

AN ANALYSIS OF THE 1875-1877 SCARLET FEVER
EPIDEMIC OF CAPE BRETON ISLAND, NOVA SCOTIA

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by
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A candidate for the degree of Doctor of Philosophy

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Dedication

This dissertation is dedicated to the people of Cape Breton and in particular the people of Chéticamp, both past and present. Your enduring spirits, your pioneering efforts and your selfless approach to life stand out amongst all peoples. Without these qualities in you and your ancestors, none of this would have been possible.

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ABSTRACT

An epidemic of scarlet fever on Cape Breton Island, Nova Scotia, Canada between 1875 and 1877 is analyzed in the context of a larger, world-wide pandemic of scarlet fever that occurred between 1825 and 1885. Data derived from public records on national censuses, provincial vital death records and parish records suggest that the epidemic impacted the two main ethnic groups of the island, the Acadians and the Scots, in very different ways. Statistical analysis was done considering the temporal and socio-cultural context of cause of death reporting in order to examine if this initial reading is valid. A deterministic computer model was also created to analyze the effects of each factor on the overall course of the epidemic. Results suggest that although the two groups did experience the epidemic in different ways, this difference is partially attributed to the terms used to describe cause of death information. Occupation, and household type resulting from occupation, is found to be a key indicator of epidemic experience. Differences in person to person contact rate are associated with the different occupations/household types. Ethnic group preferences for the occupations of fishing or farming inextricably tie the issues of ethnicity and occupation together. The number of

contacts people have per unit of time was found to be one of the major factors correlated to the epidemic experience. These results emphasize the importance of socio-cultural factors in an age where drug therapies are becoming less effective. They point to a need to understand the interactions between biology and behavior when examining such complex phenomena as human epidemics.

Chapter 1: Introduction

In the summer of 1995 my parents and I drove to Cape Breton Island, as we had done many times before, to visit with my father's family and friends where he grew up in the Sydney area. At this point in life I had become intensely involved in researching our family genealogy. Not much was known on either side of my family beyond my great-grandparents. All of my grandparents were deceased by 1986, most by 1977, so there were very few sources to turn to for new information. After many years of searching, I was finally able to find the birth certificate and census records linking my paternal grandfather to his family in London, England, from which he emigrated around 1900. Now I was interested in finding out more about my Acadian roots through my maternal grandmother.

My first cousin once removed, Angela M^{ac}Leod (née Gray), was working in the city library of Sydney at the time and also had caught the "genealogy bug". One day at work during our visit she stumbled across a book by Jean Doris LeBlanc entitled *Genealogy of the Poirier Family in Chéticamp, Volume 1 - The André Line*. Taking a quick peek through the book, Angela managed to locate the name of my grandfather (her great-grandfather) "Bill Parrish of England" listed as the spouse of Rose Deveaux. Instantly, we went from having four generations of information to 14. From then on Angela and I sought to fill in the missing pieces. My father, my uncle, and I took a trip to Chéticamp to see if we could find more information. My uncle knew of the cultural centre *Les Trois Pignons* located in Chéticamp and knew that genealogical information

was available there for descendants of *Chéticantins*¹.

We happened to show up just after the arrival of Père Charles AuCoin, a retired priest and renowned genealogist of the area. Amazingly, Father Charlie (as he was known) only came into the center about once a week and we somehow managed to time our four-hour drive to intercept him. We told him who we were and the beauty of rendering Acadian genealogies was revealed to us. “You are descendants of Rose à Nectaire à “P’tit Martin” Deveux”, Father Charlie explained. Because of the similarity in names of the Acadians, and the paucity of surnames due to a small number of progenitors, the typical way to identify oneself in Acadian culture is to recall a few generations of one’s genealogy. Father Charlie proceeded to draw up a full six-generation genealogy, starting with my grandmother, going back almost 300 years. We had gotten some of this information out of LeBlanc’s (1985) book, but Father Charlie’s information gave us a more specific genealogy pertaining directly to us.

Upon returning to Ontario that summer I was motivated to continue my effort to learn more about my genealogy and the Acadian ancestors I had known so little about while growing up. In examining LeBlanc’s book more closely, I found a list of individuals who had been victims of the 1875-77 scarlet fever epidemic. I had recently been exposed to the work of Ann Herring, who was one of my professors at McMaster University where I was taking my bachelor’s degree. I was also taking a demography course with her and started to realize the potential of this situation. I could conceivably combine my love of genealogical research with my love of anthropological research!

I began to design a research project through independent study courses and a plan

¹ Chéticantins are inhabitants of Chéticamp.

emerged that would have me involved in my first field work experience in the summer of 1997. It was then that I began to compare the grave markers in the Chéticamp Cemetery with the parish records. My goal was to examine the scarlet fever epidemic through the grave markers as well as looking at the under-representation of juveniles in both sources. I rented a room with the AuCoin family of Chéticamp and made daily field trips to the cemetery and *Les Trois Pignons*. This project in 1997 exposed me to my first real research venture and is the original basis for the current project. I became more interested in finding out why so many had died in the scarlet fever epidemic and returned to this general question for my Ph.D. research.

During the mid to late 19th century a world-wide pandemic of scarlet fever was present from Australia to Europe to North America. This project will focus on the specific epidemic of scarlet fever that surfaced at various times throughout the 1860s and 1870s in Cape Breton Island, Nova Scotia. Cape Breton Island has a rich social and cultural history surrounding three principal groups of inhabitants — Acadian French settlers, Highland and Western Islands Scots settlers and the original Mi'kmaq inhabitants. This unique cultural setting of Cape Breton Island provides an opportunity to examine how epidemics are affected by social and cultural factors that influence trade, migration and class. The goal is to examine how health, disease, ethnicity and social structure may have interacted within the context of the contact, spread and waning of an epidemic disease.

Scarlet fever is one of several streptococcal diseases caused by the bacterium *Streptococcus pyogenes*. Streptococcal infections can be divided into several alpha-

numeric designated categories based on the type of toxin that each strain produces. Scarlet fever is a manifestation of the largest and most common group of agents, known as Group A Streptococci (GAS). Despite the ubiquity of antibiotics in the general population of both developed and developing countries, there has been a resurgence of often fatal diseases caused by these bacteria in the late 20th century, including flesh-eating disease (necrotizing fasciitis) and streptococcal toxic shock (Katz and Morens, 1992). Though scarlet fever specifically has all but disappeared in the 20th century, it provides a proxy for the presence of streptococcal diseases in general in any time period, since it is easily identified in the historic record. This project is an important area of research because it is believed that study of the complex factors involved in the course of historic health and disease will lead to a better understanding of current and future disease problems and epidemic courses related to these bacteria. This project is also very timely as the world's concerns about terrorism are turning to epidemiologists for answers about the potential threat of biological weapons. This may not specifically apply to *Streptococcus* as we know it, but our knowledge of all epidemic systems needs to be strengthened.

The following research questions will be answered with this project:

1. *Is there a difference in the patterns of spread and timing of scarlet fever epidemics between the two major ethnic groups on Cape Breton Island?*

This question addresses the unique setting of the 19th century communities on Cape Breton Island. Though the original Mi'kmaq inhabitants made up the sole human groups present on the island at the time of European contact, by the 19th century their

numbers had become so decimated by famine, conflict and disease that they only came to represent a small fraction of the population during the time of this epidemic. While the two major ethnic groups, the Acadians and the Scots, co-existed side by side, interspersed throughout the island, a preliminary reading of the historic record suggests that there were differences in how these groups were affected by the arrival of streptococcal epidemics. These differences seem to have weighed against the Acadians and this initial appearance of difference in the record will be investigated.

2. *Does the inclusion of cause of death information for diseases reported as something other than scarlet fever, but which are nonetheless equally likely to have been caused by streptococcal bacteria, present a different picture of epidemic spread than analysis of scarlet fever deaths alone?*

There is some confusion in the historic record about cause of death reporting. Though “scarlet fever” is easily recognized in the records as a cause of death, this is unlikely to be the only manifestation of streptococcal disease that resulted in death during the epidemic. Several other causes of death will be examined and included in the data analysis to see if a different picture of epidemic spread is created when all possibilities are considered.

3. *If there are differences in the patterns of spread and timing of scarlet fever epidemics between the two major ethnic groups on Cape Breton Island, as I expect there will be, what are the behavioral or cultural factors that may be contributing to these differences?*

This project tests, through modeling and traditional demographic analysis, whether different behavioral or cultural factors might be contributing to possible differences in the spread and timing of scarlet fever epidemics. Factors such as household type (e.g., fishing vs. farming), number of children per household, population density, population structure and geographic distribution are analyzed based on data from the historic record.

4. *What is the effect of multiple strains of streptococcal infection on the course of an epidemic?*

Epidemics of *Streptococcus* became pandemic and cycled throughout the 19th century. The surface proteins of the *S. pyogenes* bacterium have helped it to develop into over 130 different strains making full immunity a near-impossible state to achieve. The research suggests that there are several mechanisms these bacteria can employ to avoid detection in the human body and force the immune response to be different for each strain. I was unable to alter the computer model to demonstrate such a situation for reasons explained in the analysis of the model. Therefore, though this question was not answered with this project, the literature suggests that all streptococcal epidemics show this feature and future efforts will be made to model such a scenario.

To address these questions national censuses, provincial vital death registers, parish records and modeling software were used to determine the path the epidemic followed on the island, the timing of its course, the demographic impact of its effects, and the socio-economic and demographic structure of some of the affected towns and families. These sources facilitated the examination of factors such as parental occupation,

family size, number of children, age at death, cycles of pandemics and demographic impact of the epidemic.

Parish record data for the town of Chéticamp were collected in 1997 during the first field work venture to the region. These records were transcribed and type-set by Père Charles AuCoin from original hand-written records. With the permission of then-director of *Les Trois Pignons*, Paul Angus Deveaux, I made photocopies of the transcriptions for 1868 to 1891.

Civil register and census data were collected at the Beaton Institute at the University College of Cape Breton in Sydney, Nova Scotia. The Institute houses an archive of several civil register records, parish records and census records, as well as a wealth of local historic maps, diaries and other historic sources dating from the 17th to the 20th centuries. One month of fieldwork was spent there in the summer of 2002 gathering data recording *all* deaths that occurred in one of the four counties of Cape Breton Island from 1864-1877. Data for the remaining three counties and census counts for the communities of Judique and Chéticamp were gathered in the summer of 2003

The history of the principal ethnic groups, the Acadians and the Scots, as well as the original inhabitants, the Mi'kmaq, provides the context for the relationships among these groups. It also demonstrates the origins of the occupational roles tied to each group. This context is essential to understanding the environment that any epidemic of the 19th century would have arrived in. These subjects are covered in Chapter 2.

A thorough understanding of the biology of the infecting agent, *S. pyogenes*, is also necessary in order to grasp the nature of the epidemic and this is summarized in

Chapter 3. This bacterium in particular has many manifestations in its victims and, for that reason, a broad base of understanding of these effects must be built. The epidemic took place at a time in history when antibiotics were not even conceived of as a cure for the ill. So, we are dealing with a disease in its “natural” state as well as people who had little more than home remedies and sound nutrition as weapons against infections. A detailed review of the biology and effects of *S. pyogenes* in the human host is provided.

Interpreting this epidemic in the way that the participants themselves interpreted it would help us understand how it was dealt with and categorized. An understanding of medicine, both popular and professional, aids in this goal. Medical theory underwent a period of transformation in the 19th century that changed the way people thought about disease and how doctors, and their patients alike, reacted to illness. Medical terminology and language were in flux at this time and these must be understood and appreciated in order to have a solid framework on which to base our own understanding of the events under study. These subjects are examined in Chapter 4.

It must also be pointed out that issues of ethnicity and household type/occupation are difficult to separate in these data. Associations between the two are so close that there is no practical reason to treat them differently. This does not imply that ethnicity is equated with household type or occupation. Rather, it suggests that there are strong traditions of occupation, and thus the resulting household type, within the two main ethnic groups. These traditions muddy the waters making it difficult to determine which one is truly being examined, ethnicity or household type. Therefore, where ethnicity or occupations are being separated out for analysis, it should always be kept in mind that the

two categories are conflated and it is asserted that the two are inextricably linked to one another.

Other studies of scarlet fever epidemics and epidemics of related manifestations of *S. pyogenes* provide a literature base for the study. They also fill out the historic context of the pandemic in other parts of the world. Detailed studies and literature reviews have also provided many of the data on the biology of the epidemics of 1820-1880. Factors such as the virulence of the strains involved in the epidemics, the numbers of people killed, the numbers of carriers, etc., have all provided invaluable information used in the mathematical model and in the general data analysis. These are reviewed in Chapter 5.

Chapter 6 presents the mechanics of disease modeling theory. This subject has many facets in modern research and it is helpful to have some point of reference for where this project fits into contemporary work. The terminology is explained and the specific model expressions and equations are pieced apart.

This project supplies the necessary data to achieve the goals laid out in the research questions, save for the modeling of multiple strains. The sources of these data are described in detail in Chapter 7. Chapter 8 discusses the details of the methods used to analyze these data and Chapter 9 presents the results of the analysis. This approach leads to a richer and fuller understanding of any epidemic in history, even a contemporary one. Even when a researcher is studying his or her own culture or time period, there is a wider context that must be understood so that the viewpoints and interpretations of all those who are touched by the experience of an epidemic can in turn

be understood and integrated into a plan of action. It is suggested here that the “thick description” of an epidemic in terms of multiple lines of evidence and overlapping sources of information can help to strengthen this and future projects. A model could not have been built and an accurate analysis of the data could not have been rendered without the study of all these aspects of the epidemic. People are, after all, the true landscape of human diseases and their social and cultural interactions are as necessary to understanding an epidemic as their biology.

The potential for further analysis of this population and/or this epidemic in other populations is great. There are many other untapped sources of data as well as aspects of the currently transcribed data set that can be analyzed further. Conclusions and future directions of the project are presented in Chapter 10.

Chapter 2: The People and History of Cape Breton Island

The history of Cape Breton Island contains the very earliest seeds of exploration by the First Nations² to the eastern edges of North America as well as those of European exploration of the West. This chapter seeks to establish a brief historic account about a part of the world that really is only known by those who seek to learn about it or who grew up there. Cape Breton is somewhat remote because it is difficult to reach, even with modern roads, since it lies at the eastern edge of North America (Figure 2.1). This was not always the case as the seas, and later the railroads, linked the island to the world economy. With a shift in the focus of transportation to roads and away from rail and sea, Cape Breton has become somewhat less accessible. The island is sparsely populated by today's standards³ and even the major towns contain fewer than 50,000 people. In the 19th century, the island's population consisted of several ethnic groups. The principal groups in shaping the history of the island were the Aboriginal Mi'kmaq inhabitants, the Acadians and the Scots. The small segment of the population was comprised of Irish (MacKenzie, 1979), English and a small number of other European immigrant groups. The ethnic character and social behavior of each of these groups is the direct result of each group's history and likely contributed a great deal to how the scarlet fever epidemic of 1875-1877 played out. The remainder of this chapter will present only a summary of the historical events germane to the inhabitants' responses to the epidemic; it is not the intent to provide a comprehensive literature review of Cape Breton history.

² First Nations is a common phrase used in Canada to refer to Native Americans.

³ 147,454 residents over 6,352 sq. km, or 23.21 persons/sq. km, as of 2001 census.

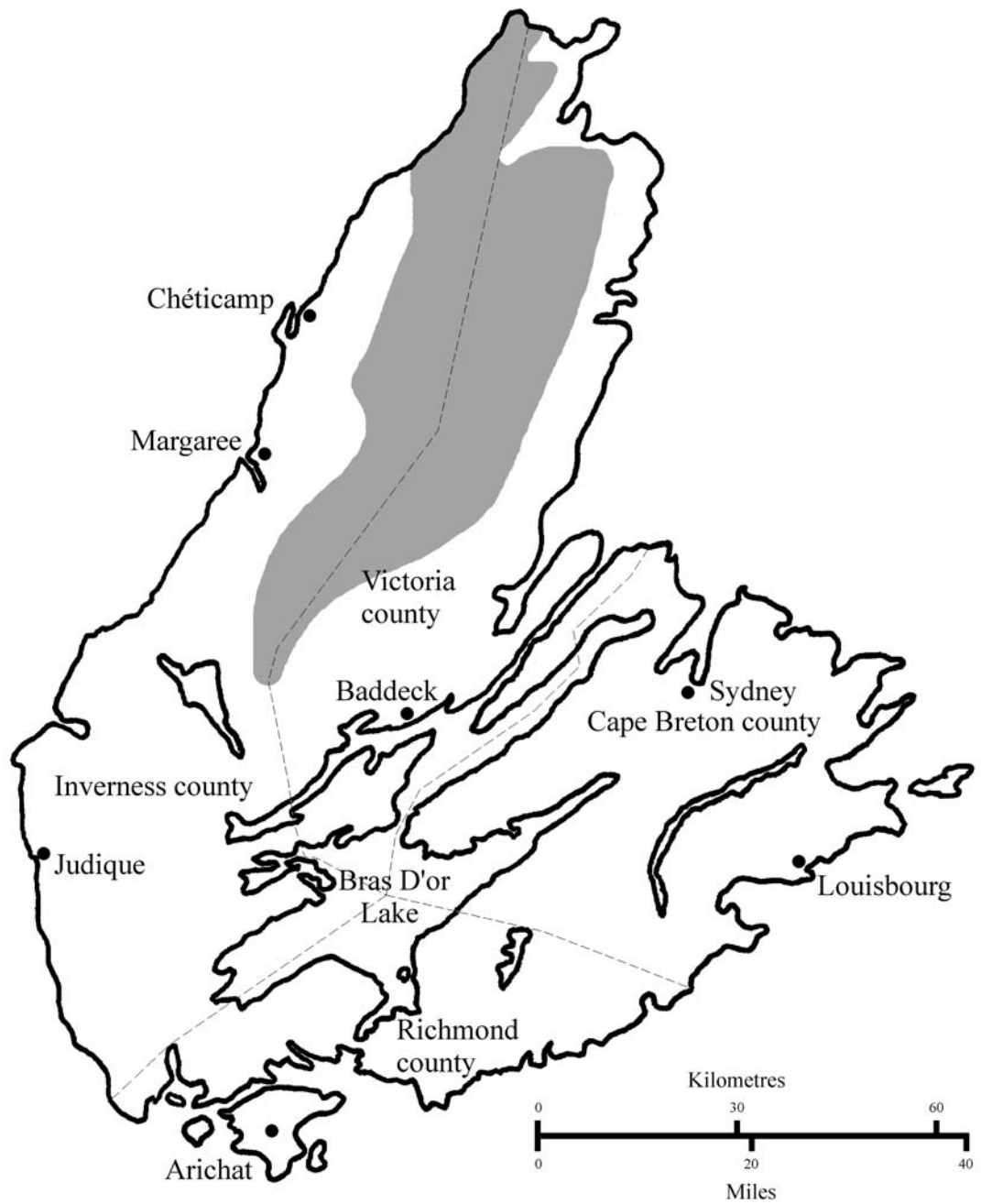


Figure 2.1. Cape Breton Island with the counties and some of the major towns noted.

2.1 The Aboriginal Inhabitants, the Mi'kmaq

The Island of Cape Breton has been occupied for about 10,000 years beginning with her first inhabitants, the Mi'kmaq (pronounced Mig-maw, also spelled Micmac, Mi'qmaq and Micmack). The Mi'kmaq are an Algonquian-speaking First Nation inhabiting the maritime provinces of Canada (Nova Scotia, Prince Edward Island and New Brunswick), the Gaspé peninsula of Québec, parts of Newfoundland and the northeastern corner of the United States.

Their traditional subsistence base was one of gatherer-hunting that involved a diverse array of food sources. Archaeological remains of summer camps show evidence of hunting and fishing with occasional inclusions of whale and porpoise remains. Winter camps were built inland along waterways to supplement the diet of hunted land animals with speared and trapped eels and other water creatures (Saltzman, 1999). This supplementation was likely for variety rather than for a lack of land animals in the winter months.

The Mi'kmaq were part of a loose coalition of First Nations known as the Wabanaki Confederacy, which was established before the arrival of Europeans. This alliance included the Maliseets, the Passamaquoddy, the Penobscots, and the Eastern and Western Abenakis. All of the Wabanaki groups were concentrated in the Northeastern US states of present-day Maine, New Hampshire, and Vermont and the Canadian provinces of New Brunswick, Nova Scotia and Québec. Modern borders were not in place at the time so classification of groups as “Canadian” or “American” is impossible and inapplicable. Their government system was a complex organization of multi-tiered chiefs,

councilors and advisors all presiding over various duties and land districts. These chiefs comprised the body of the Grand Council which made decisions on hunting, fishing and camping rights of families and also functioned as the body which dealt with other First Nations and confederacies (Johnson, 1990; Johnson, 1996; Knockwood, 1992).

It is likely that the Mi'kmaq were one of the first Aboriginal peoples to encounter Europeans. Fishing vessels from Portugal, France and Spain had likely crossed the Atlantic as early as 1510, and probably earlier, to exploit the rich cod-filled waters of the western Atlantic. The Mi'kmaq were not as welcoming as these fishermen would have liked them to be and early records suggest that a Portuguese fishing outpost on Cape Breton island was attacked in the 1520s, deterring at least these Europeans from settling on the eastern shore of North America (Plank, 2001).

Shortly after the arrival of the Acadians in 1604, the Mi'kmaq underwent a lengthy process of integrating their traditional beliefs with those of Roman Catholicism. The Mi'kmaq and Acadians became locked in a relationship of trade and co-operation with one another. With the later arrival of Protestant English and Dutch settlers to New England in the mid-17th century, the Mi'kmaq and Acadians would find a common enemy. The problems that arose and escalated between the New Englanders and the Acadian-Mi'kmaq groups are discussed later in this chapter. However, it suffices to point out at this time that the commonly shared religion and enemies of the Acadians and Mi'kmaq may have strengthened their reliance on one another.

Today the Mi'kmaq of Cape Breton number approximately 20,000 (about 10% of the island's current population) and about one third of this number are fluent in their

mother tongue (Johnson, 1996). Despite their high representation in today's population, little is known about the exact whereabouts and precise numbers of the historic populations of Mi'kmaq from the early 18th century up to the early 20th century. Though this project has tried to include as much historic evidence as possible about the influence of these people on the spread of epidemics on the island, there is scarce direct evidence in the form of enumeration records. Most likely, the majority were not living on reserves as they do now and lived in the forests and "back-lands" as they had traditionally done.

Doubtless their presence and their own socio-cultural decisions and events played a role in the spread of infectious diseases amongst European settlers but evidence for this is sparse. Despite choosing to live apart from Europeans, they were likely not completely isolated, especially with European trade goods only being available in towns settled by Europeans. However, the censuses and vital records fail to accurately represent the numbers of Mi'kmaq that were likely in existence during the years that the records document the presence of Europeans. The reasons for this are discussed in more depth in Chapter 8. It will suffice to say for now that their lives were not as integrated into the European custom of thorough documentation and record keeping as historians would have liked. They did not seem to report the occurrence of any vital events that we would be accustomed to find for Europeans of the same period.

2.2 The Acadians

The Acadians were the first European immigrants to establish a permanent settlement in North America, but Cape Breton Island was not their first home. In 1604, a

group of French settlers came to the northeastern shores of North America and established the colony of Port Royal (later Annapolis Royal) on the southeast shore of the Bay of Fundy on the southwest mainland of Nova Scotia. These settlers came to be known as Acadians. Their name derives from the territory they inhabited, Acadia (*Acadie* in French). This name is attributed to the Italian explorer Giovanni Verrazzano who is said to have originally applied the name *Archadia* to a stretch of coast along what is now part of Chesapeake Bay. He named it this because it reminded him of the beauty of Arcadia's trees in Greece. Over time, through map makers' errors, the name was changed to Acadia and came to be applied to the entire Atlantic region of New France (Cormier, 1999; Casselman, 2004; Centre Acadien, 1996).

The Acadian people mainly originated from the west and central French provinces of Poitou, Aunis, Saint-Ogne and Guyenne (Chiasson, 1998; Brasseaux, 1996:8). Their language, even today, is often described by other French speakers as one frozen in 17th century France. The original settlers brought with them skills of farming and various crafts, especially carpentry and shipbuilding. Fishing seems to be a skill acquired by the later generations, mainly out of necessity. However, it became just as important, if not more so, than the once ubiquitous occupation of farming (Chiasson, 1998).

The Church was an ever-present force in the lives of the Acadians. Acadians were largely Roman Catholics, though some Huguenots⁴ were among them as well. As Roman Catholics, they shared with the converted Mi'kmaq a common faith. Religion provided a common ground that no other European settlers had shared with this First Nation, and the Acadians would come to lean on this friendship later.

⁴ Huguenots is the term used to refer to French Protestants of the 16th or 17th century

The 17th century was one of constant struggle for the new Acadians. A handful of families tried to carve out an existence in a new land desired by many. This century saw constant back and forth attacks from Bostonians and New Englanders, both those who were loyal to England and those who were loyal to the ideals in the seeds being sown for a young United States of America. This conflict prompted many families to emigrate from the Nova Scotia mainland to what are now Cape Breton Island, New Brunswick and Prince Edward Island (P.E.I.). By 1713, the entire mainland peninsula of Nova Scotia was ceded to England under the terms of the Treaty of Utrecht, which ended the War of the Spanish Succession. The rest of New France, including modern-day Cape Breton Island, P.E.I., New Brunswick and Québec, remained under French rule. Of course, in hindsight, this was only the beginning of England's expansion plan to control North America.

The Acadians have been characterized in two ways. The first is that they were a dangerous group of traitors and rebels who greedily wanted their own sovereignty. This view seems to be the one favored by British officials and loyalists opposed to the inhabitation of Acadia by the Acadians (Plank, 2001). The second, and the one that is generally promoted in this work and by many modern historians (Plank, 2001; Brasseaux 1991, 1996; Chiasson, 1998; Griffiths, 1973, 1992), is that they were a group of independent, democratic and hard-working people who recognized the tyranny of France and that of her enemy, England. They worked to be free from tyranny and sought to establish congenial relations with the original inhabitants of the new land they came to in Acadia. They refused to swear allegiance to either France or England because they no

longer viewed themselves as French and certainly weren't going to side with the most powerful empire of the time (Britain). As a result of this pride, perhaps arrogance or foolishness, the Acadians were in the middle of a deadly game being played between France and England. A people caught in the cross-fire, their history became one of being forced to choose; they chose non-violence over violence and paid dearly for it.

In the 1750s, a large number of Acadians realized their difficult situation and chose to leave Cape Breton and Nova Scotia voluntarily. Some of them even fled into the interior of Nova Scotia and Cape Breton to live with their Mi'kmaq allies who had taught them how to survive in this unfamiliar land generations earlier. Acadian families living in various English-controlled garrisons were given the opportunity to swear allegiance to the King of England. Most refused. On September 3rd, 1755, all Acadians that could be found who had not sworn allegiance to the King were:

...lured into their churches by a wily stratagem and made prisoners of the King. All their goods, so long coveted by the English, were confiscated. This was The Expulsion: they forced everyone at bayonet point to embark on boats in the midst of confusion, without concern as to whether they put on the same boat members of the same family. (Chiasson, 1998:3)

The boats were sent in every direction along the eastern seaboard of North America, from Boston to Georgia. Some were even sent back to France. Many died from the conditions on the ships and some ships were not even allowed to disembark upon their arrival at their destinations (Plank, 2001; Brasseaux, 1991). The Acadians were viewed by the English as prisoners of war and no Geneva Convention was in place to assure their humanitarian treatment. Those who escaped made their way to French-controlled P.E.I., New Brunswick, Québec and even to the Louisiana Territory, where they eventually

became known as Cajuns.

As a result of England's certain control of the new territories in North America, Cape Breton Island became annexed to Nova Scotia in 1763. Between 1796 and 1815, England and France became entrenched in various wars over land in North America, including the French and Indian War, The American Revolution and the wars of the French Revolution later known as the Napoleonic Wars. Over this time, some of the scattered Acadian families who survived The Expulsion managed to return to various places in Nova Scotia. The towns of Arichat and Chéticamp became the two major centers of Acadian settlement on Cape Breton Island. In 1790, the first settlers of Chéticamp, known as *les quatorze vieux* (the fourteen founders) were granted 7,000 acres of land by the government of Nova Scotia (Chiasson 1998). From 1784 to 1820, Cape Breton Island remained a distinct province. By the end of 1815, England had established its major foothold in Canada and had established the province of Nova Scotia. In 1820 Cape Breton was annexed back to Nova Scotia.

Caught between two major world powers, the Acadians became prisoners of one, and were then released and finally (unofficially) allowed to live in the land that they were removed from in the first place. This set of events embittered the Acadians and created a fear in them that prompted some unexpected practices. Most notable of these is that even up until the 20th century, fishermen in Chéticamp chose to live in the woods in the mountains because they feared the English would remove them as they had done before. They did this despite being a half to a full day's hike from the shores of the Gulf of St. Lawrence where they fished. This is a practice that I am personally aware of in my own

family at Chéticamp as recently as my grandmother's generation of the early 20th century.

In the years immediately following The Expulsion of the Acadians in 1755, Île Royal (Cape Breton Island) was one of the last remaining possessions of the French in Canada. The French then lost the fortress of Louisbourg in 1758 to the invading English under Admiral Boscawen and lost Cape Breton Island in its entirety by political annexation to the English in 1763. As Plank (2000) tells us, or rather lets the British and New Englanders themselves reveal, the grand picture was to make the Acadians subjects of the Crown in no uncertain terms or to get rid of them. The English perceived the Acadians as a cultural and economic threat in Nova Scotia and resolved to assimilate them linguistically and religiously. The English knew the close relationship that the Acadians had with the native Mi'kmaq. The Mi'kmaq themselves had been troublesome to both the settled New Englanders and the British throughout the Atlantic shore region since Europeans arrived in the region. The Acadians were also distrusted by the English because the English suspected the Acadians would swear allegiance to the French in times of war. The British were logically striving to remove the Acadians from Nova Scotia for good after they proved unwilling to take oath - an oath designed with military service in mind - to the English crown in 1755.

