Introduction

Between approximately 1820 and 1880 there was a world pandemic of scarlet fever and several severe epidemics occurred in Europe and North America. It was also during this time that most physicians and those attending the sick were becoming well attuned to the diagnosis of scarlet fever, or scarlatina. They could differentiate the disease from diphtheria by the presence of the characteristic rash, or ‘exanthem,’ that accompanied the sore throat, fever, inflammation of lymph nodes and abscessing of the throat and tonsils.

Streptococci bacteria were probably first isolated by the Viennese surgeon Theodor Billroth in 1874, but the association of hemolytic streptococci with scarlet fever was not demonstrated until 1884, and the specifics not outlined until 1924 by George and Gladys Dick (Dowling 1977). Once there is the onset of symptoms such as sore throat and fever the course of the infection can progress very quickly and children in the nineteenth century epidemics were known to succumb within as few as 48 hours in some cases. Streptococci may be contracted through human contact, airborne droplets, or ingestion. A common source of the bacteria in historical outbreaks was unpasteurized milk handled by infected dairy workers.

When an outbreak occurs, symptoms can vary widely within the population, or even within the same family – ranging from asymptomatic individual carriers to acute rheumatic fever and severe tissue infections. For common strains the highest risk is for young children. Infants often are at lower risk owing to the presence of maternal antibodies, and by age 10 it is estimated that upwards of 80% of children have developed lifelong protective antibodies against the exotoxins (Darmstadt 1998). When President Abraham Lincoln’s son Tad contracted scarlet fever during Lincoln’s first presidential election it was a severe case, whereas Lincoln himself had symptoms of sore throat and headache, and assumed he also had the disease (Dowling 1977: 60).
Severe outbreaks of Group A *Streptococcus pyogenes* have occurred over the past two decades in North America, England, Europe and elsewhere. These outbreaks have occurred primarily among otherwise healthy populations, raising concern about the rise of new virulent strains and accompanying antibiotic resistance (see, for example, Stevens et al. 1989). The return of this nineteenth century scourge prompts consideration of its contemporary manifestations and historical correlates. In this chapter we briefly outline present-day observations and then turn to the historical evidence, using a microepidemiological case study from the Connecticut River Valley of Massachusetts.

We wish to test the limits of historical epidemiology for providing various levels of explanation of disease processes. Secondly, we wish to contrast the pathogenic/virulence evidence with the socioeconomic data for our particular case study. After a preliminary analysis of the available evidence, we conclude that the virulence hypothesis is consistent with our findings, but also suggest that explanations of these events are complex and should encompass socioeconomic and nutritional variables to varying degrees. By starting with an international pandemic, focusing in on a regional epidemic, and then ‘microscopically’ focusing in on four communities, our purpose is to ultimately examine the details about some of the specific individuals who succumbed to scarlet fever. Using the example of the town of Deerfield, we can attempt, literally, to map the course of events in this town during an outbreak. Stylistically and methodologically, we aim to validate the particular and the qualitative knowledge of these events as well as the epidemiological.

**The return of scarlet fever**

Strep Outbreak kills 26 in Texas

El Paso, March 7 (AP) – An outbreak of streptococcus has killed 26 Texans in the past three months, including 9 children, a new report says... The most recent deaths include a 5-year-old Channelview boy who died of toxic shock syndrome on Sunday and a 5-year-old El Paso boy who died on the way to the hospital last week... Health officials have no explanation for the apparent surge, but a Health Department spokesman, Doug McBride, said it may be partly attributable to recent changes in reporting procedures.

