transmission from blood transfusion, it is theoretically possible because *B. burgdorferi* infection does have a stage in which bacteria are present in blood.

Reactions to Lyme disease in this instance seems to reflect concerns about AIDS. Early on in the AIDS epidemic, before the discovery that

HIV was the etiological agent, there was a debate about the safety of the blood supply. Most now believe that blood-bank officials erred in not taking more aggressive steps to prevent AIDS transmission, rationalizing their inaction by arguing that there was no direct and unassailable evidence that AIDS was transmitted by transfusion (Shilts 1987). Given this recent history, the burden of proof has been shifted to the blood-bank establishment to say why expensive and burdensome actions (e.g., testing donated blood for Lyme antibodies) should not be instituted in a period of uncertainty.

The debate is an example of the problems inherent in developing public policy in the face of medical uncertainty. It is not known what risk, if any, a transfused unit of unscreened blood from a donor in an endemic area poses for the recipient. Public-health authorities may not have felt the need to devote resources to the elucidation of this question because they perceived the consequences of contracting Lyme disease as minor and treatable. Critics respond that these officials have "an ostrich-like attitude towards the possible risk" (Altman 1989, C3).

As a compromise solution, the Red Cross reportedly has required that donors be checked for the characteristic rash before being allowed to donate blood. This is an insensitive, nonspecific, and burdensome way to screen for acute Lyme disease. As another illustration of the split between reasoned and emotional responses to Lyme disease, a group of doctors said they would not personally accept a transfusion of blood that tested positive for Lyme disease, yet the same group would not discard such units as a matter of policy (Altman 1989).

Conclusion

Much of the scientific and lay interest in Lyme disease results from fascination with a new disease. Yet this newness is problematic. The relationship to the ECM may not have been initially clear, but more characteristically medicine's celebratory view of Lyme disease's "discovery" has coopted the earlier history in a variety of ways. I do not intend to diminish the considerable achievements of the Lyme-disease investigators, but rather to demonstrate that both the particular history of the biomedical investigation and its perceived significance have been contingent on social factors.

Lyme disease is increasingly viewed as an elusive clinical entity, despite its straightforward textbook description. Medical investigators complain about the way scientific uncertainty is simplified in the media and the crass commercial exploitation of Lyme tests, treatments, and preventive measures. Doctors often bemoan the faddishness of Lyme disease and the growing number of patients who aggressively pursue the diagnosis. Patients with chronic Lyme disease are angered by the ambivalent way they are treated by doctors. Many investigators, doctors, and patients hope for a technological fix for the dilemma of diagnosis. Very few acknowledge, however, that these are dilemmas posed, but not resolved, by biological knowledge.

Lyme disease thus illustrates how rarely textbook prototypes of a disease, which characteristically fail to discuss these central issues, match the particular clinical encounter. Yet medicine fixes on its canonical descriptions as the rationale for the doctor–patient encounter: finding a specific disease to explain patients' complaints; curing, ameliorating, or preventing disease with actions based on the specifics of the disease's pathophysiology and epidemiology; and making specific statements about the future course of disease.

What is often missing from the idealized description of disease is the sociohistoric context in which new knowledge is constructed. To understand the present controversies over Lyme disease, one has to know its particular trajectory. The present debate about Lyme disease's significance can be viewed as the breakdown of a compromise among biomedical scientists, doctors, patients, and the lay public. Initially, there was something in Lyme disease for everyone: the rewards of discovering a new disease for scientists, and of diagnosing and treating an otherwise frightening disease for practitioners and patients. However, a number of factors led to the dissolution of this compromise. Some factors are relatively specific to Lyme disease, including the problem of seronegative Lyme disease and the aggressive marketing of Lyme products by commercial interests. Other factors are common to contemporary chronic diseases more generally, such as the large market for a new, legitimizing diagnosis and the difficulty experienced by doctors and patients in negotiating a viable and categorical boundary between what is disease and what is illness.

I have aimed to demonstrate how Lyme disease has been "constructed" or "negotiated" rather than discovered. This is more than an exercise in method or the expression of bias. By juxtaposing lay and

medical attitudes and accounts of this recent phenomenon, we see how Lyme disease embodies and reflects aspects of our current and past beliefs about sickness and how these beliefs, rather than being marginal influences on a fundamental biological reality, have shaped almost every aspect of medical practice and lay response.

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