2.3 The Scots

The other major ethnic group to become established on Cape Breton Island was the Scots immigrants of the 19th century. Eventually, this ethnic group became the dominant one in terms of population on the island. Beginning early in the 19th century,

the Scots of the Highlands and Western Isles began to emigrate to Cape Breton Island and Canada more generally. Like the Acadians before them, they brought their skills in farming as well as crafts of carpentry and blacksmithing. Also, like the Acadians, they brought a now archaic form of their language, Scots Gaelic. The modern descendants of these first settlers still retain many of the cultural traditions of 19th century Highland and Western Scotland, especially in their language dialect and musical styles (M^{ac}Donald, 1999). This is most clearly demonstrated by the Gaelic College at St. Ann's, Cape Breton which promotes the use of the Gaelic language and arts.

Religiously, they were Roman Catholics but some of them also embraced Protestantism, Presbyterianism in particular. Ultimately, they were seeking refuge from the landlords, both fellow Scots and Englishmen, who had begun to expand their sheep farms and destroy the lifeways of the Highland and Western Isles farmers. Scotland was becoming smaller, with arable land once farmed for crops given over to sheep farming. In Canada, the Scots found land, as of yet untouched by European farmers, being granted to all loyal British subjects. Those who could afford it, and could weather the ship's journey across the Atlantic, found that the rocky shores of Cape Breton Island looked striking similar to the home they had left. Arriving in a strange place without many facilities was certainly not a paradise but it was theirs for the asking. They arrived amidst the shadows of the two previous groups who had already suffered many great losses trying to stay in this place they came to call home (Hornsby, 1992).

2.4 The 19th Century Cultural Landscape: Cape Breton After The Expulsion

It is an understatement to call the post-Expulsion period of Cape Breton's history one of turmoil (Brasseaux, 1991, 1996; Griffiths 1973, 1981, 1992). For both returning Acadians and new immigrants, the island's economy would never be able to catch up to that of the highly productive territories of The Canadas⁵ to the west and the young United States of America to the southwest. This period would see the introduction of a new group of settlers, the Highland and Western Isles Scots, to an economy that would remain fragmented and a social order that would never become integrated (Hornsby, 1992). This pattern created a unique disease landscape for the arrival of the scarlet fever epidemic of 1875-1877.

Scotsman Samuel Vetch had planted the idea in British Parliament, before The Expulsion began, of encouraging Scottish settlement on Cape Breton and making it a sort of Scottish colony. He argued that the northern latitudes would be perfectly suited to the native highlanders of Scotland and that they would easily make the transition to the new but similar landscape. Though this idea didn't take root among the Scots until very late in the 18th and early in the 19th century, the idea of replacing the Acadians is likely one that never left the minds of the British administrators (Plank, 2001; Chiasson, 1998).

Meanwhile, part of that plan was actualized during the latter half of the 18th century. The Scots did arrive eventually, but the Acadian "problem" did not go away. Some of the Acadians returned in the late 18th and early 19th centuries, or stayed hidden amongst the forests with their Mi'kmaq allies. However, the distance of the American

⁵ "The Canadas" is a term used to refer to the provinces of Upper and Lower Canada (Ontario and Québec) which preceded Canadian confederation in 1867.

colonies from the British homeland had repercussions. The British could not fight a war with the French and the Acadians and keep the growing American colonies under their control at the same time. The British Empire became too thinly spread.

Immediately after The Expulsion, many of the dispersed Acadians initially made their homes in the Atlantic coast states such as Georgia, South Carolina, Connecticut, Maryland, New York, Pennsylvania, but especially in Massachusetts (both the current state of Massachusetts and the part that would later become Maine). Many of these dispersals out of Nova Scotia were not voluntary and resettlement in some areas (for example, Virginia) was outright refused by the colonial governments. Almost all were met with distrust by the local established communities and social, religious, and linguistic segregation was inevitable. Many families landed in poverty with no possessions and no social institutions of their own kind to provide social and cultural support, let alone financial support. Some compassionate members of the community collected money for support of the exiles but many of the Acadians were starving and could not find jobs due to the hostile social environment⁶.

In New England the problem was worsened by many factors. New Englanders were tight-knit, Protestant and English-speaking. They distrusted the Acadians. Many New Englanders had heard embellished stories of the atrocities that Acadians had participated in with Mi'kmaq raiders against New England privateers and rural pioneers. They already had a preconceived notion that Acadians were barbaric, godless peoples that were allied with the much-hated Mi'kmaqs and other Algonquian tribes that had

⁶ Brasseaux's (1991) *"Scattered to the Wind": Dispersal and Wanderings of the Acadians, 1755-1809* is the best source for details about what happened during this tumultuous time in the history of the Acadians.

always been unreceptive towards the New Englanders' presence. Now the Acadians were coming to live in the communities of the colonies under order of the King. Some colonial offices had no warning of the arrivals of various shiploads of Acadians. As Brasseaux (1991) notes, Virginia in particular did hear about the operations underway and refused the arrival of 1,500 exiles to its shores in 1755. They were redirected, at the expense of the colony, to England. Those who were accepted by the colonial governments were torn from their extended families and friends and divided into groups of one to three families per community in many instances — this was the worst-case scenario considering their colonial English neighbors were not very accepting of the new “immigrants”.

Other than being dispersed along the Atlantic coastal states, many escaped exile and made their way to other communities around Nova Scotia, many to Cape Breton Island. Some went to Québec to join relatives and friends. Others tried their luck in Prince Edward Island and New Brunswick, traveling on foot through thick forest with little to eat. A vast number, about 3,500 (Griffiths 1981, 1992), were taken back to France, either having been refused by colonial governments or being stragglers who were rounded up in the years after The Expulsion. For the next 30 years following The Expulsion proper, many Acadians would find each other again in communities in Nova Scotia, New Brunswick and Prince Edward Island, having traveled back from their exile in what is now the United States or from France where their ancestral homeland would not even aid them in their arrival (Griffiths, 1981).

Many Acadians had been captured by the British, dispersed along the coast of the American colonies or brought to prisons in England and Jersey, and were stripped of all

possessions. Returning to Acadia was not an easy task; ship transportation at the time was expensive and unsanitary. These difficulties were exacerbated by their new-found poverty. Many had been ill-treated in prisons and some were not even allowed off their ships. As a result, their health was already in steady decline and many did not survive the journey home to Acadia (Bible, 1906; Chiasson, 1998).

Some United Empire Loyalists⁷ also settled in Nova Scotia as a result of acquiring lands granted to them by Britain in the years following the American Revolution. These people were ethnically English for the most part and more likely to settle in the cities of Halifax and Sydney than in the hinterlands and rural areas on Cape Breton Island, which were to be settled by the highland Scots. At the beginning of the 19th century about 1,500 Acadians were split between Arichat in the southwest and Chéticamp in the northwest. A further 1,000 English and some Irish were split between Sydney (about 800) and Louisbourg and Gabarus (200) (Brasseaux, 1991; Griffiths, 1992).

At the same time, the economic structure of Scotland was changing. Hornsby (1992) describes the situation in Scotland. Essentially, up until the late 18th century the Highland Scots maintained a feudal-like system of landlords and peasants. Tenant farmers rented land from a landlord (usually a clan chief) and landless “cottars” worked for the tenants. Some commons were held and rights to the commons were decided by a town council. Farmers grew crops of potatoes, barley and oats and some raised black cattle for sale to drovers in the south.

⁷ “United Empire Loyalists” is a term used in Canada to refer to English sympathizers living in the American colonies and Canada.

Things began to change again at the time of the wars of the French Revolution, which occurred from 1793-1815. These conflicts caused a drastic rise in prices for wool and meat from the continent. This prompted Highland landlords to clear arable land and make way for large sheep farms, both to offset their own costs and to participate in the profits of war. Those who had traditionally been crofters (tenant farmers) were now pushed to the worst lots of arable land in the British Isles to make way for an industrializing economy, an event frequently referred to by historians as “The Clearances”. Many experienced extreme poverty compared to what they had known before and emigration to North America became the last refuge for many families.

In 1802, the first ship of emigrant Highland Scots arrived in Sydney harbor. The flow of people from the northwest wedge of islands and coastal communities in Highland Scotland would not end until the early 1840s. By this time 20,000 Gaelic-speaking Highlanders came to populate Cape Breton Island and, by doing so, increased the population of the island drastically enough to become the majority ethnic group. By 1871, 50,000 of the island’s 75,000 inhabitants were of Scottish origin. Many of the earliest emigrants from Scotland were the wealthier tenant farmers who could afford the passage to Nova Scotia (Hornsby, 1992). Later settlers included primarily poor cottars who arrived after the 1820 land rush.

The system of government-petitioned land grants was no different for the immigrating Scots than for the Acadians and Loyalists who had arrived earlier. Many of the Scots made petitions for land grants upon their arrival in the early 19th century. The first immigrants obtained the choicest agricultural lands as their new homes. Hornsby

(1992) reports that between 1802 and the mid-1820s, most of the fertile frontlands on the coasts and along the major river valleys were granted. These regions include the western shores between the Acadian settlements at Chéticamp in the northwest and around Inhabitants Bay in the southwest, among the Loyalist settlements surrounding Sydney harbor, the river valleys of the Inhabitants, Mabou, Margaree, Middle, Baddeck and Mira Rivers and at East Bay and McNab's Cove on Bras d'Or Lake. By about 1830, much of the best land had been taken and the poorer lots between the better ones that had previously been snatched up were filled in along the western shores, around the shores of Bras d'Or Lake and in some of the intervale areas of the west. Many of these immigrants had been poor cottars in Scotland and were destined land in Nova Scotia as poor as that which they had in Scotland (MacDonell, 1982:57-103). A vast number of this later wave of immigrants ended up petitioning for government assistance and became the poor of the port cities of Halifax on the mainland and Sydney on the island. In short, those who arrived early enough, *i.e.* before 1830, were fortunate enough to get good quality arable land. Those arriving later did not fare so well on the lower-quality lands (Hornsby, 1992).

The new farms were cheap at the beginning of the 19th century in comparison to Old World land rents; the land was good for the early settlers and the lots were large. Furthermore, the immigrant Highlanders now owned their own land and were no longer in liege to a landlord. News of this new fortune spread through letters to family and friends back home in Scotland. However, the new province was largely a lawless land and the most powerful often ended up with the most land. These problems were resolved through the "moral economy" (Hornsby, 1992:56; Thompson, 1971)

By the 1840s, most of the immigration from Scotland was complete. It is at this time that we see a geographic partitioning along ethnic lines becoming more evident. The re-established communities of Acadians in the areas of Arichat and Chéticamp, almost entirely Catholics, were mainly engaged in fishing and shipbuilding with some farming. Few of the Acadians were participating in the mining industry; and then only to supplement poor years with this activity. The newly arrived Scots Highlanders, mostly Catholics with some Protestants, were mainly participating in farming and mining with some fishing. They arrived in such numbers that they swallowed up the remaining arable front-lands along the coasts and rivers. The Highlanders were likely viewed as another British incursion and were likely greatly distrusted at first, though no source that I have consulted so far mentions much about this interaction. The Acadians knew one thing about the Highlanders - they weren't French and couldn't speak French and therefore they were likely friends with the distrusted English. More on this topic is explored in Chapter 9 in order to understand the social context of disease and its relationship to ethnicity. What kept the two communities separated was the maintenance of their own communities and also the linguistic barrier.

The Acadians maintained a culturally isolated existence, wishing to remain separate from those they considered (correctly or not) as English-speaking. The Acadians had already been devastated by the encroachment and greed of the English and did not wish to tempt fate twice. Though the Acadians traded fish and ships openly within both the domestic English-speaking economy and the foreign economy of the United States, they nonetheless went home at the end of the day and did not participate in other aspects

of life that were not Acadian.

One point of religious importance that should be commented on is the differences between the Roman Catholic Acadians and the Roman Catholic Highland Scots. Though the two groups shared a religion, the availability of services to each group was quite different. Although both had been transplanted to the New World, the Acadians had been established much earlier. This is largely because the economic situation of the Acadians who remained after The Expulsion was probably much better than that of the first immigrant Scots to arrive on the island (Chiasson, 1998). This provided support for many more clergy than the Scots were ever able to provide until much later in the 19th century and created a situation of religious desperation among the new settlers (Hornsby, 1992; Chiasson, 1998). While the Acadians enjoyed a steadier flow of missionaries and Jesuits from France, though not without some effort, the Scots could not long afford to keep such clergymen afloat and were not subject to the same frequency of religious services among their settlements. This paucity of clergy left a great void in the social and religious life of the Scots.

From the Highlanders' perspective, the Acadians were probably not needed for much other than their economic role. The Acadians were experienced Atlantic fishermen and shipbuilders and the Highlanders seemed happy to leave things this way as they infrequently took up this trade. The Scots traded with them, albeit in a somewhat piecemeal way in a fragmented economy (Hornsby, 1992), and didn't mind them one way or another. However, the Acadians couldn't speak Gaelic and were not likely to merge well with Gaelic culture. Both groups frowned upon out-marriage. The Acadians

were against it because it meant both breaking the bonds of family and friends that they had worked so hard to maintain after The Expulsion and marrying an English-speaker in most instances. For the Scots this was so because it meant breaking those same bonds that were transferred *en masse* from the Old World and were so important to keep their new land intact and within the clan (Molloy, 1986; Hornsby, 1992:143-146). These characterizations are supported by the paucity of out-marriages observed anecdotally in the marriage records that have not been transcribed for this project.

The clan system is probably the most familiar way historians characterize the organization of the Scots. To explain it briefly: it was a patriarchal system of social organization that consisted of many tiers – not unlike those of the Mi'kmaq chiefdoms. A clan leader, or chieftain, was an inherited title through the male line. Chieftains were responsible for the partitioning of land and alliances and agreements, or wars, with other clans. Sole rights over land and possessions were entrusted to the chieftain in theory, though practice was likely often a different matter if disagreements with decisions could be backed up with force. Fealty could be called upon by the chieftain in times of war, though it was not as organized as a feudal system. The clan system is an ancient one, reaching back into the Celtic tribes of Gaul in modern-day France, Belgium, Holland and Spain and it is likely that the Scots (and Irish) had inherited this tradition from their Gaulish forebears. In the 19th century in Cape Breton, the clan system was likely still remembered in theory. The infamous old rivalries, for example, between the Campbells and the M^{ac}Donalds, still strained relations between families. This strain likely became exacerbated when new land was at stake.

We have, then, a situation arising on the island where two large ethnic groups are not integrating due to the course of history and ethnic differences that are based on similar values and circumstances. Instead of mixing as creoles had in other parts of the world, who found themselves in a similar situation oppressed by a common enemy (the English), the Scots and Acadians seemed to have maintained separate identities throughout the 19th century. Hornsby (1992) doesn't comment on this factor in creating the fragmented economy of nineteenth-century Cape Breton but I suggest that it was an important one that has been missed by previous writers. There was little reason for these two established groups to interact beyond their combined economic involvement with one another. There were, in fact, several reasons to remain separate. There were mixed marriages between the groups, that is inevitable, and there are a few examples of this in the Chéticamp parish records. However, it was likely something that was frowned upon by both groups and not a common occurrence. This seems to be the underlying current in Hornsby's (1992) message and in Molloy (1986).

The situation in Cape Breton Island in the late 19th century was one with a fragmented economy; a self-segregated group of Acadian and Highland Scots settlers mixed in with minor bands of Loyalists and Irish immigrants, and a settlement pattern that supported few urban centers of any great density, with many dispersed communities of rural settlers in between. The rural landscape and geology of the island allowed only three occupations for the majority of the population: farming, fishing and coal mining. A few minor off-shoots of these three were much less common, though not necessarily less important, to the economy. These included shipbuilding, shipping (mainly rum, sugar and

fruits) and some dairy processing. The trading triangle between Chéticamp, Îles-de-la-Madeleine and Newfoundland was a strong one in the 19th century (Charlie Dan Roach, personal communication) and local consumption became an important aspect of the economy. Some ports like Chéticamp and Sydney had become successful at tapping a world economy but were not well-integrated with each other.

The population density that one is exposed to on a regular basis can range from quite low to quite high, depending on the nature of the occupation. Fishing, mining and shipbuilding tend to favor higher densities. The work involves several people at one time or another in the course of a day and workers are in contact with many of their co-workers on a daily basis. Farming, on the other hand, tends to create lower densities where people may only see their immediate family members for days at a time. Many infectious diseases are reliant upon levels of exposure to other potential hosts in order to propagate. Therefore, occupation is a focus of this project as a major contributing factor in determining the “success” of a microorganism to cause an epidemic and to sustain one. This area of discussion will become a key one in Chapters 6, 8 and 9.

As discussed earlier, religion played a key role in the development of the early history of the island with competition played out between the Acadian-Mi'kmaq allies and New Englanders. The weekly mass was a regular social event for most Acadians and had largely gone uninterrupted since their return to the Island in the late 18th century. This aspect of social life was likely missing for a long time in the lives of the first Highland Scots immigrants. As communities became more established in the mid 19th century, many Roman Catholic families among the Scots embraced the various

Evangelical Presbyterian movements as they were more suited to the lifestyle of the farmer and his kin. This did not provide the same exposure level to other members of one's in-group among the Scots as it did among the Acadians, especially not in the first half of the 19th century. The main reason for this was the approach of these Presbyterian movements. Worship revolved around the schedule of the farmer, unlike the cycles of the Catholic year as dictated by Rome. Weekly religious services could acceptably be conducted at neighbors' houses instead of at a church, especially if the activities of the farmer, such as the harvest season, made such an option easier. This created a less formal, less crowded worship experience for many of those Scots choosing to convert to these types of Presbyterianism.

Why is it important to have drawn these distinctions about social structure and population density? Essentially this dissertation is based on an approach of disease ecology and demography. In order to understand the movement of the scarlet fever epidemic of 1875-1877 across the Cape Breton landscape, we must first understand that landscape and its own interactions. The landscape of human disease is of course human beings, so understanding how that landscape is likely to change and interact during the colonization of a disease on that landscape is essential. At the present time, it seems likely that the dichotomy between Scots immigrants and Acadians of the 19th century in Cape Breton is one that created different patterns of disease interaction. We must turn now to examine the specific biology of the microorganism responsible for this disruption, *Streptococcus pyogenes*.

Chapter 3: The Biology of Streptococcal Infections

Scarlet fever, scarlatina or *la scarlatine*, in French, is one of several manifestations of *Streptococcus pyogenes*. It was responsible for the deaths of tens of thousands of children worldwide during the great scarlet fever pandemic of 1820-1880. Today, it is a rarely seen manifestation and it is one of the goals of this project to understand why that is. It is important first to outline the biology of streptococcal infections before proceeding with an analysis of a specific epidemic. A detailed description of the specific biology of *S. pyogenes* and the interactions that take place during their infection of a human host will be considered. This description is framed in the larger context of bacterial biology. It is only from here that a discussion of the specific manifestation of streptococcal infections known as scarlet fever can be made.

3.1 *Streptococcus Pyogenes*: A Brief Introduction to its Biology and Symptoms of the Infections it Causes

Streptococcus is a genus of Gram-positive bacteria. The suffix, “coccus” refers to the spherical shape of the bacteria, and Gram-positive refers to the fact that the bacteria react when presented with the Gram stain. This reaction is due to the cell wall lacking a thick outer membrane, which, when found in other bacteria, gives added protection to the cell. As we will see, *Streptococci* have their own unique methods of protection once inside the human host. They are non-motile, meaning they do not have the power of spontaneous movement. Their means of respiration is considered to be aerobic to

facultatively anaerobic, meaning they are typically air-breathing but can survive for periods of time without air. Their community organization is such that they occur in pairs or short or long chains (Chin, 2000)

Streptococcus pyogenes is a specific taxon of the genus *Streptococcus*. However, a finer level of distinction is possible. The types of toxins each organism produces merits an alphanumeric label. So far, these include groups A, B, C, G, H, J, K and M *Streptococci*, though the most common are A, B and C (Knöll, et al., 1991). Group A is the most ubiquitous, of which the type species is *S. pyogenes* (Pugh, 2000). Group B is responsible for neonatal sepsis and is restricted to this specific illness. This project deals with Group A *Streptococci*, which is frequently shortened to the acronym “GAS” in the literature (Chin, 2000)

Transmission of this bacterium is achieved through tactile means or by limited airborne capabilities in that it can be found in large respiratory droplets (Chin, 2000). Its reservoir is mainly in humans but it can survive in a dormant stage in some foodstuffs, especially those that are stored in particulate dust-like forms such as flour, cornmeal, etc., as well as in milk. Milk-borne scarlet fever was a massive health risk to children in the 19th century before pasteurization was standardized (Wilson, 1986)

The incubation period is short — rarely longer than one to three days. The period of communicability is between 10 and 21 days, though the bacteria can survive in a dormant state in the naso-pharyngeal cavities of the carrier for several weeks or months. The number and therefore degree of contagiousness of the bacteria in this carrier state is greatly reduced two to three weeks after the onset of symptoms (Chin, 2000).

Scarlet fever itself is usually only fatal to those aged 2-15 but it can kill adults on occasion. Manifestations of the disease most commonly include strep throat, and streptococcal skin infections (impetigo and pyoderma). The less common manifestations include scarlet fever, puerperal fever, septicemia, erysipelas, cellulitis, mastoiditis, otitis media, pneumonia, peritonsillitis and wound infections. Even more rare are manifestations of necrotizing fasciitis (also known as flesh-eating disease), rheumatic fever and toxic shock-like syndrome (Chin, 2000; Pugh, 2000).

The variability of manifestations is due to several factors. One of these is the multitude of toxins and problems that the bacteria create upon establishing a population in a host (for an extensive list, see Pugh, 2000:1707). The most important of the toxins that the bacteria produce is an erythrogenic toxin, meaning that it produces an eruption of red blood cells that can result in a rash or, more commonly, in sore throat. The initial characterization of the toxins produced by *Streptococci* was accomplished by Dick and Dick (1924). The toxin is usually what the alphanumeric labels are named after, though non-GAS can produce GAS manifestations; e.g., in addition to exotoxin A, scarlet fever has been associated with pyrogenic exotoxins B and C. Streptococcal toxins cause a generalized erythematous (red) rash with fever and a sore throat (Chin, 2000). The toxins are a low molecular weight protein that are produced with hyaluronic acid as the carrier medium (Pugh, 2000; Schwartz et al. 1990).

The toxin protein consists of two parts: the first part, which is heat labile, meaning it can be chemically altered or destroyed by heat, is responsible for the primary toxicity — pyrogenicity (causing fever), low lethality, cytotoxic (cell damaging/destroying)

effects on cultured spleen macrophages (these are involved in the production of antibodies and in cell-mediated immune responses) and suppression of the reticuloendothelial and immune systems. The second part is heat stable and is responsible for secondary toxicity — hypersensitivity effects including skin hyper-reactivity, myocardial necrosis (heart muscle tissue death), enhancement of pyrogenicity (fever) and lethality, and enhanced host response to any other injurious or pathogenic agents (Pugh, 2000)

Both of these parts also combine to give a toxin whose effects cannot be assessed unless the immunological state of the host is considered. In other words, the specific biology and state of the host at the time of infection contribute to a multitude of responses within the body of the host itself. Therefore, combined with the number of toxic effects, the result is different manifestations of the infection. Each of these seems different to those who are not familiar with the pathogen and, in the 19th century, they were probably considered to be different diseases entirely, based on differing sets of symptoms. This topic is covered in more detail in Chapter 4.

Bacteria in general and *S. pyogenes* in particular use several other mechanisms to survive medical treatments we have devised in the 20th century. Bacteria divide by means of a process known as binary fission. While normal cell division is accomplished through the process of mitosis, binary fission is a shorter and, hence, quicker process. This is because the entire bacterial genome is usually contained on a single chromosome consisting of a circular DNA molecule and its proteins. There is no pulling apart of chromosomes, as is necessary in mitosis. This translates into a rapid rate of reproduction

in bacterial cells with a nearly continuous synthesis of DNA (Campbell, 1996).

A rapid rate of reproduction is important because it means that genetic change and adaptation have the potential to occur at a much higher rate as well. The rate of bacterial reproduction can be compared to that of human reproduction in order to demonstrate the amount of change that is possible at this rate. It takes the average bacterial cell one to three hours to create a new generation, though some can double every 20 minutes under optimal conditions. A liberal estimate of human generation times is about 15 years. Since the time of *Homo erectus*, 1.8 million years or 120,000 generations have passed. It would take the average bacterial colony only about 27.4 years to achieve this same number of generations.

A final important feature of bacterial biology is the process of transformation. Bacterial cells have been well-documented, since the early part of the 20th century, as having the ability to incorporate useful pieces of DNA into their own genome and transmit these acquired genes to offspring (Campbell, 1996). A parent cell literally acquires a trait through exposure to the DNA strand carrying the trait and incorporates that trait into its own genome.

What this potential for rapid change and the process of transformation imply is that bacteria can, and have, kept up with any natural changes that have occurred in our immune responses to them as well as any synthesized antibiotic treatments we have devised. This is so not only because of their rapid response, but also because they have at their disposal a unique mechanism, through transformation, to “share” useful adaptations with unrelated cells in the colony or host. This is of course not an intentional

manipulation of the cell's environment, but it is a new way that bacteria have been able to “cooperate” unintentionally to ensure the survival of the fittest members of their population. The combination of these two features of bacterial biology continues to make it extremely difficult to create novel ways to combat them in the human host when they are pathogenic.

A finer level of distinction can be made of *S. pyogenes* aside from the alpha-groups. Within the species of *S. pyogenes*, over 130 serotypes have been identified within North America and Europe alone (Beres et al., 2002). Serotyping is a process of classifying organisms or cells, similar to the sub-species level, based upon their surface antigens or proteins (Pugh, 2000). In *S. pyogenes*, the serologic types are based on the presence of a protein on the outer membrane of the bacterium. The fact that one species has over 130 serotypes is indicative of how long this genus of bacteria has been evolving and changing to suit its niche in the human organism. Its greatest strength seems to lie in this variability. Perks and Mayon-White (1983) suggested that the serotypes travel in groups, that is to say that the serotypes can be found in mixed proportion in any one host at any one time occupying the same niche and acting in different ways. They found that serogroups other than the primary infecting one were found in scarlet fever patients, and in addition, other infectious agents that cause similar symptoms were found. These included staphylococcal infections, herpes, influenza and rhinovirus, among others.

This phenomenon of serogroups traveling in mixed populations may be a strategy that has allowed the bacteria to be so successful. If more than one serogroup can exist within a stable population of infecting serogroups, this can “distract” the immune systems

of the hosts and cause multiple problems in the body. This would cause the immune system to divide its resources among several problems caused by several infecting serogroups instead of the immune system being able to concentrate on only one. This is sort of like a “blitzkrieg” tactic of bombarding the immune system with several serogroups at once. The real benefit comes from the fact that each serogroup is unique and has to be dealt with individually. Additionally, because many of the symptoms caused by the toxins weaken several organ systems and create inflammations in various mucous membranes of the body, other non-streptococcal infecting agents can benefit from the presence of *S. pyogenes* creating even more confusion for the immune system of the host.

Most of the serogroups of *S. pyogenes* are similar in basic structure and are classified as a type of the M protein. This is a protein attached to the outer membrane of the cell and is referred to as a surface protein. The initial work on identifying differences in serotypes was published by Griffith and Gunn (1928) and classifying this protein was accomplished by Lancefield (1928a, 1928b, 1928c, 1928d). Each serotype gets a numeric designation, e.g., M1, M2, M3, and so on, creating an alphanumeric label for each serotype. All of the M-proteins share a basic underlying structure but have different binding sites. These can be thought of as different locks made by the same company but requiring different keys. These create the different serotypes. There are also T proteins that are much less common but also contribute to the over 130 serotypes. In addition to this system, M⁻ and M⁺ phenotypes apparently exist as well (Fischetti, 1989).

The protein is well-adapted to the human immune response. It requires T-cells

from the immune system to recognize each specific serotype. Through the creation of antibodies, an individual only gains immunity to that M- or T-specific serotype, rather than all group-A streptococcal classes. Additionally, the toxins are seemingly good at distracting the immune system from the invading bacteria by forcing the T-cells to deal with the tissue and cell damage that the toxins cause (Fischetti, 1989; Pugh, 2000).

More recently, as genetics and molecular biology have improved in examining the microscopic structure of surface proteins, types within serotypes have been found. These are numbered sequentially, e.g., MGAS11, MGAS12, MGAS13, and so on. The reason for this further distinction is that there are unique features of the strains that can spell the difference between a virulent strain and a non-virulent one. For instance, Beres et al. (2002) have recently been able to identify bacteriophage-like elements that have been incorporated into the DNA of some MGAS3 strains. What this sequence of DNA produces is an extra toxin that resembles the effects of snake venom toxins. Martin and Høiby (1990) have also shown that strains that have been previously serotyped can increase in virulence and change character, likely through the transformation process.

As members of a population, *S. pyogenes* bacteria change in their distribution of types, as any population is wont to do. The representation of each serotype in a global or even geographically isolated population of *S. pyogenes* organisms changes over time due to the natural fluctuations of their individual numbers (Moses et al. 2002; Schwartz et al. 1990). This is a reflection of their successes and failures in colonizing hosts and being reproductively successful. Reproductive success does not necessarily mean increased virulence in bacteria. It is usually quite the contrary. Some of the most reproductively

successful microorganisms are those that are able to inhabit their hosts while *not* killing them before being passed on to the next host. However, some that have multiple reservoirs, such as cholera and malaria, can be quite virulent and kill large numbers of people because killing one type of host will not eliminate their reservoir. Schwartz et al. (1990) and Moses et al. (2002) have shown that changes in human epidemiology occur as a consequence of changes in representation of serotypes. This makes sense if we return to the point of the relatively slow adaptations that our own immune system is equipped with in comparison to the speed of bacterial shifts; as the illness-causing bacteria are fought off, through natural or engineered means, the representation of other serotypes increases. Meanwhile, the decreased population of the first strain is left with only the hardiest members alive, which are then able to “regroup” and possibly adapt to the means of defense that thwarted their growth efforts in the first place.

This suggests the possibility that the method by which GAS infections have remained a problem in humans is constant cycling of the many serotypes of *S. pyogenes*. Quinn (1989), in a 150-year longitudinal study of records from several locations around the world, found results that suggest this very phenomenon of cycling for rheumatic fever, streptococcal disease and scarlet fever. His work also confirms the marked decline from the mid 19th century through at least the 1980s in both incidence and severity of scarlet fever.

In a nine-year study by Duben et al. (1978), carriers were shown to have fewer disease-causing serotypes than patients. This suggests one of two things may be going on within the population of *S. pyogenes*. The first possibility is that the more benign

serotypes occur in large numbers but have lower virulence and are not being attacked by the immune system or treatments. The second possibility is that carriers represent those who have survived a previous bout with the disease and this previous serotype was different than those now infecting patients in the follow-up study nine years later. It should be pointed out again that many of the symptoms of the disease are a result of the immune response in the host. This second possibility would, again, support the idea of the cycling of strains. It is also worthwhile to note that in the study by Duben et al. (1978) an average of 11% of individuals was found to be carrying *Streptococci*.