*(New York Times, March 8, 1998)*

Beginning in the mid-1980s, increasing numbers of invasive streptococcal infections (including scarlet fever) were reported throughout the United States, Canada, Europe, and Australia (Stevens 1992). Outbreaks occurred between July 1989 and June 1991 in four of seven US Army basic training installations (Gunzenhauser et al. 1995) and Canadian researchers have reported an increasing frequency of all types of *Streptococcus pyogenes* infections among children during the period 1984–1990 (Cimolai et al. 1992). Streptococcal
infections have now become such a public health problem that they have been the focus of increasing research by the World Health Organization (WHO) (Efstratiou 2000). In addition, invasive streptococcal disease, Type A, is a nationally reportable disease in the United States (CDC 1999). Scarlet fever and the associated invasive streptococcal infections can no longer be considered historical oddities, or the outcome of new reporting procedures by health officials, but are instead very contemporary clinical diseases with serious effects.

Symptoms in present-day cases range from classic scarlet fever to acute streptococcal toxic shock syndrome. Numerous deaths have occurred. Case fatality rates as high as 50% have been reported (Spencer 1995). In the United States during the period 1980–1998, the number of deaths per year from scarlet fever ranged from zero to three. This represented an average of 1.1 deaths per year during the 1980s and 0.6 deaths per year during the 1990s (Centers for Disease Control and Prevention data from the Compressed Mortality data set). For all invasive Type A streptococcal infections, however, there were approximately 10,200 cases (1,300 of them fatal) in the United States in 1998 (CDC 1999).

This increase in the reported number of streptococcal infections in recent decades has been compared to the pandemic of fatal scarlet fever in 1825–1885 (Katz and Morens 1992) and suggests the possibility of varying virulence factors throughout time and across geographic areas. Several researchers are reporting the presence of new variants of the speA gene, which encodes the pyogenic exotoxin causing hemolytic damage and producing the characteristic red rash. New variants have been suggested as markers for monitoring future potential outbreaks (see, for example, Musser et al. 1993).

Because the decline of severe symptomatic scarlet fever in the US began sometime after 1880, but well before the antibiotic era, many have suggested that attenuation of the streptococcal organism may have occurred, only to arise again in the 1980s (see, for example, Stollerman 1988). These arguments center on hypotheses regarding host–pathogen coevolution, or on the absence of compelling nutritional evidence or on the lack of effective medical or public health intervention. Others have argued for a significant socioeconomic component mediated through nutritional status and health (see, for example, McKeown 1976; Szreter 1988; Hardy 1993; Duncan et al. 1996).

**Historical epidemiology in bioanthropological perspective**

A comprehensive view of an historical epidemic from a bioanthropological perspective involves at least four domains of interest.

1. Historicity: situating the epidemic in time and place.
2. Medical history: understanding how the epidemic was viewed ‘then.’
3. Epidemiology: reevaluating the available data using contemporary models.
4. Molecular biology: understanding the pathogenicity and genetics of the disease.

**Molecular biology**

Taking the last topic first, we already have described briefly what is known about Type A streptococcal infections and their potential for mutating into new clones with high toxicity. The most certain method of demonstrating the periodicity of virulent variations of streptococcus is to examine samples of the bacterium over time, and this has, in fact, been done recently using multilocus enzyme electrophoresis on blood samples of individuals infected in Canada in the 1920s and 1930s (Musser et al. 1993). The results of this study support the theory of cyclical virulence as an explanation for disease fluctuations both temporally and geographically. Examination of the serotypes of Type A streptococcus associated with various outbreaks throughout the world revealed an association between the M1 serotype and the most invasive, epidemic-like outbreaks (Cleary et al. 1992; Colman et al. 1993; Efstratiou 2000; Spencer 1995). This further supports the theory of fluctuating disease virulence.

It is less likely (though not impossible, however) that we will be able to isolate streptococci or the speA gene from the remains of individuals living in the nineteenth century. Meanwhile, if we are going to suggest evidence for
virulence in the nineteenth century it would most likely be on the basis of the absence or lack of evidence for alternative explanations, with virulence being the residual explanation. Retrospective interpretation of past events is a hallmark of anthropology and historical epidemiology – for better or worse – and strong inference usually involves the rejection of some variables, with a lack of absolute certainty for those remaining.

Historicity

The demographic patterns of this historical period in the Connecticut River Valley of Massachusetts have been described in detail elsewhere (see, for example, Swedlund et al. 1976; Swedlund 1990). In brief, it was a rich agricultural valley with emerging industrialization between 1850 and 1900. The four towns selected for this microepidemiological study are Deerfield, Greenfield, Montague and Shelburne, Massachusetts. They vary in their dependence on agriculture, commercial, and industrial economies, but all are diversified with levels of each. Our data are drawn from the sum of deaths occurring in each community between the years 1850 and 1910, extending the time so that we could follow post-1880 transitions. In this chapter we concentrate on the 1858–59 epidemic, using the 1867–68 epidemic simply for the purposes of examining periodicity.

Fig. 8.2. Map of the study area in Massachusetts with the center villages of the four towns of primary interest. Working clockwise from the upper left quadrant: Shelburne Falls, Greenfield, Montague, and Deerfield. Rendered by authors.
Scarlet fever

The scarlet fever, which has very seldom prevailed in this town, is now prevailing quite extensively among children. We hear of its prevalence also in several of the neighboring towns.

(*Greenfield Courier*, December 27, 1858)

On the eve of this first epidemic, in December 1858, a growing uneasiness overtook the Valley. By means of the local newspapers, and no doubt word-of-mouth, knowledge of the epidemic’s presence elsewhere was widespread. By then, numerous individuals were being diagnosed with scarlet fever locally. In January 1859, things became much worse, with many more infected and several children dying in the four towns. A full-blown epidemic was at hand, and, as we see below, in Deerfield the main street was a site of considerable angst for the families residing there.

There were 15 deaths in the town of Greenfield in the month of January, 1859—3 of consumption, 9 of scarlet fever, 1 of whooping cough, 1 of inflammation of the brain, and 1 accidental.

(*Greenfield Courier*, February 7, 1859)

**Medical history**

Physicians were ubiquitous in western Massachusetts by the mid-nineteenth century. Each of the study communities had at least one doctor, Greenfield had several, and the relatively small town of Deerfield (population 3073 in 1860) had as many as five doctors practicing between 1858 and 1870 (L. Dame, no date). At least three of these doctors were present at meetings of the Franklin County Medical Society in 1858 and 1859 when treatments for scarlet fever were discussed, including the use of ‘tincture of veratum vivide’ (FMS no date). Medical knowledge was also increasing at the time and, as noted, the diagnosis of scarlet fever was becoming reasonably reliable. What was not reliable was any effective level of therapeutics or treatment that physicians could administer to the afflicted. Even after the bacterium was identified in the third quarter of the century, an effective treatment was not available until many years thereafter with the development of antibiotics.

*For scarlet fever:*

- keep the bowels free by a free use of fruits, berries and cracked wheat.
- keep out the rash by the prevention of chilliness and looseness of bowels.
- keep down thirst and fever by acid drinks, lemonade, buttermilk, etc.
- keep the room cool and well ventilated.
Scarlet fever epidemics of the nineteenth century

- if there is a tendency to debility, add some meat, poultry, and soups, with bread crust, to the diet.
- In great heat of the skin, sponge it frequently with tepid water.

(Hall’s Health at Home 1872)³

At the time of this epidemic, however, prescriptions were abundant. By consulting doctor’s journals and reading the home manuals written by the physicians of the period we noted many concoctions available for scarlet fever:

Carbonate of ammonia, sixty grains; paregoric, two oz.; wine of ipecac, forty drops; water, six oz. Give one tablespoon in some lemonade, or vinegar and honey every three hours.

(Hall 1872: 756)

Because scarlet fever was by no means always fatal, and because the opiates, alcohol, and other ingredients might make patients feel some relief from symptoms, there were occasions when physicians were highly valued for their efforts, despite their limited capacities to treat and certainly to cure.

Northfield – The scarlet fever is prevailing to considerable extent, but our physicians thus far have managed it very successfully. We think it no more than a just compliment to them to say they are as good as the county affords, attending early and late and at all hours, faithfully, to their not infrequently arduous duties. Success attend them.