The long-term effects of GAS infections can vary depending on the particular manifestation experienced by the patient. Obviously, something as severe as necrotizing fasciitis, which causes permanent tissue death and often loss of a limb or parts of limbs, will have major long-term effects on the patient. Scarlet fever is known to cause damage to the myocardial tissues and brain tissues as a result of the exotoxins that are released during infection and high fevers. Westlund and Schulz (1972) looked specifically at hypertension-associated causes of death in patients who had previously been diagnosed with scarlet fever or diphtheria. Their work concluded that no significant difference was recorded for those patients who had experienced scarlet fever or diphtheria earlier in life compared to a control group who had neither of these illnesses.

Treatment of a GAS infection has traditionally been accomplished with penicillin and more recently with erythromycin and clindamycin. However, Stevens (1994) suggests that more recent manifestations of the disease, such as necrotizing fasciitis, have shown large numbers of *Streptococci* in the host. The sheer increase in the number of

bacteria has rendered these traditional treatments slow and even completely ineffective at worst. Resistance to treatments is facilitated by common practices related to antibiotic use. Antibiotics are often prescribed inappropriately, either because they are not effective against a certain type of bacteria, or they are given as a method to “do something” for the patient when they are actually suffering from a virus. When they are appropriately prescribed, patients often do not follow through with the complete regimen. They often stop taking the medication once they feel better. Both of these problems have the effect of eliminating the weak members of the bacterial population in their bodies and promoting the representation of those rare few bacterial cells that have acquired immunity to the treatment. There has also been a recent change in the stationary growth phase of infecting *S. pyogenes*. Stevens (1994:564) suggests that penicillin’s failure as a treatment is likely linked to a reduction in the expression of critical penicillin-binding proteins. It is clear that the adaptive response time of the organism is going to continue to be a problem for medicine in the future.

3.2 The Implications of Scarlet Fever as a Manifestation of *Streptococcus pyogenes*

The combination of the high degree of variability in the infective agent and the complex biology of the human host generates the immense amount of variability within the biology of the infecting agent itself. Combining this with the biology of the host gives even more possible effects that can result due to co-infection and other genetic, dietary and overall health-related factors. The human immune system is not equipped with many options when combating an infection. Likewise, our interconnected organ systems react

with a limited set of symptoms to immune responses and the effects of various toxins in the body.

The typical and diagnostic symptom of scarlet fever is a scarlet rash. Chin (2000) describes it in the following way:

The rash is usually a fine erythema (*redness*), commonly punctate (*marked by points or dots*), blanching on pressure, often felt (like sandpaper) better than seen and appearing most often on the neck, chest, in folds of the axilla, elbow, groin, and on inner surfaces of the thighs (Chin, 2000:471, my additions in *italics*).

The manifestation also includes those symptoms commonly associated with streptococcal sore throat: sudden onset of fever, sore throat, swollen tonsils, and enlarged lymph nodes. Some patients may also experience enanthem (mucous membrane eruption), strawberry tongue (swelling and redness of the tongue so that it resembles a strawberry's outer skin in color and texture) and exanthem (skin eruption) (Chin, 2000).

Scarlet fever must be conceptualized not as a disease itself but as one possible manifestation of a disease. It is a response of the human body to a toxin that disrupts homeostasis on many levels. One of the questions raised by this project is why scarlet fever is so rarely found as a manifestation of GAS infection today while it was so much more common in the historical literature and documentation. The biological reason may be a simple one. All of those members of the human population who were overly sensitive to the toxins that produced the symptoms of scarlet fever may have gradually been removed from the population through death. Their sensitivity may have been due to genetics, health-related behaviors, or some combination of the two. Regardless, we may have seen a simple case of selection against those who were not adapted to survive

infection. More correctly put, those who died from the symptoms caused by their level of sensitivity to the toxins may not have survived to produce offspring of their own.

This is a difficult argument to prove conclusively, however. Several changes can occur in the biology of the infecting agent itself, such as natural cycles. We may be seeing a period of “adjustment” of the organism. Antibiotic treatment may have caught the natural cycles of the population of the organism on a down-cycle, perhaps as it was beginning to rise again. Only an extensive, longitudinal study of streptococcal epidemics will help us to understand the full picture. This would require a biochemical study of the mechanisms in the human response to the toxins. Identifying a genetic link to these mechanisms that could be shown to be shifting over time (possibly through ancient DNA of known victims) would probably be required as well in order to establish an argument for natural selection in humans.

Whatever the reason, it is clear from the symptoms of the manifestation of scarlet fever that it is easily differentiated by the characteristic rash that gives it its name. However, this does not necessarily mean that all cases of death caused by scarlet fever, or *S. pyogenes* in general, were recorded correctly in Cape Breton Island. Certainly, 19th century medicine had its own set of problems, but the nature of those problems is not much different in kind than the problems we still have today. Only the mind-set and knowledge used to approach these problems was different. It is sufficient to point out for now that today, even with our advanced scientific methods and tools of investigation, we don't always agree on the standards to classify a cause of death or even morbidity from one case to the next.

Chapter 4: Medicine in the 19th Century

The 19th century may be considered an extension of the Enlightenment period for medicine and medical science. This was an era of discovery and realization, and of the forming of medical theory that established the necessity of specialized medical professionals in the growing urban sprawl of Europe and the Americas. Those long-standing, disconnected and vague notions of the causes of infectious disease, such as filth, poverty and class, were finally solidified into a tangible theory of microorganismic causes known broadly as “germ theory”. With the perfection of the microscope in the 19th century, the theory of infective agents became a reality as microorganisms were directly observed and classified. Though subsequent advancement in treatments was found in the emerging metropolises of the 19th century, the rural countryside of all nations, especially that of Canada, was late in achieving the benefits that such advancement would bring. In the case of Cape Breton Island, this lagging behind the status quo of the day was certainly true in the 19th century and is still true today.

4.1 Science and Medicine in the 19th Century

The dawn of the 19th century saw Europe emerge out of the wake of the French Revolution and into a new era of nationhood. National characters were being solidified by the two superpowers of the English and the French as well as their respective continental neighbors. It is sometimes hard to imagine the many interconnected issues that frame the picture of science and medicine, as theorized and practiced in the 19th

century. These include philosophy, economics, social theory, tradition, nationhood and identity, among others, and perhaps most importantly, the industrial revolution itself. In order to understand the situation in Cape Breton Island, it is necessary to briefly examine these interconnected issues and how they shaped the thinking of a nineteenth-century Cape Bretoner.

England in 1800 found itself at the beginning of one of the largest population expansionary phases it has ever experienced. The class system was very well-defined and everyone had unmistakable knowledge of their place in society. The poor and rich lived worlds apart from one another. This theme impacted everything about life in 19th century England, from education to social responsibility to welfare to population dynamics. All facets of life could be directly correlated to one's social class. The only problem was that people of the 19th century were just beginning to reconcile their differences in order to implement health policies to resolve them (Ogle, 1999; Schultz and M^cShane, 1978).

The impact of class structure on medicine came indirectly through the educational system and directly through society's general perception of doctors. In England, the educational system had long been built on the idea that if it was worth studying, it was taught at either Oxford or Cambridge. At the turn of the century, both had medical faculties but their numbers were low compared to the other professions. The medical profession as it was known was lumped with the clergy and law as one of the three original professions taught in universities. The internal division of medicine could be split into physicians, surgeons and apothecaries. The education of medical professionals at Oxbridge still emphasized the teachings of Galen and Hippocrates into the 1790s.

Graduates of Oxford and Cambridge were also given exclusive “rights” to become members of the Royal College of Physicians of London. Graduates of very few other institutions were given this privilege, and even if allowed, it was a position without voting power within the Royal College (Bynum, 1996).

In the late 18th century an emphasis on blood and the nervous system began to emerge (Bynum, 1996). The philosophy of 19th century medicine became one of purging the body of its ills. Most often this took the form of forcing the body to release various fluids. Vomiting could be induced with purgatives derived from plants. Likewise laxatives could be used to induce diarrhea. Sweat could be released through heating the body. However, the most common practice perhaps was blood-letting. A lancet could be used to create a flow of blood from one of the arms or a specialized scalpel could be used to incise the forehead. Special instruments for bleeding and containers for gathering the blood were quite common in the tool-kit of the 19th century doctor. Many even specialized in purging the body of specific fluids.

Scots medical institutions came into the fore at the start of the 19th century. The main ones were centered in the larger cities of Edinburgh and Glasgow. These featured more open admissions, lower tuition costs, and eventually a more rounded education, even compared to the Oxbridge schools. One of the reasons for this was the Scots emphasis on giving their physicians practical training in anatomy. By the 1790s private schools that also emphasized anatomy and surgery began to open in the London area (Bynum, 1996). Surgeons by this time had acquired independence from barbers, with whom they were grouped for many decades. They became more numerous in the London

private schools than the other two branches of medical training. Surgery was still considered an apprenticed craft learned at the side of a “master” surgeon. Surgeons’ day-to-day duties largely consisted of blood-letting and forms of phlebotomy as opposed to the complex tasks of today’s surgeons. Due to the philosophy of medicine and disease of the 19th century, blood-letting lent itself well as a treatment to most ailments and surgeons were deemed very essential (Numbers, 1987).

Across the channel, France had a much larger number of established schools. The French Revolution opened the doors of admission to more individuals. In some cases, the doors opened too wide and degrees could be obtained rather easily and without much practical training. Those who could afford to were even able to obtain degrees *in absentia* (Numbers, 1987). However, the French were also a bureaucratic culture which required certificates and regulations in a very strict way. The French approach did feature an early tradition of the teaching hospital where real patients were attended by students being educated by their professors at the bedside of the patient. As such, the practical training of physicians and surgeons was emphasized in France.

Apothecaries played a somewhat different role in the formation of medicine in the 19th century. France had allowed them some freedom to diagnose and treat illnesses shortly after the Revolution. However, they were soon restricted to being strictly pharmacists. In England, the apothecary was quite a different professional. He was not only allowed to write prescriptions and fill them himself, but he also usually dabbled in what we may today recognize as a sort of general practice. The English apothecaries were more like shopkeepers in some places, house-calling doctors in others (Bynum, 1996).

Regardless, the common role of apothecaries throughout Europe was that they were allotted the task of preparing remedies for all sorts of medical conditions. Ultimately, in many areas the apothecary became as wealthy as a successful merchant (Numbers, 1987).

The young United States of the 19th century took its cues mainly from England, the mother country of many of its first inhabitants. The University of Pennsylvania and King's College (later Columbia University) both established medical faculties of their own, in 1765 and 1767 respectively. This led to a fairly large body of American-trained medical professionals by the 19th century. Unfortunately, due to the expansion of the U.S. at this time and the vastness of its pioneering territories, there was little regulation of the quality of medical training. As a result, a large number of charlatans and quacks with their own home remedies traveled or turned into side-show traveling salesmen. In reaction to the erosion of American medical credibility, many legitimate doctors supplemented their education with European training in France, England, Scotland or Germany (Bynum, 1996).

Post-Revolutionary France was founded on the ideas of independence, individuality and self-liberty cemented in the country's motto "*Liberté, Égalité, Fraternité*" (Liberty, Equality, Fraternity). This theme resonated in the young United States, which was newly freed from English rule, and the two shared an approach of strong individual rights for some time. However, the diversity in approaches to education between England and France, and even in the results of the lack of quality control in the U.S., coupled with the emphasis on individualism of the day, led to greater differences of opinion within the international medical community. The ideals of the age promoted each

man to think for himself, and this was applied in medicine too.

The classification of diseases, known as nosology, became a matter of observation of symptoms. The timing, degree, and order of symptoms was crucial to classifying a disease correctly. This idea was best demonstrated by the 18th century English physician William Cullen, who was influential in establishing a set of criteria and descriptions of symptoms as well as an extensive nosological scheme developed around observation of symptoms. Cullen viewed the body as a whole entity, not as a mechanical interaction of separate systems. For Cullen symptoms were the expression of imbalances in this holistic body (Bynum, 1996). Cullen had not encountered the move away from Classicism that the late 18th century ushered in.

The theory of evolution had not even begun to emerge in science and this added to problems of classification that evolutionary theory helped sort out in both biology and medicine. The idea that non-human life forms could change and become better at infecting humans was an idea that wasn't even considered logical until well after the late 19th century, when Darwin's ideas began to be promoted. Therefore, there was virtually no scholarly framework that allowed medical professionals of this time to even consider the existence of microorganisms or the forces of evolution that caused all organisms to evolve and change. Catholic priests, especially those in rural areas like Cape Breton, were probably unaware and certainly not accepting of evolutionary theory and were probably more inclined to view disease as a moral failing than a biological interaction. Stories about witchcraft and the devil associated with illness are still present in the memories of the elders of at least the Acadian community (Planetta, 1980) and this indicates the

attachment of issues of morality to illness. Metropolitan physicians of the day conceptualized biology in a different way and their approaches reflected this.

The mind-set of the 19th century still held to Enlightenment ideas of allegory and symbolism attributed to unseen forces in nature (Porter, 1995; Planetta, 1980). The attributes of a disease or a moral characteristic were symbolically transformed into tangible details through the conjuring of images of monsters, witches, or other personified and vilified creatures. Any force of nature could be classified along a spectrum running from good to evil, with its position determining the form of that force. For instance, evil forces might have taken the form of a devil or demon while very good forces might have taken the form of angels. This mind-set is described by Geyer-Kordesch (1995) in her examination of Enlightenment attitudes toward disease. Until the direct observation of pathogens could be achieved, symbolism, allegory and the Western system of the four humors combined to explain disease.

The Enlightenment produced a sort of bridge between the ancient and the modern in terms of approaches to medicine. The idea of *sensibility* was born in the Enlightenment and combined social class, etiquette, morality and bodily health into an approach to life in general. For some medical professionals of the time the concept of sensibility presented a theoretical framework in which an excess of emotion and nervousness could be attributed as the source that sent the body into imbalance and disease. For others, the concept of sensibility was the inspiration for good mental and moral health that would lead to good bodily health (Vila, 1998). The bridge that the Enlightenment formed between the ancient and the modern is embodied in and symbolized by the bridge that the concept of

sensibility formed between morality and science. Sensibility characterized an approach not only to medicine but to all manners of inquiry so that morality and philosophy pervaded subjects we would now perceive as wholly mechanistic or biological in nature.

A large part of what hampered the progress of 19th century science and medicine was the struggle between religion and science. This struggle is not to be mistaken, as it still often is today, as one of determining which approach to knowledge is correct. Rather, it is a reconciliation of two different ways of knowing about the universe. Until this century, religion in Europe had consistently taught that those things that we could know and that were knowable had all been revealed to us through our senses as they were created by God. Anything that could not be easily explained or known was a mystery and relegated to the realm of godly knowledge, unknowable by humanity. The 19th century saw the acceleration of a shift in this thinking towards secular, natural explanations of observed phenomena.

What grew out of this shift in applying direct observation and study was the idea that the work of scholars in other fields could also prove valuable. This wasn't a new idea, of course. France and England both had good examples within London and Paris of guilds and societies trying to keep intact the standards of the profession, or rather their monopoly on a growing industry (Numbers, 1987). However, the idea of the individual practitioner setting his own standards of medicine through his version of approaches and schools of thought started to become less valued. If there was to be true progress in the study of medicine, medical professionals had to be scientists, and direct observation, independent testing and re-testing were all a necessary part of scientific discovery. By the

late 19th century, a man with a medical degree could no longer concoct his own version of approaches pieced together from his own experiences and studies. In order to conduct true science he had to be open to the ideas and experiments of others and consider all of the evidence, supportive or not, relating to his ideas (Cravens et al., 1996).

The shift in understanding the universe was a critical turning point for medicine. The religious and moral reasons for disease and illness that had pigeon-holed the philosophical approach of medical professionals of the past could now be ignored. This was not an overnight process, but it was becoming acceptable to believe that the direct reason for specific ailments in specific individuals was not God's will but was the action of specific pathogens on the human body. Darwin and Wallace (Porter and Graham, 1993) demonstrated the ultimate mechanism for understanding how it was possible for this relationship between disease and humanity to continue to exist, and how it had, in fact, existed since time immemorial. It started to become clear that medicine wasn't just about the human body or spirit, it had to consider many other organisms and how their biology interacted with the human host to cause disease and death.

In summary, the atmosphere of medicine in Western Europe and North America at the time of the scarlet fever epidemic of 1875-1877 was one of flux. The classical ideas of balance that re-emerged in the preceding Enlightenment period were being rejected in favor of ideas that concentrated on specific systems in the body as the cause of disease. Purging the body of these ills became specific goals of physicians and many favored a philosophy that centered on one particular fluid of the body, such as the blood or sweat. Though scientific knowledge was on the verge of the technological advances offered by

the microscope and the theoretical advances offered by the theory of evolution, neither had yet come to the fore. Vague theories of causes of diseases relating to the specific systems of the body were pieced together by physicians, now separated from their pharmaceutical colleagues by a divide of professions. This fed further disagreement on procedures for treatments and a variety of individualized approaches, both sound and unsound, were promoted.

4.2 Determining Causes of Death in the Historic Record

If we consider the origin of cause of death information, it is likely that an inadequately trained individual was responsible for determining the cause of death in many places. A priest or an educated fellow citizen could hardly pass as a sufficient source of knowledge on the many illnesses of the day. However, what seems more important is the lack of medical knowledge, by modern standards, that medical professionals had at the time. Technology that we take for granted today was completely lacking in the late nineteenth century in most locations. The lack of powerful microscopes was a major reason for the problem: without direct observation of microorganisms it was not possible to identify the underlying cause of many infectious diseases. Diseases that manifest in many different ways, such as *Streptococcus pyogenes*, were classified by symptoms into different diseases. There could also be no reporting of observed microorganismic forms occurring in consort with a specific list of symptoms because of a widespread lack of microscopes. Though treatments could still be hypothesized and administered based on symptoms, true scientific classification could

not proceed, in this instance, without the widespread use of technology.

Once specific pathogens could be identified, as *Streptococcus pyogenes* was in 1883, better understanding of the pathogen became possible. Medical scientists determined that symptoms not only varied from patient to patient but also that they could be so vastly different in children and adults that at least two diseases previously recognized and thought to be distinct were actually caused by the same organism. The problem for medical practitioners of the 19th century was that they could not conceive of a system of pathogens and causes or reactions to those pathogens because they did not even have a scholarly framework within which to consider such things. The previous systems of medical knowledge had ill prepared them for the world of microorganisms. Untrained registrars recording cause of death information had even less direction.

What we are left with is a sense that the researcher of historic documents must be vigilant when approaching cause of death information. First-hand information has usually passed through several layers of interpretation to get to the researcher in the 21st century. As discussed more extensively in Chapter 7, the information is usually vague and unrepresentative by strict statistical standards, not only because of the lack of medical training in the recorders but also because of a lack of medical knowledge of the era in general. Nonetheless, this does not render the information useless. In fact, Anderton and Leonard (2004) suggest that cause of death information of the late 19th century in their Massachusetts sample had become quite useful. Qualifiers that had been included with the cause of death information helped to elaborate on the specific circumstances surrounding the cause of death. Considerations of the data available in the Cape Breton

sample can be made to compensate somewhat for the lack of accuracy. For instance, scarlet fever proper has specific and unique symptoms, especially in the scarlet rash produced by the disease. This is clearly discernable by the uneducated eye and so specific cases listed as “scarlatina”, or variants thereof, can be considered quite accurate.

Additional consideration must be given to more general causes of death around the time of the peak of the epidemic, however. These include notations of fevers, rashes, ailments of the throat, and inflammations of the heart and brain tissues that would otherwise seem unrelated. The reason for this is fairly simple; as discussed in Chapter 3, scarlet fever is only one manifestation of an infection of *S. pyogenes*, which has many other symptoms associated with it. Therefore, although the basic recording of “scarlet fever” or “scarlatina” can be considered accurate, the presence of other manifestations must be kept in mind and sought in the historic record.

The information from both a literal interpretation of the records and a more liberal interpretation need to be considered for this project. The nature of scarlet fever demands that both be done in order to include all possible faces of the epidemic. Though the true numbers of scarlet fever victims are difficult to extract, this project at least seeks to consider the possible scenarios that could have resulted in the historic record we are left to interpret.

4.3 Cape Breton’s Medical Situation in 1875-1877

Since the health of the people of Cape Breton Island in the 19th century is the general area of interest for this project, a brief summary of the medical situation on the

island at this time is necessary. The 1871 Census of Canada summary tables (Canada Dominion Bureau of Statistics, 1876) show a total of 23 physicians and surgeons in all of Cape Breton Island. The numbers break down to nine in Cape Breton County, seven in Inverness County, six in Richmond County and one in Victoria County. The populations, doctors and ratios for each county are given in Table 4.1 below. This table clearly illustrates the truly sparse number of medical professionals at the time in Cape Breton Island. By comparison, the ratio of physicians to the population of the United States in 1988 was 23.31 doctors per 10,000 people (Filion 1989a) and in Canada it was 21.98 per 10,000 people for the same year (Filion 1989b). Individuals practicing other occupations related to the medical profession include two medical students, one each in Cape Breton and Inverness counties, one chemist/druggist (pharmacist) in Cape Breton County and one hospital attendant in Victoria County.

Table 4.1. Number of physicians and surgeons in Cape Breton Island in 1871.

County	Population	No. of Doctors	Doctors/10,000
Cape Breton	25,454	9	3.54
Inverness	23,415	7	2.99
Richmond	14,268	6	4.21
Victoria	11,346	1	0.88
Total CB Island	74,483	23	3.09

The two major populated centers in Cape Breton in the late 19th century were the areas surrounding Sydney (Cape Breton County) and Arichat (Richmond County). A map of the island with the counties and major cities is provided in Figure 4.1 below. Sydney and Arichat were the focus of most medical activity on the island and were likely the two

places responsible for the 15 doctors between Cape Breton and Richmond Counties. Similarly-sized town centers could be found in the Port Hood and Mabou areas and are likely the places where the seven Inverness doctors were located. Victoria County was not completely rural at the time. Baddeck and Boularderie were both similar in population size to Sydney or Arichat. However, only one doctor was in the area at the time of the 1871 census and he was likely in Baddeck, the largest city of the county.

M^{ac}Millan (2001) emphasizes the vast territories these doctors had to cover. Having been a rural or country doctor himself, M^{ac}Millan could provide first-hand insights into the trials and tribulations of covering the area around Baddeck in the early to mid-twentieth century. The area was about 80 miles long by 30 miles wide. Despite the existence of telephones by this time, automobiles were still too expensive for many Cape Bretoners, and M^{ac}Millan relates that he was obliged to use horse and carriage into the 1950s. Canada did not have a system of socialized medicine until well into the 20th century, which meant that patients of the 19th century had to pay for treatments out of their own pockets. There were no insurance plans and no government agencies that compensated people for illnesses or treatment. Each doctor had to be paid directly by his patients. The depressed economic situation left many of the rural areas of Cape Breton Island no doubt void of medical practitioners, largely because no one could afford to see one, and hence, the practitioners could not make a living in the region.

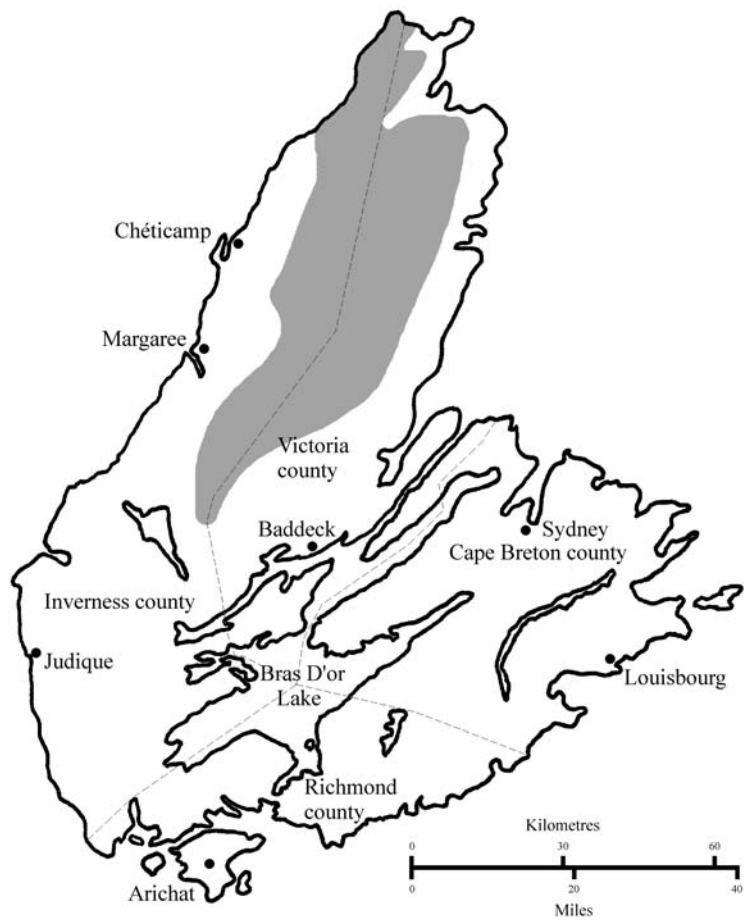


Figure 4.1. Map of Cape Breton Island showing the counties and major towns.

This situation was not new to Canada. Gelfand (1984, 1992) noted that New France of the 18th century also had a very sparse distribution of medical professionals trying to service as many people as possible over large distances. The medical landscape of New France was supplemented by the religious orders that emphasized medical knowledge. The Jesuits especially were known for their skills in pharmacy and many sisterly orders served as nurses as they still do today.

The first European settlers of Cape Breton, and likely those of the 19th century, had created a folk medicine derived from their own roots in Europe as well as their encounters with the Mi'kmaq. Knowledge of local plants and other remedies found in nature would have to be derived from this latter source by the Acadians when they arrived, for no doctors came with them. Gelfand (1992) suggests that native remedies were embraced by the first colonists of New France. However, he warns that these likely had no impact on medical theory of the time. This is likely because a folk medicine framework already existed and the new remedies could simply be “stitched into” this framework.

It is easy to imagine from census summary reports and personal memoirs the great difficulties involved in getting medical attention in the late 19th century. If a doctor did not live within a day's travel, the patient was likely not going to receive any treatment from a trained professional. Furthermore, a patient could not contact a doctor if they were without telephone or telegraph service and certainly this was a luxury in the late 19th century. In fact, the telephone was only patented by Bell in 1876. (Coincidentally his summer home and research laboratory was at Baddeck.) However, most 19th century Cape Bretoners could not afford the luxury of the telephone and a doctor's attention was only gained by sending messengers to the doctor, usually by horse.

This lack of true medical professionals placed the burden of care on the families of the ill and the clergy. The families would of course be able to apply their own traditional remedies handed down from generation to generation. It was the role of the clergy of both the Catholic and Presbyterian faiths to heal the souls as well as the bodies

of their congregation. Many, in fact, had some medical training and were at least familiar with the basics of medical care at the time. For those close to their faith, the dual role of priest and healer was likely a comforting one. However, this dual role could, and probably did, become muddied with moralistic approaches to illness that remained popular into the 19th century (Bynum, 1996; Porter, 1995).

Chapter 5: Previous Studies of Scarlet Fever Epidemics

Between 1825 and 1885 the world suffered a pandemic of scarlet fever outbreaks (Katz and Morens, 1992). During this 60 year span, every nation that was recording official cause of death information had at least one example of the pandemic's effect. Many of the studies that have been conducted in the past on this specific pandemic are centered around Great Britain and the US (Duncan *et al.*, 1996; Eyler, 1986; Eyler, 1987; Greenwood, 1976; Katz and Morens, 1992; Scott and Duncan, 1998), though some have taken a wider scope in both geography and time (Quinn, 1989).

Studying epidemics from the pandemic period in locations other than Cape Breton is essential to understanding how the Cape Breton epidemic fits into the global experience. However, we must also look at streptococcal epidemics throughout history, including contemporary studies. Modern epidemics may be skewed by the fact that antibiotic treatment is available, but there are features relating to the experiences of the sufferers and the general population that are informative for all epidemics. For this purpose a literature review of both the pandemic period and other epidemics in history is presented here.

5.1 The Scarlet Fever Pandemic of 1825-1885

Great Britain, the United States, Germany, France, Norway, Russia, Sweden, Denmark, New Zealand and Australia have all been the subjects of studies related to the pattern of the pandemic that killed hundreds of thousands, if not millions of children in

the 19th century. The beginning of the pandemic is not well-marked, considering the nature of reporting cause of death information. This is true of locations in Nova Scotia as well as around the world. Katz and Morens (1992) suggest that earlier records from Europe indicate that the disease was relatively “benign” with several sporadic outbreaks listed in specific countries or cities. The problem they note is the lack of differentiation of the disease from cases of diphtheria, measles and other similar diseases. There is fairly good evidence from France to suggest that there was a dearth of scarlet fever cases in the latter half of the 18th century and beginning of the 19th century until Bretonneau reported on a severe epidemic in 1824 (Katz and Morens, 1992). The first severe epidemic to arise in Great Britain was ten years later when the case-fatality rates jumped from 1-3% to 15%. The first solid report from the US is seen in New York City in 1828 when a severe epidemic caused a ten-fold jump in the mortality rate (Katz and Morens, 1992). Pennsylvania also experienced a severe epidemic in 1839, where the cases are reported to have killed patients in less than 24 hours (Greenwood, 1976).

Globally, the period surrounding the 1875-1877 epidemic in Cape Breton was one of decline for the large epidemics. Deaths per 100,000 started to fall after the 1875 epidemic peaks in both London and Boston (Katz and Morens, 1992; Scott and Duncan, 1998) and the following years of the pandemic petered out to much smaller epidemics in these two cities. Moreover, the epidemic peak in 1875 was small compared to preceding epidemics during the pandemic period since it resulted in only half the number of deaths per 100,000. Considering the nature of scarlet fever and *S. pyogenes*, which would thrive in areas of high population density, it is reasonable to use these two large urban centers in

two of the most populous western nations of the time as a proxy for the global situation. The waning of epidemics shown in the data on London and Boston suggest that the epidemic in Cape Breton was one of the last large ones to hit the area. However, without the existence of recorded cause of death information from 1878 until resumption of mandatory recording of vital events in 1908, we cannot be certain if this was the last part of the pandemic to hit Cape Breton Island.

The decline in severity of scarlet fever did not occur everywhere at the same rate. Katz and Morens (1992) indicate that during a Russian epidemic between 1892 and 1915 the case fatality rate was 22.3%, while England and Wales during the same period were experiencing a rate of 3.3%. China also had a significant epidemic in 1921-1922 that killed between 50,000 and 200,000 in the city of Kunming (Yunnanfu). Many of the victims of this epidemic experienced a very rapid onset of the disease, leading to death within 24 hours. Katz and Morens (1992) suggest that although there are currently still severe cases in developing countries, the severity of the mid-19th century pandemic has not been matched. It is suggested here, and supported by Katz and Morens (1992), that this difference in global waning of the pandemic is a direct or indirect result of the respective rates of modernization. Modernization may in fact be specifically interpreted as sanitation practices. These would include standards of food cleanliness, municipal sanitation via waste and water treatment, and also personal sanitation in the form of hand washing and a general awareness of sanitary behaviors like sufficient bathing that began to pervade the 19th century mind-set. Given the largely non-modern, rural setting of the majority of Cape Breton at this time, the island may have been a reservoir for

perpetuating the last peaks of the mid-nineteenth century pandemic.

5.2 Scarlet Fever and General Streptococcal Epidemics in the Literature

It is important to review the literature on scarlet fever and related streptococcal epidemics throughout history, including contemporary outbreaks. This will help to define the characteristics of past epidemics and give a good indication of the likely circumstances that surround the course of any streptococcal epidemic. This section is not meant to provide a comprehensive literature review of scarlet fever and streptococcal epidemics because there are hundreds of these cited in the literature. However, a few good, representative articles on major changes and/or trends in the history of scarlet fever and general streptococcal infections have been selected for examination. A good source for a more comprehensive review can be found in Quinn (1989).

One of the earliest studies on epidemics of scarlet fever was that of Gunn and Griffith (1928), who were contemporaries of Dick and Dick, inventors of the Dick skin test for sensitivity to scarlet fever, and Lancefield, describer of the antigenic complex of *S. pyogenes* (Dick and Dick, 1924; Lancefield, 1928a; Lancefield, 1928b; Lancefield, 1928c; Lancefield, 1928d). All were integral pioneers in research on classification and identification of the molecular mechanics of *S. pyogenes*. Gunn and Griffith's (1928) study comprised 100 cases of scarlet fever in a hospital setting in Park Hospital, London, England, during 1926 and 1927. The patients were followed from admission to discharge. The goal of the study was to differentiate reactions or symptoms of patients according to different serological types of *S. pyogenes*. These symptoms and reactions included

temperature, pulse-rate, incidence of faucial angina (sore throat), vomiting and early adenitis (inflammation of lymph nodes or glands), and a series of other complications including otitis media (inflammation of the middle ear), mastoiditis (inflammation of the mastoid process), rhinorrhea (discharge from the nose), late and suppurative adenitis (late and pus-forming sore throat), nephritis (inflammation of the kidneys), rheumatism (inaccurate term relating to pain in the musculoskeletal system), endocarditis (inflammation of the inner heart tissues), jaundice, relapse, desquamation (peeling of the soles and palms), and death.

Gunn and Griffith's (1928) study quite clearly established the multiple faces of *S. pyogenes* infections. It really wasn't until the early 20th century that medical professionals realized that one infectious agent could result in a number of different symptoms due to its effects on the immune system through the toxins it produces. By the time of this study, only four serotypes and one heterogeneous type had been classified (Gunn and Griffith, 1928). These were large type categories based upon agglutination tests and the exact nature of each serotype and their effects were still being studied at the time of this early work. The authors noted that there were differences even among these four identified types in the severity and number of complications associated with them. Gunn and Griffith (1928) also noted that scarlet fever itself seemed to be more consistently produced by the less toxic, though more abundant, serological types, rather than the more damaging, less common ones. The authors suggested that the symptoms of scarlet fever were more often associated with milder toxins and that these persisted for a longer period of time. Perhaps this is a mechanism for the disease to proliferate in a wider population while the

more toxic serotypes are restricted in spread but deadlier in their effects.

It is worth remarking that although antibiotic therapy was not mentioned for the hospitalized patients in this study (and was not available at that time), none of the patients died. This corroborates the claim by Katz and Morens (1992) that the virulence of the disease has been greatly reduced since the mid-19th century pandemic. It is also revealing to examine the testing of the patients at weekly intervals for serological types; many of the 100 cases during this testing showed infections with multiple types during the course of their illness. Some of these occurred simultaneously, others occurred at different times during their hospitalization. In fact, all patients reported had two or more serotypes present during the course of their stay. Some would remain with no serotypes present for weeks at a time, while others would have a new type in the following week. This exemplifies the ubiquity of multiple serotypes of strep in the human reservoir.

This infection with multiple serotypes suggests a couple of possible situations. One may be that because of the early stage of serotyping at this time in history, several additional stages of serotypes were not being recorded. We know that there are currently over 130 different serotypes in North America and Europe alone (Beres *et al.*, 2002). An additional possibility is that multiple serotypes of the disease travel together, infecting a host as a group, creating slightly different types of toxins to overwork, distract and confuse the immune system in order to ensure their mutual survival. A third possibility is that serotypes are mutating through acquisition of characteristics from neighboring serotypes co-present in the host and forming other recognized and unrecognized serotypes. These possible situations are not mutually exclusive.

Duben *et al.* (1978) conducted a longitudinal study using a larger sample. The study was conducted from October 1966 to September 1975 for a full nine year period in one of the six health districts of the city of Havlíčkův Brod in what is now the Czech Republic. The sample comprised 4,000 individuals representative of the 100,000 inhabitants of the city and surrounding area. Each patient presented with clinically definite or suspected streptococcal throat or skin infections and was examined for the presence of streptococci.

What is most striking about this study is the much higher percentage of cases where streptococci are present for those reporting tonsillopharyngitis (sore back of throat and tonsillar area) compared to those reporting nasopharyngitis (sore nasal passages and palate area) and laryngitis (sore larynx and loss of voice). An average of 43% of the tonsillopharyngitis cases over the entire study period had streptococci present compared to 4.8% and 6.5% for nasopharyngitis and laryngitis respectively. According to Beres *et al.* (1978), this is in agreement with earlier findings from other authors. For the purposes of my study, this suggests that a large percentage of people for whom “sore throat” was listed as a cause of death in the historic record may very likely have died of a streptococcal infection. In addition, this study provides an even more interesting statistic for the purposes of modeling. A sample of the larger population showed that a range of 6.2% to 19.3% were carriers of streptococci, with an average of 11% providing a stable reservoir. This suggests a very stable reservoir for the proliferation of the species through epidemics.

M- and T-typing of the streptococci found in the study patients was also performed. These tests discern the nature of the surface proteins on the bacteria so they can be classified under a serotype designation. The results of this part of the study showed that certain serotypes are more abundant during certain years but also that certain serotypes are always more common than others over the entire nine year period. The study also showed that certain serotypes were responsible for producing the symptoms of scarlet fever while other serotypes did not, as also suggested by Gunn and Griffith (1928). This further strengthens the notion that the extent of scarlet fever epidemics as suggested by a literal reading of the historic record is only the tip of the iceberg; the challenge of studying the epidemics using the historic record is not so straightforward.

A more extensive longitudinal study examined the mortality of patients who had scarlet fever or diphtheria 30-50 years before time of death in Oslo, Norway (Westlund and Schulz, 1972). The study was specifically aimed at examining the likelihood of scarlet fever or diphtheria contributing to complications later in life. This study concentrated on those whose cause of death information could be categorized as hypertension-associated because it had been suggested that previous infections of streptococcal diseases might cause permanent cardiovascular and/or renal damage that theoretically could contribute to complications in these systems later in life (Hewitt, 1968; Westlund and Schulz, 1972). Hypertension-associated causes and other causes included in this study were hypertensive disease, cerebrovascular accidents, coronary heart disease, sudden death, hypertensive disease, valvular disease, other cardiovascular diseases and chronic nephritis, as well as all malignant tumors and all other causes. The

study samples for scarlet fever and diphtheria numbered 985 and 724 respectively.

The authors found no correlation between having had either disease in youth and dying of hypertensive-associated causes. In fact, the numbers are so close for the observed and expected that there seems to be no underlying untoward effects of having had a bout of streptococcal disease at all. This runs counter to the evidence that there can be damage to the cardiovascular and renal systems from both of these infections. However, it may be the case that both systems recover sufficiently for the effects of the damage to be unimportant later in life. There is also the possibility that death before the 30-50 year follow-up study confounded the investigation of this study. In other words, hypertensive-related problems may show up earlier in life in such individuals and kill them before the typical time of life when such complications cause death. This is critical in understanding historic records studies of scarlet fever mortality because this suggests that the effects on generations that lose members to an epidemic are visible in the immediate mortality records. Future follow-up analysis of the mortality rates or ages of age-mates of the same cohort later in life is likely not going to differ markedly because of their morbidity from scarlet fever or other streptococcal complications.

Another study from Norway was conducted using the infectious disease notification system covering the entire country of 4.1 million people (Martin and Høiby, 1990). An increase in GAS (group A streptococcus) reporting was seen in late 1987 and early 1988 and this trend was analyzed in detail by the authors. There are some disturbing findings noted in the study. The first is that the disease notification system of the country, while good, is still insufficient for communicating noticeable changes in disease reporting

to the public. Though the epidemic began to be noticed in the early winter of 1987-1988, the first press report of this trend was not released until March 1988.

Another aspect of concern was the increase in the number of older children and young adults reported with GAS infections. Reporting in these age groups had been unseen in previous decades. As stated in Chapter 3, streptococcal infections do not usually manifest in severe form in adults. The presence of the disease across the age spectrum suggests that for this epidemic, the population had little to no previous experience with the serotypes. Or, they may have never had experience with the potency or type of toxins being produced by a familiar serotype.

The most consistent and exceptional point to note in this study was the predominance of a single serotype, M-1. Martin and Høiby (1990) report that previous studies had shown the representation of one specific serotype reached a ceiling of 20% in cases during an epidemic. The 1987-1988 epidemic showed remarkable figures ranging between 41% and 87% representation of the M-1 serotype in the counties of Norway. This serotype seems to have been quite virulent as well, causing more pronounced symptoms and affecting multiple organs. This is noted as being consistent with past behavior of the M-1 serotype (Martin and Høiby, 1990; Gaworzewska and Colman, 1988; Cruickshank *et al.*, 1981).

A great deal of study has gone into the phenomenon of disease cycling. In the field of demography, this study is dominated by the work of Susan Scott and Christopher Duncan. Their recent volume on time-series analysis brings to light some interesting studies of correlations between vital events and such seemingly unrelated phenomena as

weather, temperature and the price of wheat (Scott and Duncan, 1998). Through a complex set of operations, raw data are filtered to reveal oscillations or cycles. Leslie matrices are used to remove irrelevant information or noise in the cycles and a smoothed line is produced. The smoothed line visually and mathematically shows the oscillations in a way that is easier to read and easier to compare through regression analysis. The analyses compare whether a rise or peak in one set of data leads to a rise or peak in the other set of data. The amplitude, or height, of the peaks and valleys can also be compared but this can be misleading as data sets that are out of phase with each other by one or more cycles can be displaying effects from many years before while still in sync with the overall cycles. This lag can be corrected, though, making cross-correlative record analysis a very useful and productive way to generate data and understand a complex problem such as infectious diseases.

Scott and Duncan's (1998) chapter on infectious diseases in the 19th century reveals some interesting correlations for scarlet fever. Their analysis suggests that there is a correlation with three factors — low summer rainfall, low spring rainfall and wheat prices. They explain the correlations with low rainfall as likely being due to a direct effect of low humidity on the state of the nasopharyngeal mucosa. Waddy (1952, cited in Scott and Duncan (1998)) demonstrated the effects of dry air on the nasopharynx. This older study suggested that in the study areas of Ghana and England and Wales, such air dried the mucosa, causing the infectivity of respiratory diseases to benefit from the mucosa's loss of ability to resist infection.

There are a couple of problems with this suggestion by Scott and Duncan (1998) and Waddy (1952). The first is that streptococcal infections are not generally spread through respiratory tracts because they are not typically airborne. The route of infection is almost always tactile, though the bacteria can be found in respiratory droplets, likely because they inhabit the nasopharyngeal mucosa once inside the body. The second problem is that increased moisture in the nasopharyngeal mucosa, not decreased, facilitates the capture of airborne droplets. However, a dried mucosa would be less resistant once infective agents were in contact with it.

My alternative suggestion to this correlation with low spring and summer rainfall, and thus ambient humidity, comes primarily from the historic records. The detailed records for Norway (Martin and Høiby, 1990) and the Czech Republic (Duben *et al.*, 1978) both show that there is some seasonality to scarlet fever and that it generally kills in the winter months. This is out of phase with the suggestion that infection is occurring in spring and summer because mortality from scarlet fever is not the outcome of a lengthy infection, but rather a very rapid one, often under 48 hours. I suggest that what must be examined is the very important role of the carrier state in this disease. Recall from Chapter 3 that streptococcal infections can stay dormant in the nasopharyngeal cavities for weeks or months. This is a crucial part of the disease cycle on the grand scale because it is those who carry the dormant members of the streptococcal population, adults, who are maintaining a reservoir in humans. The implication of this is that infections in adults reflect their own childhood experience with GAS infections. Therefore, it is suggested here that the ambient humidity in spring and summer months

may be causing irritation of the nasopharyngeal mucosa in carriers, activating the dormant bacteria. These then resume a period of activation over the summer and fall months in the carriers and eventually spread to the children causing scarlet fever morbidity and mortality.

The correlation found with wheat prices of the time is explained in more detail because it is a more complex theory to justify. The data on each (the mortality and the wheat prices) are drawn from two separate sources (Creighton, 1894; Stratton, 1970). The graphs show a good match to the epidemic peaks, mirroring their occurrence, though lagging two to three years at a time (Scott and Duncan, 1998). High wheat prices are correlated with high scarlet fever mortality two to three years later. Scott and Duncan (1998) suggest that high wheat prices are causing higher levels of malnutrition in mothers during pregnancy. This sets up a malnutrition cycle in the unborn child and affects its overall resistance to all kinds of diseases. Although an interesting idea, few sources are cited to support this claim.

In order to show that this hypothesis is reasonable, one must establish that the majority of deaths in *all* of these epidemics were in two to three-year-old children. While this is a possible scenario, it surely cannot account for the entire lag of the system in question. This is a problem because the two to three year lag used in this system suggests that, in order for the impact of higher wheat prices to affect pregnant mothers two to three years ahead of the epidemic peaks, this age group of children must comprise the majority of deaths because they are the ones experiencing the nutritional strains *in utero*. Scott and Duncan cite a source from the 19th century (Creighton, 1894) claiming that 2/3 of all

deaths in England and Wales from scarlet fever in the last half of the 19th century were from children under five years of age. The specifics of this interval are not included and raw data are not cited to support this claim. Studies of other historic epidemics reviewed here do not support this specific age distribution.

A second problem with Scott and Duncan's claim about the correlation of wheat prices to mortality levels is that it assumes that all children who died were from families who could not afford to provide adequate nutrition to pregnant mothers. While this may be true in many of the cases, there are no supporting data mentioned to back this claim. An economic evaluation of families who lost children during this epidemic is required if one is to make this argument. Correlation between children dying between the ages of two to three and low economic status must be shown on a case-by-case basis to establish this factor as being a relevant one. An increase in the price of wheat does not necessarily mean it was no longer affordable. The rest of the economic system or at least a comparison to other commodities or the GNP of the country has to be taken into consideration. Finally, at the end of their discussion on the correlation of wheat prices to scarlet fever mortality, Scott and Duncan (1998) invoke the seasonal dryness explanation to account for the susceptibility in children in the age group of two to five years.

Consideration of the specifics of the disease biology again results in an easier way to explain this phenomenon of the two to three year lag that is apparent in the data . If we examine Figures 14.5A and 14.5B of Scott and Duncan (reproduced below in Figure 5.1), we see that the lows in wheat prices are actually very nicely correlated with highs in scarlet fever mortality. I suggest that a simpler explanation than the one they offer is

possible. Recall from Chapter 3 that streptococcal species find a livable reservoir in foodstuffs, especially milk and those stored in particulate, dust-like forms such as flour. I suggest here that the low wheat prices are representative of an abundance of wheat on the market. This abundance of wheat, being processed and stored in the form of flour, provided an increased reservoir for the streptococcal bacteria. There is no need to evoke a second-hand, time-lagged explanation. There is a direct causal agent possible in the abundant flour acting as a reservoir. Nonetheless, a single line of evidence should never be invoked as a sufficient explanation for such a complex set of relationships as exists with a disease like scarlet fever.

Swedlund and Donta (2003) studied the spread of an 1859-1860 scarlet fever epidemic in Deerfield, Massachusetts. Through family reconstitution and historic maps, the authors are able to identify individual houses where children died of scarlet fever. The aim of the study is to demonstrate that epidemics such as scarlet fever are complex and to emphasize the necessity of taking nutritional and socioeconomic data into consideration. It uses a microepidemiological approach by focusing in on a few communities that experienced the epidemic that occurred within the context of a regional epidemic and global pandemic.

The study also recognizes the importance of each of the components of molecular biology, historicity, medical history and epidemiology. Their findings contradict the work of Scott and Duncan (1998) showing that higher socioeconomic status was associated with as many as half of the epidemic victims. Malnutrition and under-nutrition were not known to be associated with these populations.

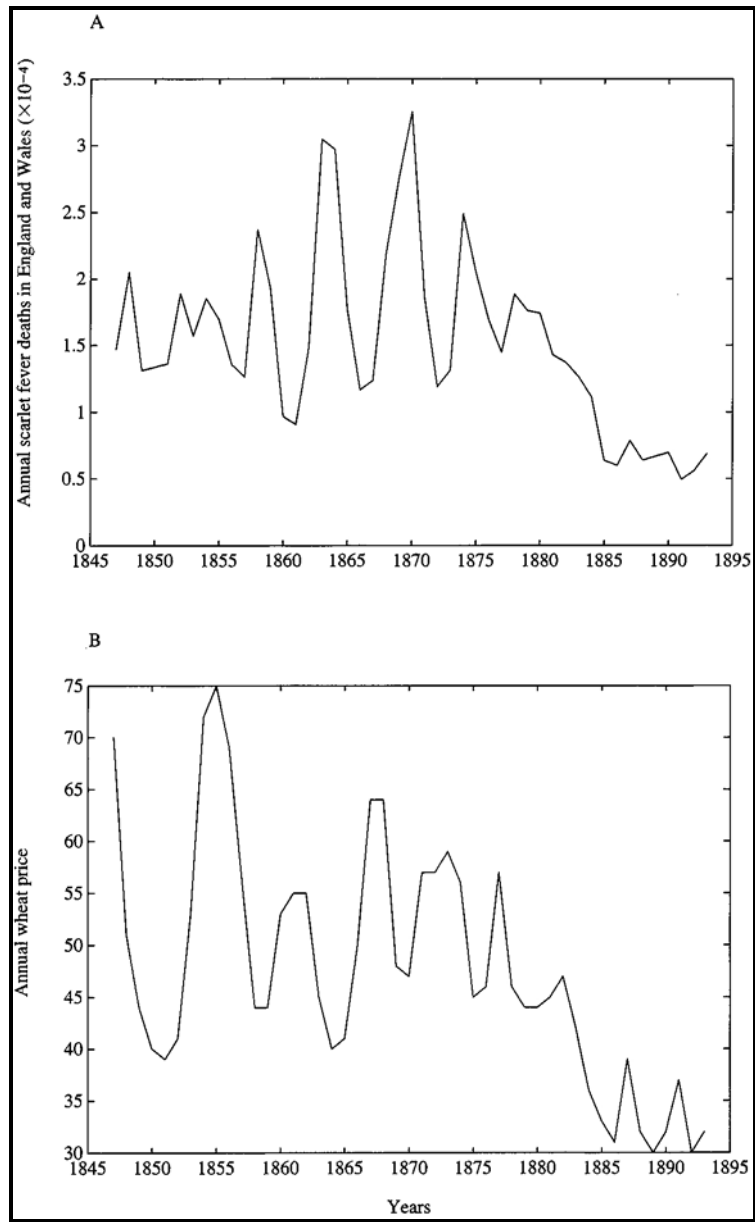


Figure 5.1. (A) Annual scarlet fever deaths in England and Wales, 1849-1893 (ordinate). (B) National wheat prices (shillings; ordinate). Marked falling trend in both series after 1875. Data sources: Creighton (1894) and Stratton (1970) (reproduced from Scott and Duncan 1998:312, Figure 14.5, with permission of publisher)

There is some concentration on treatments in the study that are derived from historic popular literature of the time. Whether or not such guides to home remedies were

being followed is in question. The exact balance of home remedies to popular literature advice may never be known but it is worthwhile to have these sources available for documentation of the latter. My one point of divergence with this study is the lack of consideration for other manifestations of streptococcal infection, but this is not an easy thing to do. Their general approach, especially in putting a personal “face” on the epidemic experience in this community is one that I eventually hope to emulate with the Cape Breton population.

The nature of an historic streptococcal epidemic can be difficult to characterize and the state of modern ones can present new problems. Schwartz *et al.* (1990) performed an extensive study on the changing nature of GAS infections in the USA. This study examined over 5,000 strains sent for typing to the CDC between 1972 and 1988. The representation of M- and T-typable serotypes in the population was analyzed to see if changes in representation might explain increased reporting of invasive infections and severity of those infections. The authors found that certain serotypes had seen a significant increase in representation in the data from 1972 to 1988. These were mainly serotypes M-1, 3 and 18. This supports the idea that changing frequencies of serotypes in the population of GAS bacteria can cause an increased number and severity of infections in humans.

The authors suggest that the increase in number and severity of infections is due to lack of previous exposure to the serotypes. Once a person is infected with a certain serotype and successfully fights the infection off, that person develops immunity for life to that serotype due to the development of antibodies against it. This suggests that the

correlation of increased number and severity of infections with increased representation of certain serotypes in the GAS bacteria population represents a general lack or decrease in previous exposure to those over-represented serotypes. M-1 is noted in other studies as being particularly well-known for causing severe infection (Cruickshank *et al.*, 1981; Gaworzewska and Colman, 1988; Martin and Høiby, 1990). This study also echoes the trend that Martin and Høiby (1990) established for Norway. For the purposes of historic records research on infectious diseases, this study suggests a general means of change to explain waxing or waning epidemic rates. However, it does not provide the specific mechanism for historic epidemics since that cannot be known unless morbidity and cytological information were available at the time.

With a much smaller sample, an earlier study showed a different trend for the Oxford region of England (Perks and Mayon-White, 1983). Throat swabs of suspected scarlet fever cases were taken so that serotyping of any viral and bacterial pathogens could be undertaken. All patients presented with rashes and the majority (71%) also had sore throat. Of the 105 patients with suspected scarlet fever only 25 were confirmed to have *S. pyogenes* on their throat swabs. Among them, M-4 was the commonest serotype. Interestingly, this serotype is specifically noted as being in decline in the study by Schwartz *et al.* (1990).

Other features of the study support previous thoughts on the disease. *S. pyogenes* was isolated from a significantly larger number of patients that had the full set of symptoms of scarlet fever than those who did not. These features included the characteristic scarlet rash, fever, sore throat, lymphadenopathy (pain in the lymph glands

of the throat) and absence of coryza (runny nose). This suggests that any difference in these features was not likely to support clinical evidence for a streptococcal infection. Also, two of the 71 controls were found to have *S. pyogenes*. This represents only 2.8% compared with an average of 11% in an urban sample from Czech Republic in the previous decade (Duben *et al.*, 1978). Interestingly, none of the cases over the age of 15 presented with symptoms characteristic of scarlet fever and only one of them had a positive swab for *S. pyogenes*. This supports the earlier claims that the disease generally does not affect older adolescents and adults.

What is of note in this study is that a clinical diagnosis of scarlet fever could not always be found for those presenting with the full set of symptoms of the disease. This again reminds the historic records researcher that one must be cautious of the cause of death information listed. Because symptoms were the only means of characterizing a cause of death, the historic records researcher can only be as accurate as the medical knowledge of the informant.

By the early 1990s, it was clear that streptococcal infections had, once again, become a major health concern and that epidemics of severe infection had been reported in North America, Europe and Australia (Stevens, 1994). Stevens (1994) examined the changing features of GAS infections, concentrating on the severe infections of the present day, including necrotizing fasciitis (colloquially known as “flesh-eating disease”), streptococcal myositis, malignant scarlet fever, bacteremia, and streptococcal toxic shock syndrome. What is most troubling about these newer manifestations of streptococcal infections is that they are typically killing otherwise healthy groups, such as young and

middle-aged adults.

Necrotizing fasciitis is an infection of the subcutaneous tissues causing the death of the outermost tissue layers (fascia) under the skin and the fatty layers, but it often leaves the skin itself intact. Streptococcal myositis is the phrase used to describe severe inflammation of the muscles presenting with a GAS infection. The symptoms usually include a great deal of pain, swelling and erythema (redness due to capillary expansion) in the area, and they are quite severe. Often cases will present simultaneously with streptococcal myositis and necrotizing fasciitis. Case fatality rates for myositis range between 80 and 100%, while those for necrotizing fasciitis are between 20 and 50%. Malignant scarlet fever is used to describe cases where extremely high fever, severe sore throat and cervical (neck) lymph nodes, severe headache, delirium, and convulsions are present. Interestingly, there is usually little if any characteristic scarlet rash and death often occurs within 24 hours. Sepsis (poisoning by means of toxins in the blood or tissues) may also be an accompanying symptom, though it is given a separate category within the severe cases. Bacteremia is simply the presence of viable bacteria in the blood vessels. Usually invasive infections are restricted to single organ types or tissue types. Bacteremia complicates a GAS infection by causing its spread to other systems in the body than those to which it is usually confined; typically this is the pharyngeal cavity and surrounding organs. The skin, sinuses, peritonsillar tissues and mastoids have all been identified as viable targets for this spread. Finally, streptococcal toxic shock syndrome is the phrase used to describe the association of GAS infections, usually in the form of sepsis or bacteremia, with shock. Soft tissue infection, acute respiratory distress

syndrome (ARDS) and renal failure are all common symptoms of this manifestation.

Stevens (1994) explains the mechanisms for infection, shock, and tissue destruction very clearly. Once again, M-types 1 and 3 are implicated most frequently as the leading serotypes responsible for causing severe manifestations of the disease. Stevens suggests that not only is there a mechanism in the immune system to recognize the serotype as M-1, M-3, etc., but there is also a mechanism that must recognize the streptococcal pyrogenic exotoxin (SPE) type. Recall that the toxin types are given alpha-letter designations such as SPEA, SPEB, SPEC, etc. SPEA and/or SPEB have been found most often in cases of severe infections. The mechanism that recognizes the serotype can help the person completely avoid the infection if that person has been previously infected with the same serotype. It will alert the immune system that a recognized foreign body is present and the antibodies that fought it before will be summoned again. However, the toxins produced now seem to be showing variants of their own so that no two SPEAs are necessarily the same.

This study illustrates the great mutability of *S. pyogenes*. It has seemingly been able to create more destructive results of infections throughout history. These manifestations are varied and grouping all of them together is only possible when clinical diagnosis of the presence of the bacteria can be established. This suggests that different manifestations of the disease were probably also present in the past as well but were unrecognized as emanating from the same source. The ability of the disease to manifest in various ways has more than likely been accelerated with the increase in antibiotic therapy, namely penicillin. However, this species of bacteria clearly has the ability to

change its disease-causing features rapidly and effectively. For the historic records researcher, this supports the earlier idea put forth that a wide interpretation must be considered to account for all possible deaths from streptococcal infections, not just one of its manifestations (such as scarlet fever). Any analysis of historic epidemics should allow for both a liberal and a conservative estimate of the number of deaths caused by this pathogen.

A recent example of studies on the current state of GAS infections highlights the effects of demographic factors, such as religious orthodoxy, on shaping an epidemic course (Moses *et al.*, 2002). This study shows that the M-1 serotype is very rare (1.2%) in a sample of 409 GAS cases from Israel. The serotypes that were present in high proportions included M-3 (25%), M-28 (10%) and M-non-typable (33%). A mortality rate of 5% for the sample points out the severity of these infections even today. During pre-antibiotic “normal” periods where no or rare epidemics of scarlet fever occurred, lower numbers of between 1% and 3% were reported.

The severity of GAS infections is clearly getting worse. Associated illnesses that were found in the Israeli sample included primary bacteremia (14%) and soft-tissue infection (63%). Amongst the patients, 30% were otherwise healthy individuals and not in a category of compromised immunity or a more commonly affected age group such as the young and the elderly. Orthodox Jews showed the highest annual incidence of GAS infections per 100,000.

In an examination of contacts, throat swabs were used to test 302 family members of 60 index cases for presence of GAS in their pharyngeal cavities. 87% of the family

members presented with the same serotypes as the index cases. This suggests a carrier rate of over four times the number of clinically diagnosed cases presenting with symptoms. No mention is made of follow-up investigation of these family members for later presentation with symptoms of illness.

This study suggests that religious orthodoxy contributes to increased incidence of infections for the community. Though the authors do not elaborate on this finding, I would interpret this as support for the belief that the features of religious orthodoxy may contribute to the higher rates of infection. Social segregation, higher rates of inbreeding, frequent and consistent religious gathering in groups and dietary taboos all likely contribute to creating both a high contact rate and lower average immunity to infectious diseases amongst this segment of the population. For the present study, these factors of religious segregation will become important in the explanation of the 1875-1877 scarlet fever epidemic of Cape Breton.

In a genetic approach to the problem of increasing severity in GAS infections, a longitudinal study compared genetic markers for the gene responsible for encoding streptococcal pyrogenic exotoxins (*speA*) (Musser *et al.*, 1993). The study examined cases in Ottawa, Ontario, Canada in the 1940s and Eastern Germany in the early 1960s to early 1990s. While the sample is not large (24 isolates from Canada, 15 from Eastern Germany) some interesting facts have come to light in this study.