(Greenfield Courier, February 12, 1859)

Epidemiology

The 1858–59 epidemic of scarlet fever impacted Massachusetts severely. This epidemic caused 2089 deaths, 95% of which were of children aged 15 years or younger. In our four-town analysis the percentage aged 15 or younger was 96.9%. There were 227 deaths from categories that likely were reducible to scarlet fever (e.g. scarlatina, etc.) in the four towns between 1850 and 1910. Of these, 220 cases occurred among children under the age of 16; 206 of them to children less than 10 years of age. From the total of 206 childhood deaths in the four towns between 1850 and 1910, we focus on 89 deaths to children for the epidemics of 1858–59 and 1867–68, and then on a small number of those occurring specifically in the town of Deerfield. In the four study towns, it should also be noted that 42% of all childhood deaths during the years 1858–59 were due to scarlet fever.

The mortality trend for Massachusetts during 1858–59 by month is depicted in Fig. 8.3. Despite a relatively small number of cases, our results
Fig. 8.3. Number of deaths due to scarlet fever, by month: Massachusetts, 1858–59.

Fig. 8.4. Percentage of deaths among children <16 years due to scarlet fever, by age: selected Connecticut River Valley towns, 1850–1910.

tend to fit the patterns outlined both in the Commonwealth as a whole and in the epidemiological literature. The age distribution of fatal cases, illustrated in Fig. 8.4, showed remarkably few infants, with the highest proportion of deaths occurring between the ages of 2 and 8 years. A low rate among infants
is consistent with the presence of maternal antibodies in children in their first 12 months. An analysis of the sex distribution of cases reveals a slight but significant difference by sex, with somewhat more boys dying than girls \((p \leq 0.01, \chi^2 = 6.37)\).

The two principal epidemics of the region show up in sharp relief for the towns (Fig. 8.5), and the reduction in epidemics in these towns by 1890 is consistent with the reduction of scarlet fever deaths throughout the entire region at this time. An analysis of the linked data from the Census and the mortality data for the following two years in the nearby cities of Northampton and Holyoke mirrors this trend. During the period 1850–1910, the proportion of deaths in the entire population due to scarlet fever ranged from 0 to 6.4\%, with the peak in both towns during the 1870–72 period and diminishing to less than 0.4\% by 1910. The age distribution of deaths by scarlet fever in Northampton during 1870–72 (the period for which there were enough deaths to yield a large enough sample size) was very similar to that of the four study towns. During these years, children aged 2 through 8 had the highest proportion of deaths due to scarlet fever. In 1870–72 in Northampton, 17.2\% of deaths among children under 16 years was due to scarlet fever. The 1867–68 epidemic in the four study towns does not show any particularly strong seasonal patterns, but the 1858–59 epidemic shows a sharp peak in January 1859 and a strong secondary peak in July, as illustrated in Fig. 8.6.
Scarlet fever in Deerfield, Massachusetts

In Deerfield, Greenfield, Montague, and Shelburne we see a microcosm of the larger State and County events. The rank order of the proportion of deaths among children less than 16 years due to scarlet fever in these four towns during the study period was Greenfield (5%), Deerfield (6%), Shelburne (7%) and Montague (2%). It is interesting to note that the town of Montague lies across the Connecticut River from the other three, and this may have provided some natural barrier to the spread of the infection, given the reduced frequencies apparent for Montague.

Our current research is focused on the systematic tracking of individual households in which scarlet fever deaths occurred, and an example from Deerfield illustrates this approach. In the Manuscript Census of Massachusetts for the town of Deerfield in 1860, we found 12 households altogether in which children were lost to scarlet fever in 1859. The average age of the children was 3 years 11 months and the age distribution fits the profile for risk of death from scarlet fever very well, which is often given as being between 2 and 8 years. Somewhat serendipitously, these are evenly divided between farmers and laborers (mostly mill operatives in the local cutlery factory). There are six households of each category. In this sample the farmers’ average wealth in real and personal property is US$4354.33. For the laborers it is US$635.50. In other words, the
farmers were on average 6.8 times wealthier than the laborers – and among the wealthiest citizens of the town – yet both lost equal numbers of children.