The first is that different alleles of the *speA* gene are responsible for causing variants in the infecting toxins; the alleles known so far are given numeric designates, *e.g.*, *speA1*, *speA2* and *speA3*. Clones of *S. pyogenes* are also identified through

electrophoresis. These may be thought of as variants within the serotypes themselves so that M-3 may have two or more clones with different designations and different capabilities for producing SPEA or SPEB toxins. The second finding of the study showed that two, three and sometimes four clones of *S. pyogenes* were simultaneously causing infections and disease. The authors note, however, that one clone always dominated in proportion to the others. The study concludes that it is likely that allelic variation in the *speA* gene is a sufficient catalyst for causing new disease epidemics. This is important to the study of epidemics in history because it suggests the importance of contact. The more contact a group has internally, the more often they will be able to quickly circulate all the variant types. However, this also means that they are more likely to suffer new epidemics with more severity than other groups.

The extreme of infection in even the youngest of patients has been independently confirmed for GAS infections (Del Castillo *et al.*, 2000). In this recent case from 2000, an 8-week-old infant was diagnosed with GAS pharyngitis and scarlatiniform rash. The child was otherwise normal clinically and behaviorally and, interestingly, non-febrile (without fever). She was prescribed a course of antibiotics and was subsequently healthy on follow-up. It is interesting that an infant this young can contract the disease, even show mild forms of the symptoms, and yet not be otherwise affected. It is unknown how the disease might have progressed if left alone but the literature on mortality from streptococcal epidemics presented here suggests that death would not have been the outcome anyway.

Finally, in a bizarre case, one study has shown that an anal-tactile route of infection is possible with this disease (Richman *et al.*, 1977). Four cases of scarlet fever and GAS surgical infection emerged closely spaced in time in a single hospital in Boston in 1976. Following normal procedures, rectal and throat cultures were taken of all staff associated with the patients. The route of infection was traced to the anus of a surgeon who had operated on three of the four patients earlier.

Hopefully, these studies have elucidated the variable findings in the etiology, history and characteristics of streptococcal and scarlet fever epidemics in the literature. There are helpful clues from each as to how to approach the study of the epidemic in Cape Breton in 1875-1877. A large literature on individual outbreaks that have happened recently is not covered here. However, in order to gain some general knowledge, more comprehensive studies defined by larger geographical areas and longer time periods will ultimately present a better picture of the nature of these epidemics.

This literature review provides several interesting points to make about streptococcal epidemics in general and about the specific circumstances surrounding the Cape Breton epidemic. The exact beginning and end of the global pandemic reputed to have struck between 1825 and 1885 are not well documented. This is due to limitations caused by rudimentary medical knowledge concerning the many manifestations *S. pyogenes* is capable of producing as well as missing data specifically in the case of Cape Breton between 1878 and 1907. Each location that was affected in this pandemic, and in other epidemics in history, showed unique death rates and cycles. All streptococcal epidemics are clearly marked by a rise in virulence reflected in increased death rates. The

normal nature of streptococcal populations is one that is cyclical and even seasonally skewed toward outbreaks in the winter months. This cyclical nature is supported by a ubiquitous reservoir in the human host globally.

Streptococcal outbreaks display multiple manifestations, not just scarlet fever. Therefore cases of scarlet fever found in the historic record should alert the researcher to a more serious problem that is not to be taken at face value. Scarlet fever, as only one of these manifestations, seems to be caused by less toxic yet more abundant serotypes. Streptococcal populations are characterized as containing many different serotypes in different proportions capable of acting together in individual hosts to bombard the immune system.

Modern streptococcal epidemics show some unusual characteristics, with increased numbers of older children and young adults being affected. These are age groups traditionally absent from historic epidemics. The M-1 serotype appears to be the most devastating and the most common serotype to cause disease in modern times, followed by M-3 and M-4. Modern manifestations of the disease have also grown more numerous and more severe in nature. These include necrotizing fasciitis, streptococcal myositis, malignant scarlet fever, sepsis, bacteremia, and streptococcal toxic shock. Research efforts today should concentrate on dealing with these conditions as well as investigating the biochemical structures of the M-1, M-3 and M-4 serotypes specifically.

We can apply these lessons to the Cape Breton epidemic. Causes of death listed as “sore throat” are positively indicated to be associated with streptococcal infection when appearing in the historic record during a scarlet fever epidemic. A wide interpretation of

causes of death possibly being related to streptococcal infection has to be considered when they occur around the same time as the epidemic. Age can likely not be used as a discriminating factor when looking at these alternative causes of death. Finally, social factors, such as religious orthodoxy, are clearly associated with an increased number of deaths from the epidemic. A high internal contact rate of an infected group is positively correlated with increased circulation of serotypes and this may lead to higher death rates and more rapid cycling between epidemics. All of these factors emerge from the readings presented here concerning historic and contemporary streptococcal epidemics. We must apply knowledge of the interactions of these factors to the interpretation of any epidemic studies in the future.

Chapter 6: Disease Modeling Theory

Though this project specifically used the Stella® modeling program to create the models used to represent the 1875-1877 scarlet fever epidemic of Cape Breton Island, a discussion of the features of other types of models is given to locate the current project in a complex web of available methods. Model types and their uses are discussed to demonstrate the implications of each. The structure of this project's model is presented in detail to provide a reference point for the analysis based on the model.

6.1 Models and Their Uses

Models, all models, are used to understand a concept, problem or tangible structure. A model is an abstraction or representation of that which one is trying to understand. It is not an exact replica, nor does it seek to reproduce exactly the specifications of what is being modeled. Instead, a model has a general skeleton or outline that represents what is being modeled. Though there are details present in a model, these may not be all of the details of the object or idea in reality, nor are they necessarily all the same details. A model seeks to generally approximate a tangible or intangible thing and emphasize some of the details of that thing. It may even present other details that are not easily visible on the thing that is being represented but are hidden beneath its structure or elude direct observation.

For instance, a model of a house may display areas of that house that are subject to heat loss. In the real house, the heat loss will not look the same as it does on the model

because of air currents and fluctuations in the surrounding temperature and pressure. This is true even if the model is made to the exact specifications of the real house. However, what the model does is to identify an area of insulation or structure in the house that may be a problem for heating the house. The model does not have to be decorated like the house, painted the same color of the house, or even made of exactly the same materials as the house. For modeling purposes of heat loss, it may only need to approximate these specifications and present a general picture that alerts an investigator to problem areas for heating.

An example of an intangible concept that can be modeled is a country's economic system. This would be a very complex model with inputs and outputs to the economy, fluctuations in the value of the currency, availability of the work force, etc. However, the model does not have to exactly represent every person contributing to the economy. Aggregate statistics on the average number of hours worked per person, the average income per person, the average spending per person, etc., can all be lumped together and treated as a single population or sub-divided into smaller demographic units. These can be based on sex, age, race, language or whatever the researcher deems important to study. The desired results of the model and research questions will direct these choices.

This is exactly the same for models of epidemics, especially where the historic record is concerned. The goal of such models is to identify factors that may be contributing to the spread or effects of one or more epidemics. The problem in creating such models is that people are fairly complex biological organisms. Not only that, but these models seek to investigate interactions between humans and other complex

organisms, namely pathogens. Biological systems are constantly in flux and, in theory, all of the contributing systems to that flux can be individually pieced apart and understood. However, there are some factors that are not likely to influence the outcome of the model significantly.

Ideally, it would be desirable to test all of these contributing factors to see which ones might affect the model significantly. However, some factors are too minute to be concerned with. For example, a person who is part of a population being modeled for an epidemic spread may decide not to go to Sunday religious services every once in a while. We may even be able to pinpoint exactly when in the model they decide to do this, to quantify the amount of time they missed interacting with a large number of people and to estimate how many contacts this caused them to miss. However, it is unlikely that this event will contribute any significant data to the outcome of an epidemic model for the entire population. So, part of creating a model at first is brainstorming ideas, understanding what may be important in the model, and then rejecting those details which are not important. In one way, we are falsely elevating the importance of some factors by including them in the model in the first place. However, surprising interactions can take place in a model when complex relationships are built into it. Factors that seemed important to include can end up being of little importance while others that seemed trivial may hold a lot of unexpected power to affect the course of an epidemic.

When including factors in the model we have to be certain that we can describe how they might impact other factors. Even if we cannot quantify them initially, we must at least be able to recognize the relationships that exist amongst them. For instance, if

sexual transmission is a mechanism for spread of the pathogen we are studying, then this must be included in our model. We have to know how it affects the populations of people and pathogens. Is it the only route of infection? Is it the main route of infection? Many questions have to be answered about how all factors in the model relate to one another.

Algebraic equations can be produced in a model to show the quantifiable relationship between two or more factors in the model. Quantifiable does not necessarily mean that units of measure are attached to each factor. In fact, relative measures of relationship are commonly used. For example, if x is present then $y = 2x$. Where epidemic models are concerned, time is usually a function of the model as well. This means that these algebraic equations must then be translated into differential equations using calculus. This allows the model to progress through time based not only on the relationships of the factors to each other but also their own relationships to the progression of time. This is called a rate. For instance, people age, pathogens die, people die, people are born and pathogens reproduce at a certain rate. All of these may need to be included in an epidemic model.

6.2 Types of Models

The possibilities of creating unique models to answer specific research questions are endless. However, every model falls into one of three discrete categories. The three types are the statistical model, the computer-based model and the mathematical model. (Sattenspiel (2003) provides a thorough overview of these model types.) Though there are many applications for each type, these models will be discussed in terms of their

specific application to epidemics. Statistical models and mathematical models have been used by researchers for some time and do not actually require the aid of a computer but most often today use one in their calculations. Computer-based models are an outgrowth of the unique ability of computers to perform millions of operations per second. Computer-based models should not be confused with mathematical models that use computers to achieve a large number of calculations. The use of the computer in computer-based modeling is different because the calculations are often so complex as to defy mathematical analysis. In addition, they do not involve equations but rather thousands of decision-making steps of the “if”...“then”... type.

Statistical models range from the simple, such as regression analysis and correlation analysis, to the complex, such as time series analysis or spatial analysis. These models are often referred to as a “top-down” approach, meaning that they work with an underlying data set from the real world and work towards finding simpler patterns in the data. The patterns are tested with statistical techniques that compare the data set internally or by comparison to some larger population. Statistical techniques can also be used to filter out anomalies in data sets. Time series analysis, such as that done by Scott and Duncan (1998, 2001), uses such a technique. This filtering can identify patterns and cycles within the data set that are not apparent otherwise. In time series analyses of epidemics, cycles are usually related to the biology of the disease itself or, more commonly, to cycles within the population it affects. This can be attributed to the unique demography created by generations, for example, the cycle of population booms, such as the “baby boomer” generation of the post-war period and the generation of their children

that followed. It can also be attributed to the cycles in the lives of the people that the disease is affecting, such as the prices of wheat as shown by Scott and Duncan (2001).

Many computer-based models use the specific ability of computers to apply decision-making steps to “if...”then”... statements. They may also use the feature of algorithms that are simply a set of rules applied to the same situation. There is usually a complete lack of mathematical equations that would normally govern a set of decisions in modeling. However, within algorithms, there can be a complex set of equations that determine the set of rules itself or a process with several steps involved. Usually, the computer is programmed to make thousands of choices based on scenarios of encounters and movements.

One of the more recent types of computer-based models to have emerged as a promising way of modeling epidemics is agent-based modeling (Sattenspiel, 2003). Agent-based models share three general characteristics: the agents, the environment in which the agents exist and the algorithms or rules that direct how agents interact within the environment. The environment can literally mean that, or it can mean some social space that is intangible, such as that of an organization of individuals. The agents are individuals who each have attributes described for them. The program can randomly assign a combination of attributes to each agent or the attributes can be linked so that the presence of one necessitates the presence of another. For instance, if the agent is infectious it may also be required to have a slower movement rate on the landscape to reflect debility caused by symptoms.

During encounters of agents with agents, or agents with a certain aspect of the environment, choices are made about that interaction. For instance, an agent who is susceptible to a disease may encounter an agent who is infective with that disease. An “if...then”... statement might say something like “if the susceptible encounters an infective then a probability for the susceptible to catch the disease must be applied.” The movements of each agent in the environment are tracked and their encounters with each other are monitored. Each agent’s position in the model can be checked for encounters every unit of time, decisions are made, attributes are updated and the model proceeds through the next step of movements all over again.

Mathematical models operate from the opposite end of the spectrum to statistical models. They use a “bottom-up” approach that starts with a simple set of patterns and assumptions about the mechanics of the epidemic and then apply these to a set of starting data that models the complexity of the real world. These data may be characteristics found within the population being studied, such as the age groups of that population. From this, the model produces other sets of data about that population’s movement through the model as it encounters the patterns and mechanics set up by the model. This type of model is used in the current project and will be discussed at more length in later sections of this chapter.

Models can be deterministic or stochastic, or some hybrid of the two called semi-stochastic. Deterministic models comprise the majority of models in the literature today. These types of models “determine” the course of subjects of the model (hence the name) through set parameters. For instance, in order for an individual to go from being

susceptible to infective, a probability equation is applied to them. This equation is set in the sense that everyone who is susceptible encounters it and everyone who encounters it encounters the same equation. There is no randomness in the encounter or in the equation. This usually assumes there are constants to the processes in the model, though time- or other-dependent factors can be set in them. Constants may be based on biological reality or on averages in a distribution that applies to a population.

A stochastic model, in contrast, emphasizes the random effects that a population can experience. Each step along a chain of events may have a range of randomly chosen values that can be applied. These can represent the entire distribution of a known set of patterns or an estimated range of possible effects. Because of the random effects that compound in a stochastic model, no two runs of the model are ever the same. This creates a situation where several repeats of the model, usually 1,000 or more, have to be performed in order to completely display the patterns that the model creates.

Deterministic models run exactly the same way every time they are run.

Models can also represent different levels of population analysis. One may work on the aggregate level where entire populations are analyzed or the individual level where every individual within a population is different. If a population-level model is used, all individuals in the population are often given the average characteristics of the population. This may be reflected in a disregard for age, or a disregard for individual differences in immune systems. Individual-level models emphasize these differences. Agent-based models are good examples of this latter type of model.

There is a sub-category of population-level models that is a sort of hybrid. It considers the population as either structured or unstructured. An unstructured population model is one that truly treats everyone in the model as if they shared all attributes pertinent to the modeling with each other. This is not necessarily a bad thing. For instance, the common cold knows no race. In a model for the common cold, the population at risk would not have this characteristic included and everyone could be considered the same. However, this doesn't capture all characteristics that a person in the model can have, which is where structured population-level models come in.

Structured population-level models consider a few key characteristics about the population to be essential to the workings of the model. These are usually the very categories that the model seeks to investigate. For instance, the age of an individual may have profound effects on their experiences with a disease. Their occupation may also present different hazards specific to the disease. A structured population-level model would consider these differences and apply them by creating sub-populations within the model corresponding to these characteristics.

Finally, all models need to render time in a specific way. This can be done in discrete units of time or with continuous time. The main difference is that discrete-time models operate using a set of *difference* equations, such as those found in geometry or algebra, while continuous-time models use a set of *differential* equations, such as those used to form the basis of calculus. Calculus divides time into infinitely small units. It should be pointed out that continuous time models that use computers for this are not exact because the concept of infinitely small is impossible for a computer to recognize.

However, very small units of measurement are used to approximate differential equations with sufficient accuracy that the differences are negligible.

This project uses a specific type of mathematical model called a compartmental epidemic model. In a compartmental model, each person in each compartment is assumed to be the same in every other respect. This is our unstructured population. Unless other compartments are created, each male is the same age, height, sexual orientation and every other characteristic that might be conceivable for a person to have. Furthermore, there are no whole integers within each compartment, that is, the compartment of male is infinitely divisible so that “partial” males are a reality of the model. There is no such thing as zero males in a compartmental model (Jacquez, 1999).

This may sound like a very impractical way to conceive of a situation involving partial people who are the same as every other person. In fact, there are remedies to these assumptions of the mathematics of compartmental models. For instance, any number of compartments can be created that are discrete and act on their own. It is conceivable that one could create a compartment box for every person in a model. However, recall that a population-level model is not designed with this purpose in mind. This would be an individual-level model. Instead, compartmental models are designed to be able to generalize at some level because they are generally used to study large populations. We have to choose which attributes of a population are necessary to emphasize in the model. Their sex, age, occupation, income, height, etc., can all contribute to creating a separate compartment for that type of person. The modeler chooses the number of factors to identify for each compartment and the number of sub-factors within that compartment.

Note that the number of compartments increases rapidly as factors are added.

For instance, one may want to identify sex and age in a population. For sex, there are two choices, male and female. For age though, there can be any number of choices since age is a continuous statistic. For ease of creating compartments, you may wish to break age down into children, young adults, middle-aged adults and older adults. This creates four sub-compartments within age. Combining age with the factor of sex, we have a total of eight compartment boxes to account for each type of person. This is shown in Table 6.1 below. The complexity of the compartments can theoretically grow exponentially.

Table 6.1. The combination of just two factors in modeling can produce many more than two compartment boxes.

	Age			
Sex	Child	Young adult	Middle adult	Older adult
Male	Male child	Male young adult	Male middle adult	Male older adult
Female	Female child	Female young adult	Female middle adult	Female older adult

The usefulness of this method of modeling comes from the fact that aggregate comparisons of groups of individuals are very easily made. We can hold all other factors that we do not deem to be important to the model as equal by not creating a compartment for them. In our example above, this model would hold that only the factors of age and sex are important. Furthermore, it would also assume that only the four sub-compartments given for the factor of age are important enough to differentiate one from another. No further age sub-compartments are deemed to be important to this model. If

they were, further sub-compartments would need to be created to account for their distinction within the model. Otherwise, every person that fits into a compartment is identical to every other person within that compartment.

The problem with this type of model is that it can limit you to the number of compartments that are practically applied. In theory, one could create many distinct compartments by which to define the individuals in the model. However, in practice this challenge is limited by the increasing complexity of relationships that necessarily builds from such increasing detail. However, the advantage of this type of modeling is that it is very good and suitable to describing large populations where each compartment contains a large number of people. It is only when smaller numbers are encountered that the compartmental model breaks down in its ability to describe a system.

The model that is used in this project can be considered a continuous-time structured population semi-stochastic mathematical compartmental model. That is, it measures time through the use of differential equations looking at a population that is divided up into several sub-populations that flow through the model via a set of deterministic and stochastic equations that govern the flow between compartments. The patterns of interaction of the sub-population have pre-determined rules and these rules generate data about the population that are then compared to the other information about the population.

6.3 The SIR Epidemic Model

The specific type of model used in this project is the SIR epidemic model. This model is being used because it has the essential structure needed to model the large structured populations often used in modeling epidemics while being flexible enough to allow the application of semi-stochastic equations that determine the rates of flow from one compartment to the next. The general epidemic model was conceived by Kermack and McKendrick (1927) and is described in detail by Bailey (1975). It is termed the SIR epidemic model because the population under study is divided into susceptibles (S), infectives (I) and recovered (R). Susceptibles are those members of the population who have not been exposed to the disease and are potentially able to contract the disease. All members of the population start out in this compartment, except for one who acts as the index case. Infectives are those who have been exposed, are infected with the disease and are also capable of transmitting the disease to others. This is the compartment in which the index case starts. Recovered individuals are immune to the disease after having experienced it (Bailey, 1975; Sattenspiel and Herring, 1998). The flow of compartments is illustrated in Figure 6.1 below. In addition to this progression the susceptibles compartment can feature an incoming birth rate. The infectives compartment usually features a death rate based on data pertaining to the disease being modeled. All compartments can also have a second background death rate to account for deaths not caused by the disease being studied.

The rates shown in the model are referred to as the rate of transmission, or β , and the rate of recovery per unit time, or γ . β is a compound parameter comprised of the

probability that a contact will result in transmission, or τ , multiplied by the average number of contacts per person per unit time, or κ . We can then render these terms into a simple transmission expression of $\beta SI/N$. This is expanded in Equation 6.1.

$$\frac{\beta SI}{N} = \frac{\tau \kappa SI}{S + I + R} \quad (6.1)$$

In this equation, transmission is multiplied by the susceptibles and the infectives divided by the total population to arrive at the transmission expression to apply between the susceptible compartment and the infective compartment.

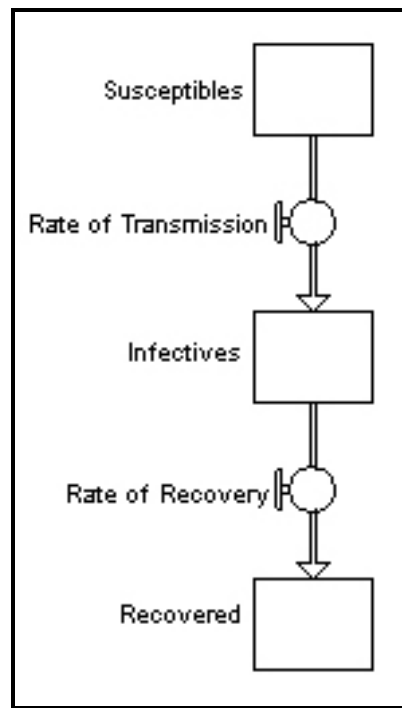


Figure 6.1. The progression of the SIR disease model compartments. This figure was created using Stella® 8.0 software by ISEE Systems.

Recovery is calculated by applying γ to the infectives. γ is the inverse of the amount of time spent in the infective state. For instance, if one typically spends an average of six days sick with influenza, the rate for γ would be $1/6$. This means that $1/6$ of the infectives will be moved to the recovered compartment with the passing of each unit of time.

This type of model is effective at describing the basics of the progression of an individual (as part of a structured population) throughout their lifetime for any disease state. However, the model is generally only applied to smaller units of time such as days or weeks. This usually suffices to describe the spread of a short-term epidemic throughout a community or area of interacting susceptibles without including births or deaths. Because smaller units of time are often the norm, background birth and death are usually not necessary to describe the system as new susceptibles are usually not present in large enough quantities to make a difference. However, diseases that have long infectious periods, such as HIV, or ones with dormant states in humans or other reservoirs, such as *S. pyogenes*, require consideration of changes in population numbers through birth, death or migration. Movement within a community can also introduce “new” individuals to other parts of a community or to other communities on a seasonal basis. This can, in turn, affect the seasonality of the epidemic itself.

6.4 SICR Epidemic Model

While the SIR equation is sufficient to describe a basic outline for an epidemic model, the specifics of each disease state demand adjustment to, and usually addition of,

other parameters. In the model used for this project, a fourth compartment containing carriers has been added to account for this important factor in the transmission of *S. pyogenes*. The revised SICR model is shown in Figure 6.2 below. The rate of flow from the infectives compartment to the carriers compartment per unit time is known as the carrier rate, or δ . This rate is not well documented, though it has been shown by several researchers that anywhere from 6.2% to 20.2% of the general population are carriers of at least one serotype of *S. pyogenes* (Duben et al., 1978; Moses et al., 2002). This carrier rate must then be high enough to maintain a constant supply of carriers within the population equal to any number between 6.2% and 20.1%. This does not mean that the value of δ should be 0.062 to 0.201 but that the proportion of carriers to the total population must be equal to a number in this range.

A birth and death rate were both added to the model in this project to investigate long temporal projections of the model. The chosen unit of time of the model was weeks and the total capacity of the model was 8,000 weeks or 153.3 years. However, the analysis for this immediate project only used a one year long cycle of 52 weeks. The birth rate comprises a single flow into the susceptible compartment. However, death rates must exit at each of the compartments, since anyone can die at any of these stages in the disease process. A scarlet fever death rate (SFDR) was also applied to the infectives compartment. This reflects a much different death rate which is caused directly by the disease being modeled. These additions are shown in Figure 6.3 below. Note that there is no need to have a compartment for any of these deaths because these become terminal points in the model. However, it should be noted that in the actual model, the scarlet

fever deaths flow does have a compartment at the end collecting those dying from scarlet fever. This is so that the number of deaths from this cause specifically can be recorded, graphed or input into a table for data analysis within the program.

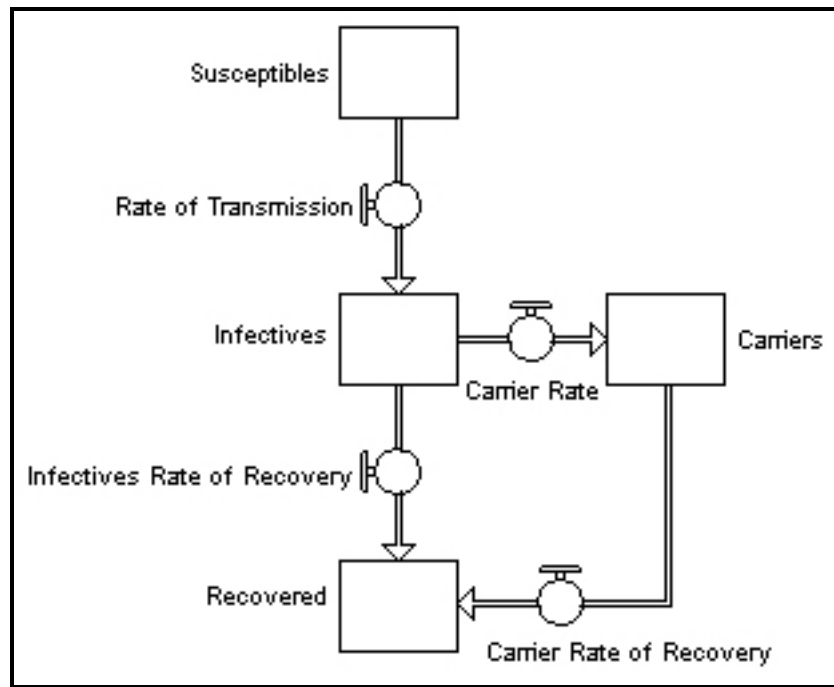


Figure 6.2. The addition of a carrier compartment to the SIR model creating the SICR model. This figure was created using Stella® 8.0 software by ISEE Systems.

The model includes several details that are specific to scarlet fever and need to be explained. The first challenge in creating this model was ensuring that it reflected the reality of the disease in many different respects. Since scarlet fever affects juveniles⁸ and adults differently, this difference must be reflected in the model. In order to accomplish

⁸ A juvenile is usually defined biologically as ≤ 15 years of age while adults are >15 years of age

this, the model seen in Figure 6.3 was essentially duplicated; the original part being for juveniles and the duplicated one being for adults. The only difference in the appearance of the duplicated model is that there is no death resulting from scarlet fever, since adults rarely die from scarlet fever. Therefore, the infectives compartment only features one death flow exiting from it.

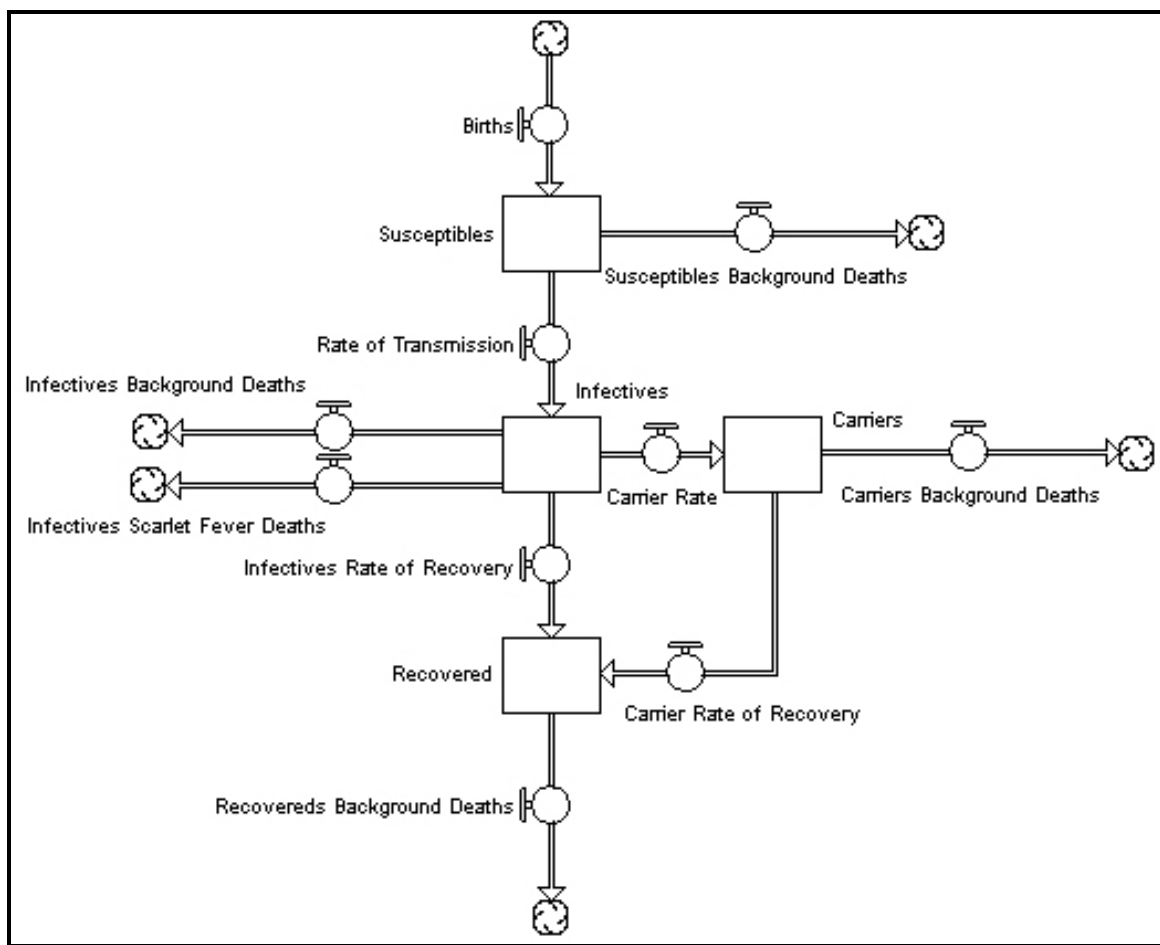


Figure 6.3. The expanded SICR including births and deaths. This figure was created using Stella® 8.0 software by ISEE Systems.

This project also sought to examine the impact of occupations on the disease. The Cape Breton sample is not straightforward in the occupations found there. However, a

large majority of individuals practiced fishing, farming or mining in the 19th century. Mining was only found in concentrated pockets of Cape Breton, though it was an important economic activity in the 19th century. Fishing and farming were much more widespread and can be found in every area in every county. Though there were still major concentrations of fishing in certain towns, it was widespread enough that it was seen as an economically viable activity in many parts of Cape Breton. This was especially true because of the island's easy access to water from most locations, be it the ocean or rivers and lakes. Even in the middle of the island, the long shoreline of Lake Bras D'or allowed access to the ocean, if needed, from some of the more remote inland areas. To incorporate these occupations, two additional structures were added, giving four sub-diagrams of similar structure. These four sub-diagrams represent juvenile farmers, adult farmers, juvenile fishermen and adult fishermen. Obviously, this does not imply that juveniles were necessarily employed in the farming or fishing industries. The occupation categories are therefore more reflective of household types. For example the daughter of a fisherman would be considered a juvenile fisherman for modeling purposes.