Of the seven deaths occurring to children in January 1859, two occurred to partially linked families who were most likely recent immigrants, and who resided in Cheapside, the most working-class district of the town. Four occurred to farmers, and one we have as yet been unable to link. Of the deaths that occurred in farming families, we illustrate the locations of three of the residences in Fig. 8.7. A cluster of deaths occurred at the north end of the old village center in January, claiming three boys who lived in close proximity to each other, two of whom were very close friends. On January 10, Elihu Ashley, age 8, died, on January 18, Jesse Stebbins, age 2 years and 4 months succumbed to the disease, and then, on January 20, Frank Sheldon, age 6, joined his friends in death.⁵

Early photos or renderings of the homes of the street (see McGowan and Miller 1996), and other records reveal that these were not only the sons of successful gentleman farmers, but also among the most prominent families in the community. By way of contrast, Elihu Ashley and Frank Sheldon were buried in Laurel Hill Cemetery sitting on a hill above the prosperous section of Deerfield, whereas Gustavus Roos and Maria Logan were buried in the ‘German section’ of the Green River cemetery in the neighborhood of Cheapside. All were victims who died in Deerfield during the month of January.

As we slowly add to the list of individuals linked in the 1867–68 epidemic, and in the town of Greenfield, we are seeing similar trends: deaths occurring in households from middle-class neighborhoods, or productive farming sectors, as well as those occurring in neighborhoods known to be much less well-off. In the case of Deerfield, the former are not households that would lack for food, warmth, or other economic amenities. Neither of these neighborhoods are characterized by large tenement dwellings, but a significantly larger number of poorer citizens likely resided in the Cheapside district of Deerfield, and in multi-family dwellings.⁶

Discussion

Based on typical epidemiological patterns for scarlet fever from accounts of the past, it would be common for many people in any given community to be infected and symptomatic. Within families several members might be in various stages of the disease, with the children aged 2–8 most vulnerable to severe symptoms, and perhaps death. Even from our very small samples these patterns are apparent.

The data component of this study has been derived from a predominantly rural area of western Massachusetts in the nineteenth century. Unlike previous
Fig. 8.7. Deerfield Centre Village showing the three households affected by the epidemic (arrowed). From Atlas of Franklin County, Massachusetts, F. W. Beers and Company (New York, 1871). Reproduced with kind permission of The Henry N. Flynt Library of Historic Deerfield.
analyses of scarlet fever epidemics (see, for example, Hardy 1993; Duncan et al. 1996), this population was less subject to urban patterns of disruption in food supplies as a function of economic recessions. While the Massachusetts population was residentially and occupationally diverse at the time of study, we are confident that nutritional stress would be much less severe than in poorer, urban populations in North America and Europe at the time. The limited but detailed evidence available in our preliminary analysis suggests to us that neither nutritional nor socioeconomic factors can be considered sufficient risk factors for explaining the distribution of scarlet fever deaths in our sample during the epidemics of 1858–59 and 1867–68 in the Connecticut River Valley of Massachusetts.

Research on England and Wales over the last decade or two has re-evaluated McKeown’s classic study (1976) of mortality decline in Britain, in which he argued that this decline was due primarily to improved standard of living, which translated into improved nutrition for those at greatest risk for infectious disease. More recent research (see especially Szreter 1988; Hardy 1993) has carefully evaluated the historical record and McKeown’s arguments. These studies find a much more compelling explanation in the rise of social interventions inaugurated primarily by public health reformers in the mid-nineteenth century. And, in the case of scarlet fever, they see a case of a disease that may be the best candidate for attenuation of virulence among all the major infectious diseases investigated by McKeown and others.