Once these four sub-sections or sub-models were established, they needed to be linked. The groups must act as one population and interact with one another. First, juveniles needed to become adults at some point. For models that are only concerned with a few days, weeks or months of output, there is no need to make this kind of connection. However, since long temporal analysis was a feature included in this model, a maturation flow was included. A simplified version of this is shown in Figure 6.4 below. A straight flow from juvenile farmer to adult farmer was not used in this model.

The reason for this is that this would pre-determine that only juvenile farmers became adult farmers and likewise for fishermen. Instead, a common “susceptible adults pool” compartment was created where juveniles of both household types could flow into the compartment. When the output flows left this compartment, the mixed “susceptible adults pool” was distributed to either fishermen or farmers based on the proportions of each found in the adult population at the outset of the model. This ensured that the same proportions of each occupation/household type were maintained.

Once the basic skeleton of the model was created, “converters” had to be applied to the flows in the model. The flows themselves did not contain the rates that were applied during each time unit. Instead, a converter had to be linked to each flow in order to supply the flow with a rate. The rate itself was contained within each converter and “packages” were also created below the main model where more complex operations within each converter were created.

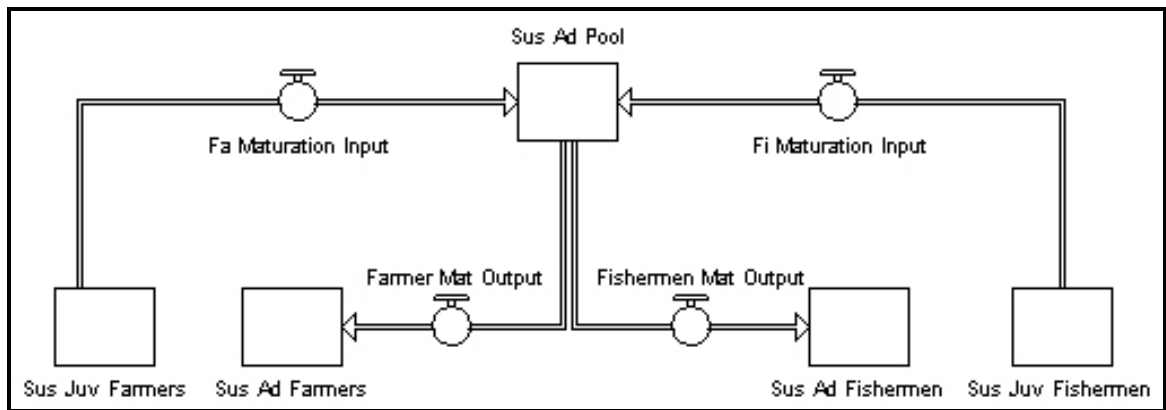


Figure 6.4. The part of the model showing the maturation flows from juveniles to adults. This figure was created using Stella® 8.0 software by ISEE Systems.

The simplest set of converters to explain is the crude rates. The crude birth rate (CBR), background death rate (BDR) and crude maturation rate (CMR) were all applied to their respective flows. All of these were derived from the population being modeled. The CBR was multiplied by the respective total population of farmers or fishermen. The BDR and CMR were multiplied by the respective compartments from which they flowed. A separate BDR was created for adults and juveniles reflecting their inherent differences in mortality. Juveniles are generally known to have higher rates of mortality, especially in historic populations that experienced high rates of infant mortality and the normal course of childhood diseases when vaccination was yet to be invented.

The scarlet fever death rate (SFDR) is a rate that is based on the available literature. This death rate is not based in demography but in the biology of the pathogen and the population's biological reaction to it. The death rates for scarlet fever varied from community to community and from year to year during the 1820-1880 pandemic. However, a range of 5%-30% is clear from the literature with typical numbers in the 10-20% range (Katz and Morens, 1992;Quinn, 1989). The rate in the model was set to randomly choose a number between 5% and 30% every time unit.

The flows from infectives to recovered and carriers to recovered both had recovery rates (γ) applied to them. γ is always equal to the reciprocal of the amount of time spent in the respective preceding stage. So, the γ from infective to recovered is equal to the reciprocal of the amount of time being infective, while the γ from carrier to recovered is equal to the reciprocal of the amount of time spent as a carrier. The carrier γ is obviously going to be smaller than the infective γ since the amount of time one spends

as a carrier is comparatively longer than the amount of time one spends as an infective. This represents the lingering of a disease in the carrier state as opposed to that unfortunate, uncomfortable, yet comparatively brief period during the infective stage. Both of these rates were multiplied by the preceding compartment to produce the rate of flow per unit time from that compartment to the next.

6.5 Applying the Data to the Model and the Remaining Rates

The remaining rates in the model are data-intensive and the explanation of each requires information specific to the data being used to create the model. The easiest rate of these to explain is the carrier rate. This is essentially the rate of infectives becoming carriers per unit time. The problem with creating a reasonable rate for this is that there are no good data for scarlet fever morbidity, or even streptococcal morbidity. This is especially true for the historic period under investigation. Illness is not something that is well-documented in many countries. Being a carrier (*i.e.*, essentially an asymptomatic infective) is even less well-documented. However, there are ways around this lack of direct data, and these ways nicely demonstrate the power of the Stella modeling program.

Several studies from recent epidemics have documented the number of carriers in a sample of the general population when investigating other aspects of streptococcal infections. Some have even broken this number down into juveniles and adults using the same criteria as this project. Recall from a previous explanation of the carrier rate that a recent study from Israel showed that 61 of 302 household relatives (20.2%) of index patients were shown to be carriers of GAS (Moses et al., 2002). Among this group, 75%

of the carriers were juveniles. The percentage of carriers (20.2%) was slightly higher than the range of 6.2% to 19.3% reported in a study from Czech Republic (Duben et al., 1978). Quinn (1989) reports on two much higher examples among juveniles. A Kuwaiti study in 1978 showed that the prevalence of GAS carriers was 47.5% among school children (Quinn, 1989). To further confirm the results of this study, a second study from Oxfordshire, England from the late 1970s showed that 51.4% of children were carriers (Quinn, 1989).

As stated earlier, the actual rate applied in the model should not directly reflect any of these numbers. However, the results from running the model should be used to adjust these numbers as necessary. Obviously, there is a difference in the rates of carriers amongst juveniles and adults. This is more than likely due to the undeveloped immune systems of juveniles. The ratio of adults to juveniles will also skew this number. If we have a total prevalence and a population count then some real data can be extracted for comparison. Unfortunately, both of these pieces of information are rarely seen together. We can piece apart the likely situation with the Israeli study (Moses et al., 2002). Their statistics show that the adult population of Israel was 3.86 million while the juvenile population was 1.688 million. The total population is therefore 5.548 million with 69.57% adults and 30.43% juveniles. 75% of the 61 carriers were juveniles, or 46 individuals. The expected number of juveniles amongst the 302 individuals studied would be 30.43% of 302, or 92 individuals. This suggests that 50% (46/92) of juveniles are carriers from this population. If the carrier prevalence rate of 20.2% is applied to this number, only 19 juveniles would be carriers instead of 46. The rate of 50% is a general

one and should be taken with a grain of salt since the sample of 302 individuals may not be representative of the ratio of adults to juveniles present in the total population.

When the rates were introduced into the model in this project, a graph was created to show the prevalence of juvenile carriers and the prevalence of total carriers in the population. By monitoring these numbers throughout the running of the model, adjustments were made to the actual carrier rates in the model so that the prevalence rates were affected properly without significantly disturbing the other results of the model. This was very simple to do in the Stella program with the aid of a “slider”. A slider simply allows one to adjust a converter rate in a sliding scale, much like a volume level adjustment on a stereo console. The rates can be manually adjusted and the model repeatedly run until the results run close to the actual historic data that are available for the sample.

This final point is an important one. The ideal of modeling is to reflect a real situation that took place at some point in time supported by actual data while demonstrating the effects of the factors that one has chosen to include in the model. End results of the model are analyzed and then checked against the pattern or total effects of data that are extracted from the historic record. If the data do not match the model, there is likely something wrong with the model. If the data do match the model, this does not necessarily guarantee that the model is accurate and correct in every respect. With several complex mathematical functions occurring simultaneously in a model, two “wrongs” do sometimes make a “right”. One must try to provide the model with as much robustness from data as possible without making assumptions that are untenable. The results and

patterns that the model produces can then be checked and re-checked to adjust the inner workings of the model. This is a type of *sensitivity analysis*, the details of which will be discussed in Chapter 8.

The final rate to explain is the rate of transmission, or β . It was mentioned previously that this rate is a compound parameter that can be broken down into two smaller components, the contact rate (κ) and the probability of transmission (τ). The probability of transmission is a largely biologically determined probability that is not well understood or documented. It essentially describes the probability that a susceptible will become infected when coming into contact with an infective. The amount and type of contact with an infective is obviously important. The mode of infection of the pathogen also figures into this probability. For instance, one can catch a cold in any number of ways; it can be transmitted via infected air droplets from a person within close proximity, or from a sneeze from a person 20 ft. away, or from mucous which has been exchanged over a medium such as a metal door handle or faucet from someone relatively far away and otherwise not directly contacted. Thankfully, *S. pyogenes* is not as opportunistic as the common cold. However, recall that there are several modes of infection with the added possibility of certain foods containing it (namely milk and powdered foods such as flour). There are few to no good data on this probability for good reason. The issue is currently too complex to study as it would involve tracking individuals, their current disease state, and the multitude of possible ways they can transmit *S. pyogenes* through direct and indirect means over the course of several weeks or months. This is simply not feasible.

This is one of the variables in modeling that has to be adjusted via the same method described for the carrier rates. Again, we look to the actual data and compare them to the results of the model to see if they match in overall appearance. We perform sensitivity analysis again, keeping all things in the model constant except for the rate or probability or factor we are analyzing. In Sattenspiel and Herring (1998), a probability of 0.5 was used for the highly infectious disease of influenza. This number was used as a starting point for this project. Through sensitivity analysis, a probability of 0.05 to 0.15 was found to be appropriate for the less infectious disease of scarlet fever. However, this probability was only useful for the infectives in the model. A τ for the carriers in the model also needed to be provided. Since the activity of the pathogens in the carriers is known to be much lower than in the infectives, we know that the probability must also be lower. We know from the information in Chapter 3 that the pathogen essentially stays dormant in the nasopharyngeal tract of the carrier until another disease, usually a cold, causes the inflammation of these tissues and re-activates *S. pyogenes*. Since the amount of time this could take ranges from several weeks to several months, the chance of catching the disease from carriers has to be much lower than the chance of catching the disease from infectives. In fact, it was found that a probability of 0.001 to 0.003 was effective at producing the desired effect of the carriers in the model.

The contact rate is essentially the number of people that a person comes into contact with per unit time. This is a difficult concept to completely capture in studies. For the purposes of this project, the major differences in contact rate are reflected in the different groups that are being modeled. Recall that there are four groups in the model;

these include juvenile farmers, adult farmers, juvenile fishermen and adult fishermen. The occupations and housing locations of these groups are largely responsible for creating all of the differences in their contact rates.

The contact rates are a reflection of three separate but interrelated concepts. First, they reflect the nature of the two occupations of farming and fishing. Second, they reflect the amount of daily community interaction within and between the members of each household type. Third, they reflect the relative amounts of trade engaged in by people in each occupation. Contact rates are also indirectly associated with age since scarlet fever has differing effects in juveniles. Juveniles are much more likely to reduce their daily interactions due to the debilitating effects of the disease. Reasonable constraints, based on historic reality of the two occupations and their associated households, have been outlined in terms of relationships of various characteristics of populations represented in the model. The relationships used are as follows:

1. Carrier contact rates > Infected contact rates
2. Infected adult contact rates > Infected juvenile contact rates
3. Adult fishermen contact rates > Juvenile fishermen contact rates
4. Fishermen contact rates > Farmer contact rates

Relationship 1 was based on the fact that carriers were more mobile in the population due to being free of any debilitating symptoms of the disease. Infected individuals were more likely to remain at home and not be in contact with members of the community outside of the immediate family. Relationship 2 was based on the fact that infected juveniles had much more debilitating effects associated with the disease in general. This is borne out

especially by the fact that scarlet fever almost never kills adults.

Relationship 3 was based on the fact that all adult fishermen had a higher contact rate than juvenile fishermen. This is a complex assumption that is largely tied to the ways of life of the two household types. Farmers were most likely to see only their immediate family for most of the week. This had the effect of equalizing adults and juveniles in the farming households, at least for this particular aspect. However, fishermen were most likely to see both their immediate families and their fishing group. Children of fishermen families were likely to see their play group and their siblings only. The children's play group size was, therefore, smaller than the adult fishing group size. Thus, relationship 3 served the purpose of reflecting this difference at all stages of the SICR model for the specific case of fishermen households.

Finally, relationship 4 was based on the previous argument for relationship 3 except that it was expanded to the general relationship between the two household types. As a result of these relationships, the following contact rates were devised, shown in table 6.2 below.

The basic transmission model, given above in Equation 6.1, can be adjusted to include a carrier state (Equation 6.2). This can be rewritten to include the occupation household and age categories. Packages were written to reflect the transmission for the four designated groups (Equations 6.3-6.6).

$$\frac{\beta SI}{N} = \frac{\tau \kappa (IS + CS)}{S + I + C + R} \quad (6.2)$$

Table 6.2. The κ values per unit time for the different groups that can infect a susceptible.

Group	κ Value
Infected juvenile farmer	3-7
Infected adult farmer	4-10
Infected juvenile fisherman	5-15
Infected adult fisherman	8-24
Carrier juvenile farmer	6-9
Carrier adult farmer	8-12
Carrier juvenile fisherman	15-25
Carrier adult fisherman	20-33

$$P_{JFa} = [(\tau_{IJFa}\kappa_{IJFa}I_{JFa}) + (\tau_{CJFa}\kappa_{CJFa}C_{JFa})] \quad (6.3)$$

$$P_{AFa} = [(\tau_{IAFa}\kappa_{IAFa}I_{AFa}) + (\tau_{CAFa}\kappa_{CAFa}C_{AFa})] \quad (6.4)$$

$$P_{JFi} = [(\tau_{IJFi}\kappa_{IJFi}I_{JFi}) + (\tau_{CJFi}\kappa_{CJFi}C_{JFi})] \quad (6.5)$$

$$P_{AFi} = [(\tau_{IAFi}\kappa_{IAFi}I_{AFi}) + (\tau_{CAFi}\kappa_{CAFi}C_{AFi})], \quad (6.6)$$

where P = package, J = juvenile, A = adult, Fa = farmer, Fi = fisherman, I = infective, C = carrier, τ = rate of transmission and κ = number of contacts. The individual packages were then used to create a total infected package for disease transmission within the total population. The total package equation is expressed in Equation 6.7.

$$P_n = \frac{P_{JFa} + P_{AFa} + P_{JFi} + P_{AFi}}{n}, \quad (6.7)$$

where n = total population.

Originally, the model was designed in such a way that, despite differing contact rates, there was an equal chance that contact could be made with any of the members of the other groups. That is, a juvenile farmer was just as likely that all seven of his contacts for the week would come from adult fishermen. This did not capture the intended role of the contact rates.

Part of the problem was that the model was designed without movement equations. Sattenspiel and Herring (1998) and Sattenspiel and Dietz (1994) have shown that mobility built into a model, even when done with no more than three locations, can greatly affect the outcome of the model. Movement within the community, because of differing household types, still requires that this be taken into consideration. The intent of the model for the current project was such that single locations would be modeled.

To fix this problem in the model, another rate was introduced into the transmission equation. This rate was termed μ and is referred to as the interaction modifier. The interaction modifier supplied the equation with a number out of two that represents the fraction of the amount of time spent interacting with members of one's in-groups and the fraction of the amount of time spent interacting with members of one's out-groups. This was applied to each of the transmission packages so that each could be adjusted. To simplify things, the in-groups were only split along household type lines, not age lines. Because there were four groups, the total of all μ 's in the equation must be four. Because these groups were further combined into one in-group and one out-group, the interaction modifier for each in-group and each out-group must equal two ($4 \div 2 = 2$). This meant that the two groups within each in-group (e.g., adult fishermen and juvenile

fishermen) and each out-group (e.g., adult farmers and juvenile farmers) had the same interaction modifier applied to them. The interaction modifier with one's in-groups was set at a random range of 1.72 to 1.8. The interaction modifier with one's out-groups was simply calculated as 2 minus the in-group interaction modifier. The new transmission equation appears in Equation 6.7 below.

$$\frac{\tau\kappa\mu (IS + CS)}{S + I + C + R} \quad (6.7)$$

Finally, a calculation problem was noted with the model. Long-range forecasting of multiple epidemics was attempted with the model. Cycles of epidemics do appear in the model if the time series is run for long enough. These even occur with some regularity as if to mimic actual epidemic cycles that a normal population of the 19th century would be prone to experience. However, the source of new index cases for each new cycle could not be identified. It was discovered that the cycles were due to a mathematical quirk of the model whereby the number of carriers never reaches absolute zero. Since computers cannot perform actual calculus, this number keeps getting infinitely smaller until the population of susceptibles is allowed to grow again. Once this susceptible population reaches a certain threshold, the “fractional” carriers eventually exceed one and a seemingly new index case of the disease is introduced to the population.

6.6. The Random Features of the Model

A brief statement needs to be given about the ranges of numbers that appear in some of the rate equations. The ranges of numbers that are given in some rates reflect the random function of the Stella software. This feature allows one to create a randomized

selection of a number within the given range per unit time. This feature was included to reflect the often random nature of disease transmission characteristics and the sometimes large ranges of variables shown in the data in the literature. The result is a model that creates non-smoothed results. The fluctuations in the selection of a random number within a range of possibilities alters each equation that includes such a range slightly every unit of time so that the outcome is never quite the same upon every running of the model. The random number generator in the Stella model was investigated with a simpler model in order to assess the manner in which random numbers were chosen. Over long repeats during the randomization process, it was found that each number within the range is equally likely to be chosen, i.e., the numbers are uniformly distributed.

The features and components of modeling are complex. These have been summarized here in order to present the basic mechanics of modeling and the important factors of the specific model used for this project. No model is a perfect description of a scenario that has taken place or of one that is yet to take place. However, they can clearly include enough detail to provide us with insights into what may have happened or what could happen. Models allow us to make adjustments to a representation of reality to see how that adjustment will affect the behavior or reality. They should, however, never be mistaken for reality. Details of the results of the model will be provided in the results of Chapter 9. Since this model is intimately tied to data derived from historic records, a description of these is provided next.

Chapter 7: The Cape Breton Sample

The island of Cape Breton, as part of one of the original provinces of the Confederated Dominion of Canada, had the institutions in place to comply with the record-keeping demands of the British Empire very early on. As the inheritors of this past, we are privileged to have at our disposal both official vital records and parish records. The nature of these records is variable in quality. However, it is this vast collection of information that allows us to peer into the past and interpret the way things were for our own benefit and to give the lost lives of so many people additional meaning for us.

7.1 Introduction to the Data Set

The process of recording vital statistics has a long history in Nova Scotia. Beginning in 1763, licenses for marriage by means of bonds were the first recorded vital events under mandate from the British government. The bonds are not very valuable to the demographic historian, however, since they only recorded the names of the potential bride and groom and the intent to marry, not the actual ceremony. In 1864, the British government passed legislation requiring the registration of all vital events (birth, marriage and death) throughout the British Empire. The onus for registering these events was upon the citizens themselves, with heavy fines levied against those who disobeyed the law. Government deputy registrars were appointed in each electoral district. These districts usually corresponded to the census subdistricts, which were centered on the largest towns

or cities in the counties with the smaller towns and villages reporting to the larger ones. The transcription of the registrar's data set used in this study suggests that a single registrar had the job for a very long time, probably as long as he was able. Each registrar was likely at least to be a somewhat educated man, one who could obviously read and write, and was probably a well-respected individual in the community with ties to many families in his district. It is unknown if this was a full-time position or a part-time designation that was called upon whenever vital events needed to be recorded.

Judging from the available records, compliance with the 1864 law mandating the registration of vital events was slow. However, eventually, the recording of events seems to have been fairly complete for the majority of the population. Around 1877, however, the British government repealed or struck down the law, although it is unclear from the literature what prompted this move. However, it is likely that there was an imbalance in its application throughout the Empire or there were insufficient funds to conduct the business accurately. Whatever the reason, the recording of vital events was abandoned as quickly as it was taken up. The records for Cape Breton counties in the year 1877 drop off abruptly mid-year. Some ended in the spring while others seem to have stopped in the fall. Unfortunately, there are no other contemporaneous sources that provided the same amount of detail that the government registrars provided. It wasn't until 1908 that another law was passed in the Canadian legislature that reinstated the mandatory recording of vital events.

Meanwhile, parishes throughout the island continued to record vital events as they had always done. However, this set of records not only lacks standardization among

religions, but is not consistent from priest to priest or minister to minister. The ever-present problem of relying upon religious organizations to record vital events is that religious beliefs will necessarily bias the nature of the information recorded. For example, many churches do not condone the baptism or christening of illegitimate children. If an illegitimate child dies, the child is not likely to receive the same type of religious burial and is also unlikely to be entered into the records of the parish. Unfortunately, many more illegitimate children are born into situations of poverty. Marriages between people of different religions, especially between a Christian and a non-Christian, may fail to be recorded by Christian parishes. Many churches in the 19th century, and even today, demand the Christianization of the non-Christian partner in the marriage in order to sanctify the marriage.

The data set used in this project consists of four overlapping types. The first, and perhaps what can be considered the most important source, is the vital death registers of each county of Cape Breton from 1864-1877. These counties, Cape Breton, Inverness, Richmond and Victoria, are shown below in Figure 7.1. The registers contain a summary of information recorded on the individual death records. Each death would have been recorded by the district deputy registrar on a death record certificate. These certificates were compiled at a later unknown date into a register. The registers are in the form of books with lined pages separated into columns so that facing leaves record the information of around 20 single deaths. All of these register entries are in hand-writing from the 19th century and require some practice to interpret. Registers were photographed and put onto microfilm at various times during the 20th century.

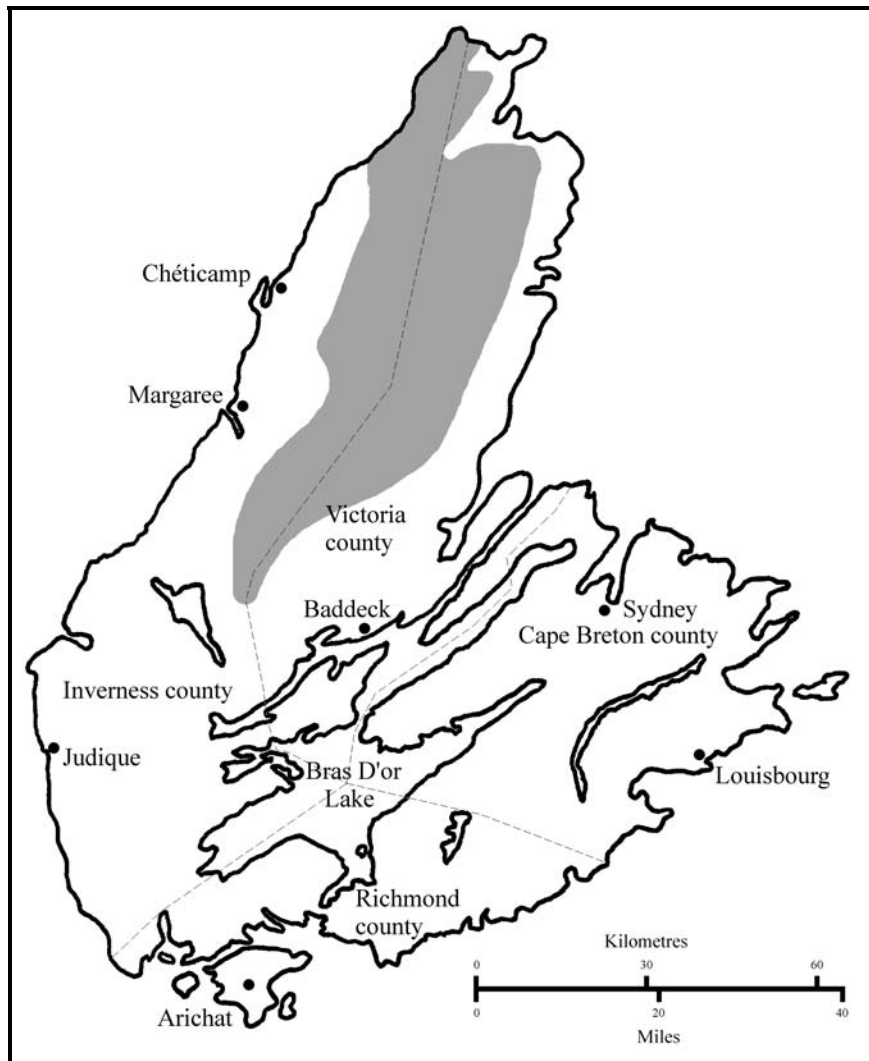


Figure 7.1. Map of Cape Breton Island showing the four counties. Grayed area depicts mountainous and largely uninhabited area of the Cape Breton highlands.

The second type of source used is the 1871 and 1881 censuses of each of the census subdistricts of Cape Breton. The island was divided into approximately 54 of these with the number of subdistricts within each county ranging from 12 to 16. They were not equally sized parcels of land but were meant to reflect population areas over the island. The region between Inverness and Cape Breton counties, referred to as the Cape Breton Highlands, is a mountainous region that remained crown land until a national park

was created there, and no subdistricts cross this boundary since very few people, if any, resided there.

These two census years of 1871 and 1881 bracket the eruption of the 1875-1877 scarlet fever epidemic so they are important in assessing the demographic nature of the population at the time and any changes that occurred in that ten year span. Since the majority of the population can be split into Scots and Acadians, two towns were chosen to serve as index towns for these populations. The subdistrict of Chéticamp was chosen to represent the Acadian regions, and the subdistrict of Judique was chosen to represent the Scots regions. Both of these are in Inverness County and were selected for their similarity in size. It should be noted that both are also named after their primary towns. A third subdistrict was chosen to gain a better picture of the Mi'kmaq population. The subdistrict of Red Islands has the largest percentage of aboriginal people in any subdistrict (8.5%) and the third largest absolute number of Mi'kmaq among all the subdistricts. It should be pointed out that none of these areas should be taken to represent cultural monoliths. Though these three groups maintained their identity and separation for the most part, intermarriage also occurred and will be an issue explored further in Chapter 8. The index towns served to provide more detailed information about the demographic make-up of the three cultural groups.

The census records were also used in reconstructing the families of the scarlet fever victims. Once scarlet fever or other streptococcal victims were identified in the death records, the 1871 and 1881 censuses were consulted to find the victims and their respective families. Though the epidemic was concentrated between 1875 and 1877, the

census years provide a summary of the family and household structure at the time of each victim's death. This included both the household type (based on the occupation of the father and any other adults in the household whose occupation is listed) and the number of adults and juveniles living in the household at the time. These data are available in aggregate form in the 1871 census tables mentioned below (Canada Dominion Bureau of Statistics, 1876).

Occasionally siblings, parents or other members of the household would go "missing" between the two census years. That is, individuals would appear in the 1871 census but not in the 1881 census. This occurred in five instances out of the 33 streptococcal deaths where both census records were available and transcribed. The death records were consulted to see if the individual had died in the interim or if they had otherwise left the household. If it could be confirmed that the missing person had died before the epidemic victim had, the missing person was removed from tracking. This was done since the missing person was not present, and therefore not a factor, during the infection period of the epidemic victim. If the missing person was inexplicably gone, as a result of migration within or away from the town, they were counted as present. This is obviously an imperfect system as many scenarios can be responsible for the missing person's whereabouts. However, the important thing was to remain consistent. When a person was present in the 1871 census, but absent in the 1881 census, and was not found to have died in the interim, it was best to assume they were alive and present in the household when the epidemic victim died. This assumed the 1871 information to be accurate, as did the use of the 1871 tables (mentioned below) for the aggregate statistics

on the same information. This also relies on the burial and marriage records of Chéticamp being very accurate and assumes nearly every burial and marriage in the community was recorded.

One effect this assumption can have is to falsely reduce the number of dead between the two census years and, in turn, it can falsely raise the presumed population during the epidemic. A quick test can be run to determine the validity of this assumption. If we calculate the natural increase by subtracting the annual burials from the annual deaths for Chéticamp between the two census years, we can add this to each preceding year starting with the 1871 census population count of 1,915. The result can then be compared to the census population count of 1881, which is 2,726. The results from following the annual natural increase show the 1881 population should be 2,722. This means that an overall natural increase discrepancy of four people exists between the censuses and the parish records. This does not necessarily mean that four whole people were missing in the baptismal records or that four whole people immigrated to Chéticamp. However, with a population of this size, it likely represents one or two families immigrating to the town.

Subdistrict boundaries do not coincide with county lines in this region, which can be confusing for the historian. This is not a problem when working within the census records by themselves but it is when trying to apply census data to vital records data. The vital records are organized by county and the registrars were appointed by subdistricts that roughly corresponded to the census subdistricts. However, it seems that the residence of the deceased in life was the deciding factor for where within a subdistrict their death

was recorded. For example, the subdistrict of River Inhabitants crosses the southern end of the boundary between Inverness and Richmond counties. While all the families within River Inhabitants may be enumerated on one census form, their deaths will be split between Inverness and Richmond counties depending on which county they resided in during life.

The tables supplied in the *Censuses of Canada 1665-1871* volume published in 1876 (Canada Dominion Bureau of Statistics, 1876) assist with summarizing the data contained in the censuses. Despite the title of this work, it mainly focuses on the 1871 census and has 55 tables summarizing the 1871 census data in different ways. This includes summaries of the population, properties, industries and land. For many of the tables the information is broken down by subdistrict, though some only contain information at the county or provincial level.