The strength of the arguments now seem to favor the health reform/social intervention hypothesis over the standard of living hypothesis for the greater proportion of infectious disease mortality in England and Wales during this period. However, an important element of nutritional status should not entirely be dismissed in the British case, nor in North America, even though its correlation with standard of living may be more complex than that encompassed in the traditional arguments and debates (see, for example, King and Ulijaszek 1999: 164–71). We are referring to the close and perhaps synergistic interrelationship between nutrition and infection that can occur in a host of disease contexts and which can be influential not so much in who becomes infected, but in who survives. A mother’s ability to pass on sufficient antibodies to her child surely depends on her own nutritional status and on her ability to effectively nurse. Likewise, an infant or child who is compromised nutritionally – when ‘challenged’ to mount an effective immune response – is perhaps less able to do so than his or her well-nourished counterpart. Important in this regard are key micronutrients that may or may not be available in the impoverished diet.

A growing body of clinical research is demonstrating that infections in infancy and early childhood, particularly dysentery and enteric infections, affect the child’s ability to resorb key micronutrients and can then leave the child
more susceptible to severe outcomes from secondary or later infections (see, for example, Guerrant et al. 2000). Although there do not seem to be studies specifically referring to this sequela in relation to scarlet fever, we consider it an important problem for many of the diseases we are currently investigating in the historical context, including scarlet fever. Moreover, this scenario is a very likely one for urbanizing and industrializing communities in nineteenth century Britain and North America, where dysentery among infants and children was ubiquitous due to unsanitary living conditions, artificial feeding of infants and substandard working conditions. We do not raise these issues of nutrition here because we have the data to test a specific hypothesis. Rather, we believe that nutrition is important for the reasons described above and should not be dismissed entirely because of the somewhat naive way in which it was proposed by McKeown and others. McKeown's (1976) model was developed to explain a long secular trend in declining mortality that was believed to have occurred in Britain (and the US), but, in fact, during rapid industrialization in the early nineteenth century UK and in the later nineteenth century US, mortality for children was not showing a systematic decline, but in fact was on the increase at various points in time. The water- and food-borne diseases to which many children and some adults succumbed certainly can be expected to have been accompanied by an important set of nutritional risk factors.

Conclusions

While lacking sufficient data at this point to make the strongest possible case, our preliminary findings lead us to the following provisional conclusions.

1. Molecular biology. From molecular biology we find clear evidence for the capabilities of evolving virulence and attenuation in Streptococcus pyogenes, particularly with regard to what is being learned about the speA gene and associated M proteins. The fact that a virulent strain was cultured from tissues preserved from the early part of this century pushes our knowledge back closer to the time of the pandemic in the nineteenth century.

2. Historicity. The time, place, and nature of the epidemic in western Massachusetts is well established by vital statistics data derived from the target communities and from the historical record as revealed in newspapers and regional writing of the day. We can date the incidences quite specifically and graph their trajectories.

3. Medical history. Documents from the time suggest that the medical community could offer little in the way of treatment of those infected with scarlet fever during the time period under consideration. Indeed, many medical historians (e.g. McKeown and Record 1967; McKeown 1976; Bynum 1994) suggest
that the decline in scarlet fever occurring after 1880 was primarily related to improved preventive public health measures such as quarantining, improved nutrition, and antiseptic practices. To this, however, we can add one important caveat: if the children of well-off families are consistently under the care of physicians because the family can afford it, then those children may be vulnerable to the complications of treatment as illustrated in the historical example quoted below.

Homeopathy gives belladonna as soon as any dryness or burning is noticed in the mouth and throat, and there is a desire to drink but no ability. Give mercurius in six hours after the second dose of belladonna; and six hours later, arsenicum, if there is great prostration and the ulcers emit an offensive odor. If arsenicum does not restore reaction, then give nux vomica. If inflammation, give aconite, followed by belladonna, if the pulse falls and fever abates. If the skin burns and there is drowsiness and stupor, give opium. If convulsions are present and are not relieved by opium, give zincum. If the eruption is intense, give sulphur.