The third primary source of information being used in this project is the parish registers of the town of Chéticamp. The religious centers of Chéticamp have seen many changes since the founding of the town in 1782 and its official recognition by land grant in 1790. Up until the construction of the first chapel in 1812, religious ceremonies were held in the home of the family where the priest was lodging. However, after construction of the first chapel in 1812, consistent recording of religious ceremonies began. These include baptism, marriage and burial. Though the ceremonies of baptism and burial do not exactly match the vital events of birth and death, the use of these records as substitutes of the vital events is an acceptable practice in demography (Hollingsworth, 1969) as long as the date of the event and the date of the ceremony are known not to

differ much in the religious-cultural setting. This is the case in Chéticamp, an almost entirely Roman Catholic community. Some entries in these records even give the date of both the birth and baptism.

These registers provide detailed information about the population fluctuations of one typical community on Cape Breton Island on a yearly basis. Burials and baptisms, in conjunction with the 1871 and 1881 censuses, allow for the reconstruction of population numbers while tracking the annual changes between the two censuses. Perhaps more importantly, these records provide the basis for genealogical reconstitution. This is important when information about families within a population is needed. Though censuses provide us with a snapshot of individual families in the population, parish registers usually provide us with enough information to link families together. Anecdotal connections are also visible in this type of records since godparents are often named in baptisms, as are witnesses in marriages.

The town of Chéticamp during the 1875-1877 scarlet fever epidemic was one of the hardest hit communities. The geographic limits of the parish records at this time run from the subdistrict line with neighboring Margaree up to the region known as Cap Rouge (Chiasson, 1998). This essentially covers the entire subdistrict of Chéticamp. By 1879, a second church was erected in the community of St.-Joseph-du-Moine, just southwest of Chéticamp. Until this time, any religious ceremonies were recorded by the church in Chéticamp.

Like the vital records that they complement, this source of information is not without its own problems. As mentioned already, religious biases often prevent the

completely accurate recording of events. Historians have noted gaps in the record, most notably a ten year block missing between 1846 and 1856 (Chiasson, 1998). Though it is not known specifically what happened to the original records in this period, Chiasson (1998) suggests that an entire volume, in the form of one book, went missing or was destroyed. This does not make sense. In viewing the original volumes, I noted that the years did not correspond to a single volume, nor is there any reason to believe that one decade would fit within a single volume.

My 1997 fieldwork conversations indicated that there is also the possibility that a priest had fathered a child during this time period. These conversations arose independently from two very credible sources directly associated with the church. They are both trusted authorities on the history of the region but wish to remain anonymous due to the nature of the subject. The story goes that in fear that a visiting bishop would find out, the priest himself, or perhaps a later priest, removed the years from the records permanently. This suggestion is corroborated by Chiasson's (1998) description of the priest present for a year or two during this period. Father H. J. Chénal was perceived by the people to be quite an arrogant and irritable character. It appears that letters were written by the lay church wardens on behalf of the congregation to the bishop in charge of the diocese concerning Father Chénal's behavior and demeanor. He may have been the culprit who lost (on purpose or by accident) the records, or the priest who is suspected of fathering a child.

The original books used to record the ceremonies are still available at La Société Saint-Pierre in Chéticamp, located at the cultural centre Les Trois Pignons. By the 1980s,

the late Father Charles AuCoin completed a typewritten transcription of these records. He was also instrumental in beginning the current database project that is underway to use these records to trace the genealogies of all Chéticamp residents and their descendants. Photocopies of these transcriptions were obtained for the years 1868-1891.

The final primary source of information used in this project is the maps of A.F. Church. In 1864, Ambrose F. Church was commissioned by the Nova Scotia legislature to create a series of maps for each of the 18 counties in the province. The work was completed in 1888. Cape Breton County was completed in 1877 while the other three counties of the island were completed from 1883-1887 (Applin, 2004). These maps use a 1 mile:1 inch scale and are so detailed that individual buildings are shown depicting the first initial and surname of the proprietors. The maps also include many topographic features and insets of major towns.

7.2 Summary of the Data Set and Accompanying Maps

The types and value of information available from each of the data sources varies. Each is valuable in its own way since none contain all of the information of the others. Some have direct application to this project while others offer indirect evidence to tie in events or people to the equation. Each will be explored in detail here so that their uses in this project are explicit.

The vital death registers contain the most direct evidence for identifying and tracking the scarlet fever epidemic. These hand-written records are organized on two facing leaves of a book. Each person's death is entered on two lines of these pages, each

of the pairs of pages holding an average of 20 death entries. The columns contain the following information:

- Number (usually numbered consecutively starting with “1” for first death of year)
- Name and Occupation (first name and surname, some middle names)
- Sex
- Age (fractions often included for infants)
- Condition (child, youth, single, married, spinster, bachelor or widow)
- Where Born (varies in specificity of information included)
- Parents’ Names, Occupation, and whether living or dead
- Date and Place of Death
- Certified Cause of Death (blank if not given, unknown or unspecified)
- Signature of Informant
- Where Registered and Signature of Deputy Registrar (included date in earlier entries instead of location)

Each page also contains a page number, and the county and years of death. The information given for the occupations of the parents is almost always only the father’s. This is likely because of a simple lack of registered female work, if any, outside the home during this time. The closest mothers get to having their work recognized is that sometimes “Farmer” is pluralized to “Farmers”, suggesting a family operation. However, it is hard to imagine that those entries that do not make this explicit only had the father working on the farm. There are a few exceptions to this and occasionally mothers’ occupations are listed separately. Their maiden names are also occasionally included in this section.

With the place of death information also comes a mixture of levels of specificity that is similar to those found for the place of birth, though locations in both entries are usually not outside of Cape Breton and they are often easy to identify for that reason. These can also be cross-checked with the place of registration, since the two are usually

close to one another, if not the same place. Some unhelpful notations simply state the place of death as “At Sea” which suggests a fishing or merchant voyage of some sort but does not specify the location. The safest assumption to make in this case is to look at the places of birth and the category of “where registered” to garner the likely location of the person’s residence in life.

The “informant’s signature” occasionally had a small cross or “X” after their name. Though the registers are simply transcriptions and do not contain actual signatures, it is believed that this mark indicates that the person signed with an “X” instead of being able to actually sign their name. There was also occasionally a place of residence of the informant mentioned in this column. The rest of the columns mentioned are fairly self-explanatory.

It should be noted that several different types of handwriting have been used in these registers. The hands change after a double line is struck between the two differing records. There is usually no consistent date when this happens, such as at the end of a month or year. This suggests that the original certificates were being copied *en masse* by several different employees likely working for the provincial and/or federal government. There are even detectable errors between transcribers where one entry might show the middle initial of an often-repeated Deputy Registrar as “M” while a little further along in a different hand the letter is written as “W”. Spelling differences of deceased person’s names, place names and causes of death are quite common, especially where English-speaking transcribers or registrars are copying the names of deceased French speakers, and sometimes Gaelic speakers. These are minor problems that are overcome with a

sustained familiarity with the region and its people.

A final point should be made about the nature of these records concerning the causes of death and some of the dates of death as well. A later hand of a different, more modern, cursive style appears written above several of the entries. For example, over the entry of “Inflammation of lungs” (*sic*) may be written “Pneumonia”. At first, these seem like corrections or addenda to causes of death once more information was available. However, after much consideration of *what* is being written it seems clear that a later transcriber or (hopefully) official viewer of these records was reinterpreting the cause of death information and substituting the then-modern word for the condition. These entries were not entered in my transcriptions on account of the amount of text required to explain such a “second entry” in spreadsheet format. The interpretations are anecdotally helpful but not practical for the evaluation of the cause of death for the current project. In fact, much misinformation may be contained in these “corrections” and it is my opinion that future viewers should be aware of their possible and probably misleading inaccuracies.

All causes of death were standardized to a single spelling. The specific interpretation of each disease state related to scarlet fever is elaborated upon in Chapter 8. After standardization of cause of death information was completed, the closest modern counterpart to the cause was entered in a separate column. These counterparts are consistent with the International Classification of Diseases (ICD) and correspond to an alpha-numeric code as well (National Center for Health Statistics, 2004). Victims of scarlet fever, rheumatic fever, and possible victims of other streptococcal infection, were assessed using these codes.

The census records have taken on a different role. Since they do not contain cause of death information, they have been used to supply information about the families of the scarlet fever victims. An attempt was made to locate the family of each victim in the 1871 and 1881 censuses. Helpful information from the vital death registry included names of parents and their occupations, age of the deceased and locations of death and registration. The census allowed for the analysis of some additional information. The columns included on each 1871 census form included the following:

- Vessels
- Shanties
- Dwelling houses in construction
- Dwelling houses uninhabited
- Dwelling houses inhabited
- Families
- Names
- Sex
- Age
- Born within last twelve months
- Country or Province of Birth
- Religion
- Origin
- Profession, Occupation or Trade
- Married or Widowed
- Married within last twelve months
- Going to school
- Over 20 unable to read
- Over 20 unable to write
- Deaf and Dumb
- Blind
- Unsound mind
- Date of Operations and Remarks

The 1881 form differed only slightly. Three columns were added at the beginning citing the division (geographic), page and number and the “over 20 unable to read” and “over 20 unable to write” columns were lost.

The census summary tables collected in the 1876 publication *Censuses of Canada* are too numerous to detail here. There are over 50 tables, many of which are broken down at the subdistrict level. They summarize the demographic composition of the 1871 census, for the most part, for such features as age, sex, religion, origin (ethnicity) and occupations. These are included for both the living and the dead of the 1871 census⁹. Several additional tables, broken down at the county level, summarize the land and the sea and their respective economic products. The economic tables are quite detailed giving values of wages for each occupation.

These tables were used in many ways. In the initial stages of the project, they served to guide me to the appropriate census subdistricts to act as comparisons for Acadian and Scots settlements. They also provide a numeric map of the region in terms of its industries, their concentrations over the landscape, and their relative values within the economy. Products such as butter, coal and fish are good economic indicators of the main industries on the island just before the time of the epidemic. Hornsby's volume on the 19th century in Cape Breton is extremely helpful for conversions of many of these tables into maps with pie charts and bar graphs superimposed on the regions (Hornsby, 1992).

The parish registers of Chéticamp contain three different types of entries. These are baptisms, marriages and burials. The baptisms contain the name of the baptized, their date of baptism, names of the parents and the names of the godparents. Occasionally these also contain the year that the parents were married. The marriage entries contain the names of the bride and groom, the date of the marriage and the names of the witnesses.

⁹ Refers to those who died within one year of the taking of the census, which most often occurred in April.

Degree of consanguinity, or relatedness of two people, is also occasionally mentioned. If two people were consanguineous to the 3rd degree, that is, they were 2nd cousins, they usually had to seek the dispensation of the local bishop for the marriage to take place and also pay a fee. Consanguinity to the 2nd degree required dispensation of both the bishop and the pope and a larger fee (Chiasson, 1998). The burial entries contain the name of the deceased, the date of their burial, their parents and/or spouse's name (differentiated if predeceased), the age of the deceased and the name of a witness to the burial. Cause of death is never mentioned on these records.

Finally, the A. F. Church maps of Cape Breton Island counties contain some very detailed geographic information about the people and the land. Buildings and houses are shown on the detailed maps. Each house shows the proprietor's name with the first initial and the surname. The other buildings are noted by their function; for example, saw mill, church, forge, etc. Barren land is also noted, which basically indicates land that is "unsettled" by Europeans. Rivers, brooks, creeks, topographical features and roads (permanent and seasonal) are all shown in detail on the maps. This great detail has aided in the reconstruction of the social geography of the landscape.

Despite the formal introduction of reserves as early as 1821 in Nova Scotia, Mi'kmaq encampments and even villages existed that are probably not noted on the maps. There are also names missing from the maps that otherwise appear in the 1881 census. This may be because of movement of the proprietors or because of lack of accessibility to the property by the cartographer. It is unknown if Church himself actually covered the entire length and breadth of Cape Breton Island, gathering the recorded

information by knocking on every door, or if he had enlisted help from assistants. Either way, it is conceivable that houses and people located in the deepest wilds of Cape Breton in the late 19th century were not easily found, as such is still possible even today.

All of these very useful primary sources have provided the raw data needed to undertake this project. They are not a complete list of what is available for the region. Many parish registers still exist that have not been consulted here due to time constraints. Digging deeper into the region may also prove that some form of morbidity data may exist in doctors' diaries or hospital reports. Nonetheless, these data provide a vast array of information to coordinate into meaningful stories about the lives of the victims of the 1875-1877 scarlet fever epidemic.

7.3 Identifying Scarlet Fever and Other Causes of Death from the Historic Record

One of the most daunting and challenging tasks of the archivist is to correctly interpret the records of the original sources used in the research. This project is no different. There are several layers of reporting, each with their own context that must be explored to illuminate the way in which the end interpretation is reached. For the censuses and vital records of Nova Scotia, these layers of reporting include the first-hand account or witness, the doctor's diagnosis (if present), the registrar's written account and any copyist's hands that may have re-copied the information after the original log books were compiled.

The first-hand account for a census was usually, and still is, a local resident who was a respected member of the community and had a minimal amount of education. For

census information, a vast number of things can be miswritten at this very first step. Cause of death information, for this particular data set, is not present. Because of these problems this source is useful only for back-checking family groups and perhaps determining ages of individuals at death.

The first-hand account for a vital record is often much different. This can include the person who witnessed the death, the doctor attending a patient, the priest who gave the sacrament of extreme unction (last rites), or simply the person who reported the death for the family because they were a family friend or an educated individual. There are obviously varying degrees of education possible amongst these candidates. This is important to point out because the cause of death may not be correctly identified by a relative of the deceased. In fact, it seems that the cause of death was not reported in a large percentage of cases (upwards of 40%) and no entry is even listed in this field. It is likely that in many of these cases an uneducated family member reported the information.

To whom were these causes of death reported? In the case of the vital records, at least for the island of Cape Breton, it seems to have been a similar type of person who would have been responsible for taking the census. It may have even been the same person. This would have simply been a respected member of the local community with at least a minimal education. This person probably had some folk medical knowledge through experience in his job and was generally good at spelling in a consistent fashion the variety of conditions that would qualify as a cause of death. Apart from that, the person was not expected to have any medical training.

The main job of the local registrar was to fill out the death certificate. It is possible that priests were responsible for helping with this stage of the process. They were trusted members of the community. They were educated and some even had a smattering of medical training and certainly Latin, which helped in classifying diseases. They were also responsible for the inhumation of the corpse. Therefore, priests probably fit nicely into a role of advisor to the registrar in confirming cause of death information. Bynum (1996) suggests that in the 19th century clergy were often cast in the role of not only healing souls but also bodies.

What is interesting about this stage of reporting is that in Canada before 1944, all deaths reported were classified by place of occurrence, not place of residence as is now the practice (Statistics Canada, 1944). These two were usually the same in late 19th century Cape Breton, but not always. Because of the manner in which information is organized, one must be vigilant to note the birthplace of the deceased. This is not a fool-proof method either because people did not *always* live where they were born, especially for the Scots immigrants. However, with regard to this project, place of occurrence is more relevant because we are looking at where the person likely caught the disease. For a disease like scarlet fever that kills rapidly, the two places are almost always going to be the same.

After compilation of the death certificates, there is some debate about the next step. Either the local registrars made the trip to the county office to enter their rolls into the logbooks, or the certificates and logbooks were compiled by an unnamed master registrar in charge of a certain area. There is consistency in hand-writing between

registrars in some places, indicating the latter. There are also instances where the handwriting of the reported death changes depending on the registrar, indicating the former. However, instances of a master compiler being responsible for final entry into a logbook seem to be more frequent. This causes a third-person account to slip into the process, thus creating another potential chance for mis-recording information.

After this, the logbooks likely sat at the provincial ministry (department) office branch in charge of such things. The current federal embodiment of this is Statistics Canada, but it was the Canada Dominion Bureau of Statistics at the time of the scarlet fever pandemic. This body works in cooperation with each of the provincial branches of the Archives and Records Management. Hand copies were likely made in the 19th century of the logbooks that contain the summary information from the death certificates. These would have been sent to the federal office in Ottawa and retained there. The records available to the 21st century researcher are those that were preserved in the Ottawa branch, now residing at the National Archives of Canada. Photocopies, microfilms and/or microfiches of each of the vital statistics registers have been made for various genealogical and historic institutes, such as the Beaton Institute of the University College of Cape Breton, Sydney, NS where data for this project were acquired.

Given all of these steps, from the death of the person to a microfilm in a research institution, there is certainly margin for error. The largest margin likely did not occur in copying. At least four stages of copying can be identified in this process: witness' word to local registrar's written word, local registrar's written word to master registrar's written word, master registrar's written word to copyist's written word, and copyist's

written word to researcher's database. Though these present several opportunities where miscopying could easily have occurred, a much more likely source lies in the medical knowledge of the time. Diseases with many variable manifestations, such as those caused by *S. pyogenes*, require clinical microscopic diagnosis or serotyping to be positively identified, even today. Therefore, regardless of a lack of clinical diagnosis in the historic account of a cause of death, the historic record still at least reveals the symptoms surrounding the death as well as a demographic profile of the individual. These can be used in conjunction with the epidemic context to compensate for insufficient medical knowledge.

Chapter 8: Methods of Analysis

The number of methods that can be employed to analyze a problem of this scope is tremendous. The data set is large and the ways one chooses to sift through the data are important in determining *what* can be explored with those ways. Basically, three approaches were taken to analyze these data. The first was to use traditional demographic tools to organize and sort individuals based on shared characteristics such as age, ethnic group, household type, cause of death, etc. These help to demonstrate trends within the data that may not be readily apparent otherwise. The second was to include a geographic analysis, albeit a small one for this project. The main focus of such an analysis in this project was to look for any trends in the epidemic along geographic lines. The third was to design and test a computer-based mathematical model to examine factors related to the course of the epidemic under study. As discussed in Chapter 6, this included sorting individuals into compartments based on shared characteristics. The outcome of such an approach differs from traditional demographic tools because the model seeks to create a moving representation of the living population as it experienced an epidemic of scarlet fever.

The initial step analyzing any of these data is to identify cultural groups in the historic record. As mentioned before, if the historic record is taken at face value, it appears that the Acadians were hit very badly by the scarlet fever epidemic and that the Scots were barely affected. In order to determine if this was truly the case, the ethnic groups themselves were identified. Once the ethnic group was established for the

majority of people in the record, the data could be sorted according to this characteristic and compared to their role in the epidemic, occupations and any other characteristics identified.

8.1 Identifying Cultural Groups in the Historic Record

The ethnicity of individuals or the cultural group with whom they are associated is one of the most difficult pieces of data to extract from written historic records . The question of personal identity is bogged down by many layers of culturally complex issues and factors that go into the making of that identity, sometimes changing over a lifetime. It is also deeply molded by the groups with which one is socially and culturally associated. These groups can be intentional (i.e., friends) and unintentional (i.e., family). All of these factors combine to make a complicated picture of one's own ethnic identity. Additionally, this personal view may be slightly or very different from the way society at large views that same individual.

This project attempted to draw a few broad categories that would roughly describe the *most likely* group an individual would be placed in by society at large. Personal choices or views in this matter are practically impossible to get at in the historic record unless a personal statement on them is recovered. The researcher working with historic records is then left with the choice of taking the extreme view that such a task of assigning ethnicities is impossible or by taking the middle road and saying that some assumptions can be made if one is consistent and careful about them.

For this project, two assumptions were made to determine the cultural group or ethnicity of an individual. The first of these is that the surname of the individual bears a link to the ethnicity of the individual. The second is that the individual or the society at large would recognize the individual as such. There is obviously a male bias in making the first assumption. Unless dealing with marriage records over a broader period of time than this project sought to cover, one cannot extract the maiden names of females from the historic record. This is because families would have to be researched for each married female so that her maiden name could be determined. This would require finding the marriage record at least for each female. In death entries and in censuses, married or widowed females are almost always listed with their married names only. For all cultural groups involved in this study, male and female children also inherit their father's surname, not the mother's. Therefore, an assumption about a person's ethnicity based on their surname has a male bias. Even if a maiden name is available for a married female through mention of her father's name, it is still her father's name and the male bias is intact.

Before data were examined for other features pertaining to the epidemic, each individual in the vital death registers was assigned an ethnicity based on the surname method. All males were assumed to carry their father's surname. All single or juvenile females were also assumed to be carrying their father's surname. All married and widowed females were sorted by their fathers' surnames and were assigned an ethnicity if a surname was listed for their fathers. All those whose own surname was not listed or who were married or widowed females without a father's surname listed were given an

ethnicity of “Unknown”.

Some surnames on the island of Cape Breton can be clearly associated with particular ethnic groups. This is because up until the 20th century, the number of ethnic groups was very limited, with most deriving from a few European groups that immigrated to Cape Breton. The Acadians come from a relatively small number of original immigrant families. The surnames are quite limited and are even largely exclusive from those who settled in Québec and sometimes from those who settled in other parts of Acadia (New Brunswick and Prince Edward Island). A fairly complete list of Acadian surnames for the entire Acadian region is available online (Muhn, 1998). Due to English-speaking census-takers, registrars, and transcribers along the path from data collection to the historic record, many of these Acadian surnames have been Anglicized or simply linguistically butchered and occasionally bear little resemblance to their actual names. For example, LeBlanc is Anglicized to White, Sançon is Anglicized to Samson or Sampson, AuCoin is Anglicized to O’Quin. A list of the most common changes is given below in Table 8.1.

Many of the Scots families who settled in Cape Breton came from the Western Isles and the Western Highlands of Scotland. Due to the clannish nature of the Scots, many surnames are found in pockets divided along geographic lines in Scotland proper. The combination of these two factors has resulted in a limited number of surnames from the area. Many of the surnames begin with the Gaelic prefix “M^c” meaning “son of”. It is a common misconception that the spelling of this prefix determines the difference between Scots and Irish but this is not true. Spellings of this were not standardized until the 20th century and are likely a fabricated convention. Amongst the most common “non-

M^c names are Beaton, Campbell, Cameron, Ferguson, Kennedy, Matheson, Morrison, Ross and Urquhart.

Table 8.1. The most commonly misspelled Acadian surnames and their most common Anglicized versions.

Acadian name with probable spelling	Most commonly Anglicized version(s)
AuCoin	O'Quin, O'Quinn
Chiasson	Chessong, Chaison
Gaudet	Godet (Often pronounced "GOD-it")
Haché	Ache
Josse	Joice, Joyce
L'André/L'Andry	Landry
Langlois	Langly, Langley
LeBlanc	White
Maillet	Mallet, Mallette
Marchand	Marshong
Muise	Muse, Meuse
(De)Roche	Roach ¹⁰
Sançon	Samson, Sampson
Thibault	Tebo, Tibot

Irish ancestry is very difficult to discern at times. For instance, the name Kennedy is common in the Scots that immigrated to Cape Breton (based on place of birth information) but not in the Irish, as one might think. Kennedys exist both on the west coast of Scotland and throughout Ireland. Jamiesons exist throughout the British Isles. Population movements throughout the British Isles have been massive and have a long history. The period of the Industrial Revolution especially saw the immigration of large numbers of both Scots and Irish to England creating "English" families with Irish and

¹⁰ Note that the spelling of Roach has become an accepted Acadian spelling due to the presence of an Irish root to the name as well.

Scots surnames. This is likely one source of those of unknown ethnicity in the death registers. The Irish prefix “O” is common to some names and also means “son of” in Erse. Doyle, Fitzgerald, Kavanagh, Kehoe, and Murphy all appear as easily distinguishable names not usually found elsewhere. However, there is likely an under-representation of Irish using this method considering the point made about population movement in the British Isles. It should be noted that Irish and Scots names are also Anglicized but have become more standardized after hundreds of years of Anglocentric rule.

Mi’kmaq names are fairly easy to spot in the historic record, but their bearers are often elusive. Very few Mi’kmaq appear in the death registers of any of the counties. Recorded Mi’kmaq deaths account for only 0.7% of all deaths registered in all four counties between 1864 and 1877. However, according to the 1871 census figures, they represent only 0.63% of the population. So, despite the death records revealing very few Mi’kmaqs, their numbers are not under-represented according to their overall presence in the general population. The surnames of Mi’kmaqs are usually quite different from Acadian, Scots or Irish names. Names such as Cremo, Googoo, Joe, Michael and Sylliboy are just of the few more obvious surnames. However, there are many others, such as Chaumable, Gabriel, James, Newell, Philips and Richard, that can easily be confused with Acadian or English surnames.

There is some reason for making an assumption of ethnicity based on father’s surname and it is possible to check the likely amount of error in assigning ethnicity in this way. The percentage of out-marrying can be used to roughly determine how often

ethnicity is incorrectly assigned based on surname. Out-marrying is defined as marrying outside of one's ethnic group. For this project, an example of this might be an Acadian bride and a Scots groom. After ethnicities were assigned in the death registers, one county, Inverness, which had decent-sized populations of both Scots and Acadians, was checked for errors in the assignment of ethnicity. Out of 131 married or widowed Acadian females in all four counties, only eight, or 6.11%, were found to have non-Acadian married surnames. Out of 590 married or widowed Scots females in all four counties, only 44, or 7.46%, were found to have non-Scots married surnames. Interestingly, both groups most frequently married men with English surnames or with surnames of other unknown ethnicities, not each other. Therefore, the two principal groups of study, the Acadians and the Scots, are not likely to be confused for one another using this method.

Despite this small percentage of out-marrying, married females were not assigned the ethnicities of their husbands for a simple reason best explained through example. This method was chosen for future research consideration where more than one generation might be concerned. Let us visit the example of a marriage between an Acadian bride and a Scots groom. She is found in the historic record with no mention of her maiden name so we assume she is Scots. Her sons all marry Acadian women. Are the children of those sons Acadian or Scots? If we assign the original bride an ethnicity of "Scots" then her grand-children, who are three-quarters Acadian, will be considered Scots through this system. However, if we assign the original bride as "Unknown" her sons must also receive that designation. The Unknown category must suppress all other ethnicities in

order for the system to remain as close as possible to the true distribution of ethnicities in the population. The “Unknown” category is tedious because it forces us to eliminate part of our sample, but it maintains the integrity of the actual population until a more fitting ethnicity can be determined.

The second assumption made in assigning ethnicities to individuals in the historic record is whether the person or the society at large would recognize the individual as such. There is little doubt that those who emigrated from Scotland or Ireland would have been perceived as Scots or Irish respectively. Acadians too had an established lineage and history in the area and viewed each other as such. Undoubtedly the Mi’kmaq had long been established before anyone else arrived from Europe. However, what did the many groups think of each other?

The Irish and Scots may have been grouped together by the Acadians and the Mi’kmaq. They looked similar to the English who had arrived to claim Cape Breton earlier and many spoke English. However, one notable difference may have been the high number of Roman Catholics in the group. This difference would have been noted by the Acadians and perhaps even the Christianized Mi’kmaq. The linguistic accents of each group when attempting to speak English as a second language would have also been apparent to the others. The Acadians may have been viewed by the Irish and Scots as French in general. However, their establishment on Cape Breton would have clearly marked them as pioneers on the landscape. To the Mi’kmaq, the Acadians were trading partners and allies against the English for a long time, as discussed in Chapter 2. Their role would have been clear to the Mi’kmaq.

In mixed marriages, this assumption of assigning ethnicity based on paternally-derived surnames gets quite murky. Although the small amount of this occurring for both the Acadians and Scots has been demonstrated, this is only one generation. What did it mean to an individual who had one out of four grandparents who was Mi'kmaq and the other three Acadian? Was it significant? Did it define them as a member of a third group, the Métis¹¹?

These are important anthropological questions to ask but without documentation of how individuals felt, they cannot be adequately addressed here. The last group, the Métis, most certainly did and still does exist today. However, there are no distinct surnames that arose from such a cultural group. The fact that the languages of the three main groups of the Acadians, Scots and Mi'kmaq still thrive in Cape Breton today suggests that the groups had separate identities that maintained an internal cohesiveness.

Though there is no way to be absolutely certain of the assignment of ethnicity to individuals, we can compare the aggregate numbers of those assigned to individuals in the death records with those found in the 1871 census where individuals identified themselves as belonging to one or another group. The percentages for all four counties are shown below in Table 8.2. Note the proportional differences in the English and Irish numbers. This is largely due to the fact that English and Irish names are difficult to distinguish with certainty in the historic record. Some of the names are shared by other cultural groups, and with each other and were likely classified as “Unknown” when ethnicities were assigned.

¹¹ Métis is a French word applied to an individual of mixed Aboriginal and European (usually French) heritage. It is similar to the Spanish word *mestizo*.

Note the large percentage of “Unknown” in the list of dead. This demonstrates that the assignment of ethnicity to the dead by surname is not a perfect method. If a cautious, conservative approach is taken, a large percentage of the dead will always end up in this category because of the inherent problems mentioned above. However, the method is fairly accurate.

Table 8.2. Numbers of dead and living by ethnicity.

Ethnicity	1864-1877 Dead		1871 Census Living	
	n	%	n	%
Acadian	1,510	16.22	10,864	14.39
Scots	5,227	56.13	50,111	66.39
Irish	323	3.47	7,311	9.69
Mi'kmaq	65	0.70	473	0.63
English	68	0.73	6,013	7.97
Other	65	0.70	711	0.94
Unknown	2,029	21.79	0	0.00
Total n	9,287		75,483	

There is an additional problem with this method. The very inspiration for this project was that undue numbers of Acadians seemed to be dying from scarlet fever during the 1875-1877 epidemic, and one of the goals of this project was to demonstrate why. When cultural groups or ethnic groups seem to be experiencing disproportionate impacts of disease or other causes of death, comparison to the living population will of course not match exactly. It is a bit of a conundrum for the historic researcher to solve this problem and the only certain way is to find every deceased individual in the census and identify their ethnicity this way. This undertaking is a difficult task and is beyond the scope of the current project.

8.2 Geographic Plotting

One of the goals of this project was to view the spread of scarlet fever over the Cape Breton landscape as a whole. To aid in this task the A. F. Church maps mentioned previously were reviewed. The maps show the buildings and names of every proprietor on the island by identifying the buildings with a small square or dot and the names by the first initial and surname of the proprietor. The original goal of this project was to identify the household of every scarlet fever victim and create a series of snapshots at one week intervals of the advancement of the epidemic. These could then be run together into a PowerPoint® presentation and played as a movie.

The difficulties of accomplishing this task became evident early on. To have accurately accomplished this task on this scale would have required the positive identification of over 122,000 family homes on Cape Breton Island. The reason that such an extensive identification project would have to be undertaken involves several factors. The first is that the very nature of the population is such that the occurrence of the same first initial and surname of a proprietor in the same census subdistrict is very common. For example, the Acadians in the Chéticamp region share about 15 surnames. Being devout Catholics, and given the naming trends of the 19th century, they also share a total of about 75 first names with a handful of these being very popular. Therefore, the names of Jean, Joseph and Julien Deveaux will all appear as J. Deveaux on the map.