(Hall 1872: 451)

4. Epidemiology. The epidemiological literature does suggest that there are associations with adequate nutrition and other advantages that can be purchased by a higher socioeconomic status. Duncan et al. (1996), looking at time series analyses of scarlet fever deaths in England and Wales, show a lag effect of deaths that they believe is associated with poor maternal nutrition and subsequent loss of their young children. Since resistance to the Streptococcus pyogenes bacteria would primarily be due to the immune response, with the presence of the proper antibodies, nutrition would play a reduced role. It could only serve either in weakening the mother’s own ability to transmit those antibodies through placental transfer neonatally and nursing postpartum, or by preventing a malnourished child from mounting a sufficient immune response or becoming more susceptible to other opportunistic infections. In contrast, in our microcase study, as many as half of the victims we have surveyed thus far come from families that would not appear to be at risk of under- or malnutrition and whose families could have provided the best level of nutrition and care available at the time. Since streptococcal bacteria can be both respirated and ingested, contamination may be even more significant a problem in this population than issues of malnutrition.

Although we cannot claim that the microepidemiological approach illustrated above can resolve the answer, in light of the evidence presented above, the virulence argument remains to us the stronger residual explanation. It is also the conclusion of Szreter (1988) and Hardy (1993), after extensive review of the historical context in England. Given what is being discovered about
virulent and antibiotic-resistant streptococci in contemporary populations, we have no reason to doubt that virulent strains evolved in the past. Medical historians and epidemiologists have certainly acknowledged this without providing much evidence to demonstrate its plausibility. Spontaneous and cyclical changes in disease patterns, often resulting in epidemics, have been noted historically not only in the case of scarlet fever, but also with measles, syphilis, and tuberculosis (Dubos 1980). Continuing research on this topic which bridges the work of epidemiologists, historians, and anthropologists, can only serve to expand the understanding of scarlet fever virulence and of epidemic disease in general.  

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Notes

1 Our understanding of events in the nineteenth century is frequently dependent on reports from the popular media and particularly local newspapers. This contemporary analogy reminds us that our understanding of disease events is strongly shaped by that same media today.
2 At least two strains of *Streptococcus pyogenes* are recognized, M and T. The M and T antigens are cell wall surface proteins eliciting type-specific protective antibodies (Katz and Morens 1992: 298).
3 Hall’s *Health at Home* went through many editions, was readily available to literate citizens of western Massachusetts, and was typical of such advice manuals of the day.
4 Although in the town of Shelburne the reverse appears to be true, with more boys dying than girls. It should be added that the results presented here are not affected
by differing age structures in the four towns, Franklin County, or the Commonwealth of Massachusetts. Censuses reveal very consistent age structures for the age groups at risk.

5 Biological anthropologists continue frequently to avoid putting names and faces on their subjects. When this is done to protect the anonymity of subjects who desire or require it, this is commendable. When it is done with the intent of giving the appearance of some level of greater detachment or objectivity, it is not. The project from which this chapter stems is, in part, about giving ‘statistics’ a face and an identity.

6 Cheapside was a large district at the north end of Deerfield along the confluence of the Connecticut, Deerfield, and Green Rivers. Cheapside was annexed to Greenfield in 1896, and Green River Cemetery is in the Greenfield portion. In addition to some farming areas within Cheapside, there was also railroad access, manufacturing, and commercial activity.

7 There are several other problems with this analysis; for good reviews see Luckin (2000) and Mooney (2000).

8 Although clearly beyond the purview of this chapter, we caution against a third hypothesis that is occasionally proferred by adaptationists, and that is that the human host populations have coevolved genetic resistance to the streptococcal pathogen, as well as others. As Levin and others have shown, the evolutionary odds are generally in favor of the rapidly reproducing pathogen at the expense of the slowly reproducing human host. General immune competence is strongly affected by environmental factors and genetic factors are thought to be present as well. However, since ‘specific immune defenses are adaptive at the somatic level and, as such, reduce the intensity of (and need for) selection leading to germ-line evolution,’ infectious disease is very limited as an effective agent of natural selection in human populations (Svanborg-Eden and Levin 1990: 43).

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