The second is that there are no addresses on either the census or the maps that identify the houses. The census takers simply seemed to have walked to each house in the manner that seemed most logical to them with some geographic flow. Though it is

possible to follow some of the patterns left by this method of census-taking, there are discrepancies that confuse the reader.

The third problem is the discrepancy of time itself. The Cape Breton maps were likely completed sometime between 1883 and 1888. The deaths of the epidemic victims occurred anywhere from seven to 12 years prior to this information. The closest census that can be used to follow the individuals is the 1881 census. Coordinating information from these three times does not always result in perfect matches and discrepancies can cause confusion, leaving holes in the transcription.

The fourth problem is that the total of all scarlet fever deaths and other possible categories of strep infection is a mere 432. Finding these 432 without transcribing the locations of all 122,000 homes first is like the finding proverbial needle in a hay stack. Transcribing the homes first would allow for sorting and searching in spreadsheet format that is nearly impossible to do with a disorganized and random arrangement of homes on a map.

Although accomplishment of this task is feasible, many more hours of analysis and transcription would be required. Consequently, this activity is being left for future research. Once accomplished, however, it will serve as an excellent tool for tracking any of the many epidemics present in the historic record for Cape Breton. Including a GIS analysis within the framework of the task, so that geographic features and roads could be included, will also allow spatial distancing analyses to be done.

For the present project, aggregate totals for the census subdistricts are provided instead. These demonstrate the clustering of deaths in the county of Inverness and

Chéticamp in particular. High mortality (death) and high morbidity (illness) are usually closely linked, so that those areas with high mortality probably also had high levels of morbidity. The aggregate totals identify the areas which likely experienced high morbidity as indicated by high mortality. They also use the same subdistrict categories as the 1871 census tables making comparison to this source easy. A map of these is shown in Figure 8.1.

8.3 Determining the Presence and Extent of Streptococcal Mortality in Cape Breton

The process of determining whether someone died from scarlet fever or some other form of streptococcal infection using the historic record is a difficult undertaking. At one extreme, a conservative approach stipulates that only those deaths explicitly marked as being caused by scarlet fever or some other streptococcal infection should be counted. This method creates a large number of falsely unclassified deaths that are too vague for classification or simply not mentioned. At the other extreme, a liberal approach allows the researcher to determine the actual cause of death of the individual, even when the category seems to defy classification, such as “the common cold”. This method lumps too many vague causes of death into categories that they might not necessarily belong in. A middle road between these two must be used that is sensitive to the historic context of medicine and the temporal swath of the epidemic in question.

Interpreting cause of death information to try to determine actual infective agents is a delicate task. Scarlet fever is only one of many manifestations of the group A streptococcal agents. Others are included in Table 8.3 below.

“possible scarlet fever” include “infantile fever”, “brain fever”, “canker rash”, “cerebro-spinal meningitis”, “erysipelas”, “fever”, “swelling in the head”, “hydrocephalus”, “inflammation of the brain”, “meningitis”, “quinsy”, “rash”, “red fever”, “slow fever”, “sore mouth”, “sore throat” and “water on the brain”. All of these had to be combined with an age of 15 or under to be considered a possible case of scarlet fever. Though adults are known to contract and die of scarlet fever, a conservative approach was taken to age for this method.

Table 8.3. Other manifestations of *S. pyogenes*

Easily identified in historic record, discrete causes of death	Rarely fatal, not usually in historic record	More recently recognized
Puerperal fever	Strep throat	Mastoiditis
Septicemia	Strep. skin infections (impetigo and pyoderma)	Otitis media
Erysipelas	Cellulitis	Wound infections
Pneumonia	Peritonsillitis	Necrotizing fasciitis
Rheumatic Fever		Strep. toxic shock

Those causes identified as “other strep infection” included “billious fever”, “brain fever”, “dropsy”, “erysipelas”, “fever”, “inflammation of brain”, “puerperal fever”, “quinsy”, “slow fever”, “sore throat” and “swelling in the head”. There is an overlap with the previous category due to some of these causes (namely “brain fever” and “slow fever”) affecting both juvenile and adult individuals. Because scarlet fever as a manifestation is considered to be restricted to juveniles in the literature, only juveniles with these two specific causes of death were placed in the category of “possible scarlet

fever” while adults with these two specific causes of death were placed in the category of “other strep infection”.

The aim is not to resolve every individual case in order to confirm or deny the role of a GAS infection in the individual’s death. Rather, it is more important to get a general picture of epidemic spread facilitated by this type of investigation that considers all possibilities, knowing the nature of historic record keeping and 19th century medicine. It is deemed essential to the understanding of an epidemic to consider these ethnohistoric factors that have shaped the ways in which this information has come down to us and the ways in which we interpret it.

8.4 Sensitivity Analysis from Modeling

The mechanics of the mathematical model used for this project have been discussed in some detail in Chapter 6. Sensitivity analysis has been left for this section of the discussion because it concerns some specific methodological steps to ensure that accurate results are obtained. Sensitivity analysis simply involves detecting patterns in the factors that have been included in the model. These patterns inform us about how the factor affects the entire workings of the model and what this might imply about the epidemic under study.

Sensitivity analysis must be done in a precise manner that ensures what is being studied is isolated from the rest of the mechanics of the model. In order to accomplish this, all of the other factors that the modeler can change through input into the model are kept constant. The factor being analyzed is then put through its range of possible values

to see what effects those changes produce in the model.

Any random factors must be reduced to one simplified constant number. This may be an average or it may mean going through more than one run of analysis that covers a range of constants that may be equally possible. For instance, the model in this project includes a rate of death for scarlet fever, once contracted by a infective juvenile, that is randomly chosen between 5% and 30%. In order for *other* factors to be analyzed correctly, this first factor must be held constant. However, there is really no good average to assign for the rate of death for scarlet fever because it depends on the virulence of the disease and the population's previous exposure to it. Instead, one could hold the rate of death constant at 5% and then at 30% to examine the extremes of the range of this factor. Any of the *other* factors that then need to be analyzed would have to be run through their respective ranges for each constant used for these random numbers.

When changes occur in the model due to sensitivity analysis, the type and amount of change indicates the role that the factor plays in the model. It may at first seem that this is a fruitless effort because the role that the factor plays is already programmed into the model by the modeler. This is a deceptive over-simplification. Because of the complex nature of the factors that comprise a mathematically based computer model, and because of the inherent complexity of the calculus equations themselves, each factor's effects on the model are often hidden and can only be explored through sensitivity analysis. For instance, Sattenspiel and Herring (2003) have found that the timing of implementing quarantine for influenza very specifically affects its effectiveness. Without sensitivity analysis, this effect would not have been realized.

A model is often built and re-built on the basis of preliminary analyses of the factors built into the model . The appropriate value or range of values for a factor may not be known until sensitivity analysis is done. For instance, the number of contacts per unit time (κ) and the probability of transmission due to contact (τ) are often not known before creating the model itself. A rough estimate may be obtained from similar studies or from common sense but determining a reasonable range is often done through a preliminary type of brainstorming sensitivity analysis.

Finally, it should be noted that the results of sensitivity analysis usually do not speak for themselves. Simply discovering that factor A affects factor B in an X to Y range of values needs some interpretation. We have to bring these factors back to the “field” and ask ourselves what the implications of these effects really mean to the “participants” in the model. For instance, what does it mean if the number of dead increases proportionally with the number of contacts per unit time? We have to read into the context of these factors and interpret their meaning for all participants in the model. We must also interpret the model as a whole. Does the alteration of a specific factor reduce the number of deaths significantly? This is one tool that can be used to determine the relevant factors in driving a real epidemic so that we can react more effectively to similar situations.

Chapter 9: Results and Analysis

The analyses of the data that have been transcribed and modeled provide a picture of what occurred during the 1875-1877 scarlet fever epidemic of Cape Breton Island. These data show that 77 individuals are listed as having died from *scarlet fever* on the island of Cape Breton. When the data are re-interpreted with the more liberal approach to causes of death explained previously, the number of dead from the streptococcal epidemic balloons to 241. The deaths occurred in 44 of the 55 sub-districts (80%) with major concentrations found in the sub-districts of Chéticamp, Mabou, Margaree, Baddeck, Lingan Mines and Sydney Mines. There are strong indications in the data analyses that implicate household types as a determining factor for the arrival of the epidemic as well as the severity of its impact.

9.1 Differences in the Effects of the Epidemic on the Two Major Ethnic Groups

When first glancing at the historic data, one is immediately struck by the odd coincidence that the majority of victims from this epidemic were Acadians and that their Scots countrymen did not suffer the same impact from the epidemic. The population figures can be consulted to see if there is an overall trend in increased Acadian death for all causes before taking the historic record at face value.

The percentages of each ethnicity were given previously in Table 8.2. For ease of reference these are included in Table 9.1 with three columns added to show the scarlet fever deaths and deaths caused by other strep-related infections, including all those

mentioned for the “possible scarlet fever”, “rheumatic fever” and “other strep infections” categories mentioned in Chapter 8. This table shows first that Acadians and Scots differ in their overall death proportions for all causes (16.22% vs. 56.13%). These numbers are similar to the census proportions of living, though the Scots are under-represented amongst the 1864-1877 dead and the Acadians are over-represented. When we move to the column for causes of death explicitly stated as “scarlet fever”, we see a dramatic difference in the experience of the Acadians and Scots. Despite making up less than 20% of the population, 47.5% of deaths from scarlet fever are attributed to Acadians. In addition, despite making up over ½ of the population, only 28.75% of deaths from scarlet fever are attributed to Scots.

Table 9.1. Ethnicity of all 1864-1877 dead, 1871 census of the living, scarlet fever dead of 1875-1877, all other possible strep deaths of 1875-1877 and total of both scarlet fever and other possible strep deaths of 1875-1877.¹²

Ethnicity	1864-1877 Dead		1871 Census Living		SF Deaths		Other Possible Strep Deaths		Both SF and Other Possible Strep Deaths	
	n	%	n	%	n	%	n	%	n	%
Acadian	1,510	16.22	10,864	14.39	38	47.50	59	16.76	97	22.45
Scots	5,227	56.13	50,111	66.39	23	28.75	203	57.67	226	53.31
Irish	323	3.47	7,311	9.69	1	1.25	6	1.70	7	1.62
Mi'kmaq	65	0.70	473	0.63	1	1.25	2	0.57	3	0.69
English	68	0.73	6,013	7.97	0	0.00	0	0.00	0	0.00
Other	65	0.70	711	0.94	2	2.50	3	0.85	5	1.16
Unknown	2,029	21.79	0	0.00	15	18.75	79	22.44	94	21.76
Total n	9,287		75,483		80		352		432	

¹² These figures are for all four counties in Cape Breton Island.

An interesting thing happens when we examine other causes of death that could possibly be caused by a streptococcal infection. The criteria for evaluating each listed cause of death are applied equally regardless of ethnicity. What emerges from the data is that a large percentage of the other possible causes of death occur in the Scots and not in the Acadians. This amount is so great that when the numbers of all four categories are combined the percentages of deaths possibly caused by streptococcal infections of unspecified manifestation come close to being equal to the aggregate percentages of deaths for each ethnic group. This suggests that the experience of the epidemic was still different for the two groups but perhaps not to the extent suggested by the initial reading of the historic record. The difference may even only be one of terms used by each culture to identify the same phenomenon with the term “scarlet fever” being a more common use among Acadians and the term “rheumatic fever” being a more common use among the Scots.

To examine this difference statistically, we can isolate the two groups and perform a chi-squared (χ^2) test on the data. Table 9.2 presents a comparison of the 1875-1877 deaths from possible streptococcal causes to all other causes of death from those same years for the Acadians and Scots. This table shows that the results of the Mantel-Haenszel χ^2 test equal 19.54 and are significant at a p -value of less than 0.001. Table 9.3 presents a slightly different comparison of the 1875-1877 deaths from possible streptococcal causes to the living populations derived from the 1871 census for the Acadians and Scots. This table shows that the results of the Mantel-Haenszel χ^2 test equal to 31.68 and are significant at a p -value of less than 0.001. Both tables show that

Acadians are more than two times more likely to have died of streptococcal causes than Scots by the odds ratios for each test.

Table 9.2. Comparison of all possible streptococcal deaths to deaths from all other Causes during 1875-1877¹³.

	Acadians	Scots
All possible strep deaths (1875-1877)	60	116
All other causes of death (1875-1877)	276	1130

Table 9.3 Comparison of all possible streptococcal deaths to the living population from the 1871 census¹⁴.

	Acadians	Scots
All possible strep deaths (1875-1877)	60	116
Living population (1871)	10,864	50,111

Tables 9.4 and 9.5 compare the deaths from scarlet fever only to the dead of 1875-1877 and the living population of 1871. The results of the Mantel-Haenszel χ^2 test in Table 9.4 are equal to 69.57 with a p -value of less than 0.001. The results of the Mantel-Haenszel χ^2 test in Table 9.5 are equal to 89.86 with a p -value of less than 0.001. In both cases, the odds ratios show that the Acadians are about 8 or 9 times more likely to have died from deaths caused by “scarlet fever” proper than the Scots.

¹³ A Mantel-Haenszel χ^2 test was used with the following results: $\chi^2 = 19.54$ ($p < 0.001$). The odds ratio comparing Acadians and Scots was 2.12.

¹⁴ A Mantel Haenszel χ^2 test was used with the following results: $\chi^2 = 31.68$ ($p < 0.001$). The odds ratio comparing Acadians and Scots was 2.39.

Table 9.4. Comparison of 1875-1877 “scarlet fever” deaths to deaths from all other causes for those years¹⁵.

	Acadians	Scots
SF deaths (1875-1877)	38	20
All other deaths (1875-1877)	276	1130

Table 9.5. Comparison of 1875-1877 “scarlet fever” deaths to the living population from the 1871 census¹⁶.

	Acadians	Scots
SF deaths (1875-1877)	38	20
Living population (1871)	10,864	50,111

Three factors must be kept in mind when viewing these data. The first is the margin of error created by the large number of people of unknown ethnicity. Looking back at Table 9.1 we can see that the English and Irish are massively under-represented in the death registers. The Scots are somewhat under-represented as well. This suggests that those of unknown ethnicity in the death registers are comprised mostly of English, Irish and Scots who could not be identified as such. This has implications for the accurate identification of Scots in the current project, especially since they are one of two main groups being studied. Because of the higher English presence in Ireland and lack thereof in the Western Isles of Scotland, there is probably a low percentage of Scots that have not been identified amongst the dead, likely less than 5%. That is, it is less likely that the unknown names from the British Isles are actually Scots rather than Irish.

¹⁵ A Mantel-Haenszel χ^2 test was used with the following results: $\chi^2 = 69.57$ ($p < 0.001$). The odds ratio comparing Acadians to Scots was 7.78.

¹⁶ A Mantel Haenszel χ^2 test was used with the following results: $\chi^2 = 89.86$ ($p < 0.001$). The odds ratio comparing Acadians to Scots was 8.76.

A second factor that must be considered is how the different ethnic groups break down in terms of their cause of death reporting in general. Table 9.6 below shows those who have died from unknown causes by ethnicity. Causes of death classified as “unknown” include many different categories, including a few illegible entries and those with strange causes, such as “continued fear”, “will of god”, “visitation of god” and “rapture”, as well as unheard of causes, such as “sarihus mamal”, “sclatica” or “macmoptysis”. However, the two most common types of entries that fall into this category are those where either no cause is actually listed or those where a cause of “old age”, “sudden”, “decline”, “decay”, “lingering illness”, “killed in a mill” or some other variant indicates the age of the person or other circumstances as the ultimate surrounding conditions leading up to the true (unknown) cause of death.

Table 9.6. Deaths within an ethnic group reporting an “unknown” cause of death.

Ethnicity	1864-1877 Deaths from “unknown” causes		Deaths from all causes
	N	% of total deaths	N
Acadian	724	47.95	1,510
Scots	1,816	34.74	5,227
Irish	161	49.85	323
Mi’kmaq	9	13.85	65
English	35	51.47	68
Other	14	21.61	65
Unknown	661	32.58	2,029
Total n	3,420	36.83	9,287

We can see from Table 9.6 that the percentage of reported unknown causes of death varies among ethnic groups. Among the English this value exceeds 50%, although it must be emphasized that the size of the English population was very small. Most of the

others are in the 30% to 50% range. The Mi'kmaq seem to be the best at reporting a cause of death that is interpretable by a modern researcher. The reasons for this are unknown. The Acadians and Scots, who are the main focus of this project, are somewhat different from each other, with almost 48% of Acadian deaths bearing a cause of death that could not be interpreted while only 35% of Scots deaths have this problem associated with them.

In both the Acadian and Scots cases, researchers must be guarded about such a finding. It suggests that there may be a great deal of under-reporting of the very disease being investigated. This may be especially true for epidemics. Epidemics are, by definition, characterized by the rapid spread of a disease in a short span of time. When high mortality is associated with an epidemic, this type of event can overwhelm the society at large and interfere with the social structures in place for burying the dead. Recording these deaths likely became routine for the registrars and the quality of reporting could have swung to either extreme (thorough or incomplete) during such an event. The scenario where many individuals are dying rapidly of a known cause can lead to thorough documentation of the disease. However, streptococcal epidemics are characterized by many different manifestations that were more elusive to 19th century medicine and may have resulted in under-reporting of cause of death information.

The third factor that has to be considered when viewing these data is the different population structures of the different ethnic groups. A population pyramid comparing the Acadians and Scots is shown below in Figure 9.1. An accurate representation of these data can only be obtained by a transcription of all 55 census sub-districts. There are

summaries of the ages of the people in the 1871 census summary tables, but these use different age categories (e.g., 0-1, 1-6, 6-11, 11-16, 16-21, 21-31, etc.) that are no longer standard demographic categories nor consistent with the age categories used in this study. Therefore, due to the massive amount of transcription required to remain consistent, two of the middle-sized communities were chosen as index communities for Acadians and Scots and were transcribed in order to obtain population estimates. The communities of Judique and Chéticamp were chosen for this purpose. Although both the 1871 and 1881 censuses for these communities have been transcribed, the 1871 census gives a picture of the population before the 1875-1877 epidemic. The age categories shown are standard demographic five-year intervals. The age categories represent intervals from the lowest age to the highest age one day short of the next category.

The reason that the population pyramid of the living must be considered when viewing data regarding the dead is because the very demographic structure of a population can affect how many people are dying. A growing population with an established structure is indicated by a wide base with a regular slope up to the oldest age categories. A newer population comprised of recent immigrants has a more erratic pattern with a narrower base and occasionally bulges in certain age groups where waves of young individuals made the trip to the new location.

The pyramid in Figure 9.1 shows these two shapes. The Acadian side, the longer-established population on Cape Breton, shows a very wide base with a steady slope up to the oldest age groups. There is a slight bulge in the 20 to 29 year age groups. It is unknown why this slight difference exists but it may be due to a slight population boom

20 to 25 years before the 1871 census (1846-1851). The Scots side of the graph shows a more erratic pattern. There is a much narrower base indicating fewer juveniles in the population. There are also slight bulges at 15 to 19, 50 to 64 and 70 to 84. These may all indicate true population booms due to stages of immigration. The start of the Scottish immigration to Cape Breton was underway by the 1820s and began to wane in the 1860s. The members of the older age groups could certainly represent the young, healthy survivors of those journeys while the bulge in the youngest group may represent their children.

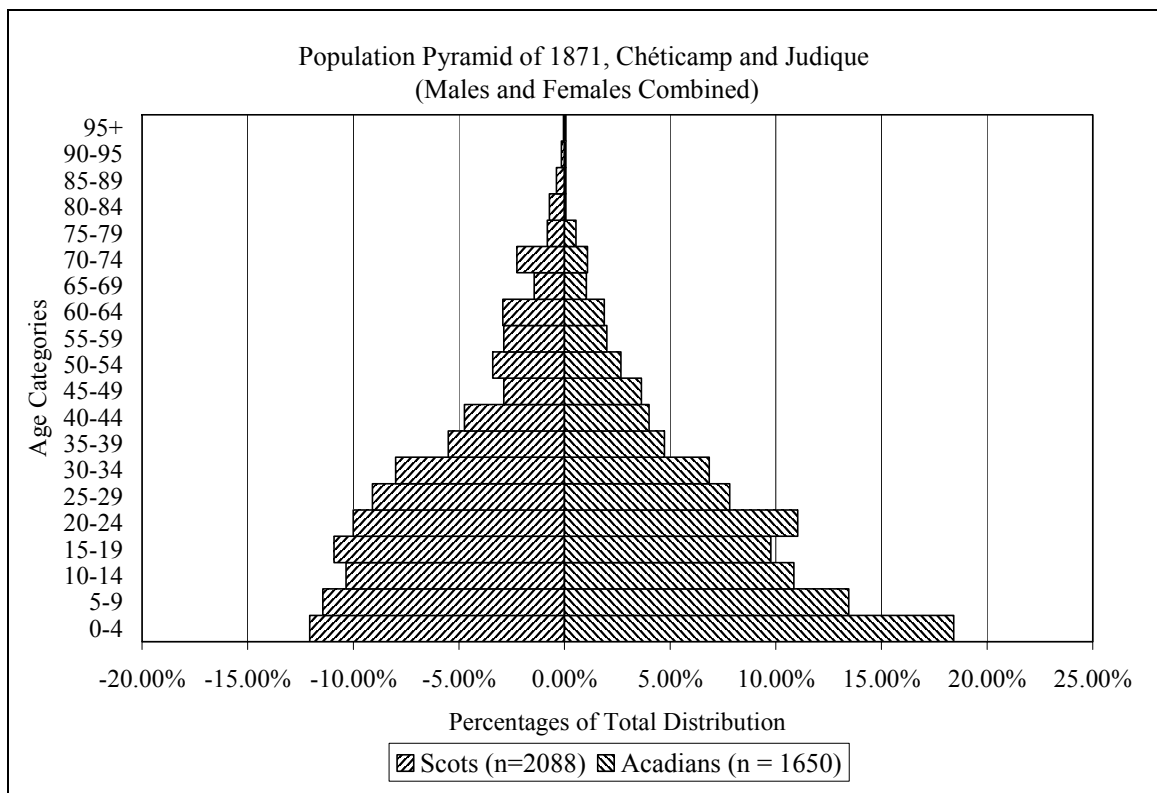


Figure 9.1. Population pyramid of Judique and Chéticamp based on the 1871 census. This represents approximately 5.51% of the total population of Cape Breton Island at that time. The negative percentages for the Scots are an artifact of the software used to create the graph.

It is also possible that age-rounding has occurred (Preston et al., 1999; Duncan-Jones 1990). This phenomenon occurred most commonly in history when individuals were asked to give their age on a census or some other official form. Usually being unaware of their exact year of birth (due to age itself, lack of education, etc.) individuals often estimated their age instead. This estimation results in a rounding of the age. The rounding is different in every culture. In European cultures, a rounding of the age to end in a “0” is most common, ending in a “5” being less common but also documented. It is also more commonly found among adults since children’s birthdays are most recent, more celebrated and more important for the various legal and religious ceremonies that occur with age advancement throughout childhood. This phenomenon is unlikely to have occurred in the Scots data shown in Figure 9.1. The use of five-year age intervals ensures that each interval contains one age ending in a “0” and one ending in a “5”. Though the phenomenon is documented as being more common among adults (Duncan-Jones, 1990:79-92), the Scots population does not show any dramatic problems of this nature.

These data show that the Acadian population is younger, as would be expected from the cultural context of the two groups. The implication of this may be that the younger Acadian population is contributing partially to the inflated numbers of deaths seen in the Acadians in comparison to the Scots and their respective living populations in Table 9.1. One might think that an older population would contribute more to this type of death inflation but the opposite is actually true, especially for historic populations, because of high levels of infant and child mortality, a situation that didn’t change until 20th century medicine. Table 9.7 below demonstrates that this was in fact a problem for

the Acadians.

Despite making up only 18.43% of the Acadian population, Acadians under the age of five account for 46.19% of all Acadian deaths. Scots, on the other hand, have a much different picture. Children under the age of five comprise 12.07% of the Scots population and account for 26.36% of all Scots deaths. This suggests that, indeed, the Acadians do have a more significant problem with infant and childhood mortality than the Scots.

Table 9.7. 1871 census living and 1864-1877 vital register dead by age category and ethnicity.

Category	1871 Census Acadians		1871 Census Scots		1864-1877 Dead Acadians		1864-1877 Dead Scots	
	n	%	n	%	n	%	n	%
<1	97	5.88	58	2.78	326	31.90	515	17.54
1-4	207	12.55	194	9.29	146	14.29	259	8.82
5-9	222	13.45	239	11.45	74	7.24	119	4.05
10-14	179	10.85	216	10.34	36	3.52	53	1.81
15+	945	57.27	1,381	66.14	440	43.05	1,990	67.79
Total n	1,650		2,088		1,022		2,936	

The higher infant and childhood death rates of the Acadians in comparison to the Scots can further be shown by comparing the death rates for the two groups. Figure 9.2 shows the death rates for Acadians and Scots for all causes of death other than all possible streptococcal causes for 1875-1877. Figure 9.3 shows the death rates for Acadians and Scots for all possible streptococcal causes. Age-specific death rates were calculated based on the living populations of the 1871 Judique and Chéticamp censuses. Acadians and Scots from both communities were combined to give a picture of

representative sample of the Acadians and Scots. The combined Judique and Chéticamp rates were then used to standardize age-specific death rates for the whole population of Cape Breton Island. The percentages for each age category in the Judique and Chéticamp samples were applied to the Acadian and Scots populations of *all* of Cape Breton Island. The resulting figures were then used as the age-specific number of individuals at risk for epidemic deaths in order to calculate death rates per 1,000 in each age category. We can clearly see that the two populations look very similar for their non-epidemic death rates from Figure 9.2. However, Figure 9.3 shows the marked problem the Acadians have with higher death rates from strep-related diseases among the juveniles (0-14 age categories). The adult epidemic deaths are more erratic for both groups and don't demonstrate any clear patterns other than a general trend towards older adults dying from the epidemic rather than younger ones. The question then becomes, "What factors are contributing to this difference?"

9.2 Differences by Ethnicity or Differences by Occupation?

The most obvious answer to this question might be that there are genetic differences between the two populations that are causing differences in their experiences with childhood diseases. This might be an immune system deficiency or an inherited set of illnesses with genetic components further encouraged by very little out-marrying. There is research to suggest that differential susceptibility to some streptococcal toxins does exist in non-human mammals (Dale et al., 2002; Goldmann, Chhatwal et al., 2004, Goldmann, Rohde et al., 2004) and that factors contributing to resistance and

susceptibility naturally occur in some humans (Kotb et al., 2003; Voyich et al., 2004; Stenfors et al., 2003; Sun et al., 2004). However, it is unlikely that many genetic differences exist between these two populations if we consider their origins.

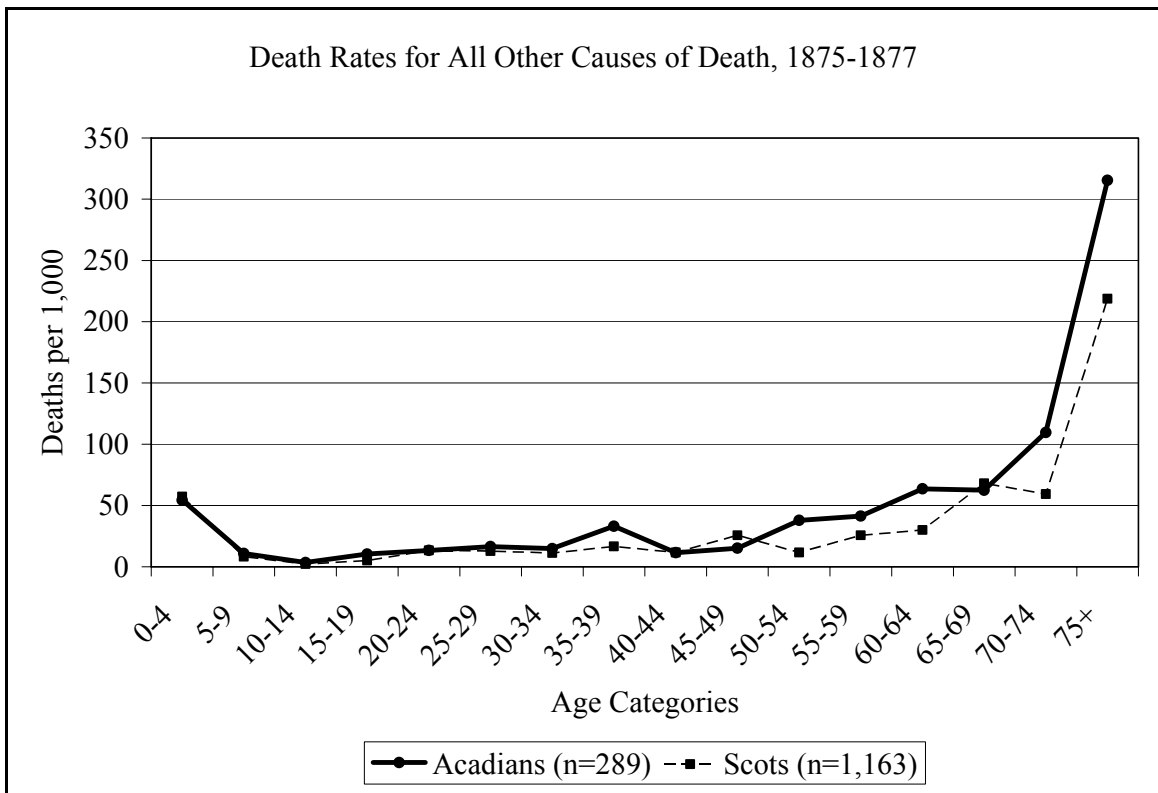


Figure 9.2. Death rates of Acadians and Scots for all other causes of death, 1875-1877. Based on those for whom ethnicity could be determined.

Despite the fact that their voyages to North America were separated by about 200 years, the Acadians and Scots are not that different. Both populations came from advanced imperial nations in Europe, albeit the populations that emigrated were likely from more rural areas of France and Scotland. Once they arrived, they found a more spacious landscape and pursued livelihoods in the manner they chose. Since both

populations originated in Western Europe, they may share a large percentage of DNA through their common heritage in the Celts of Western Europe.

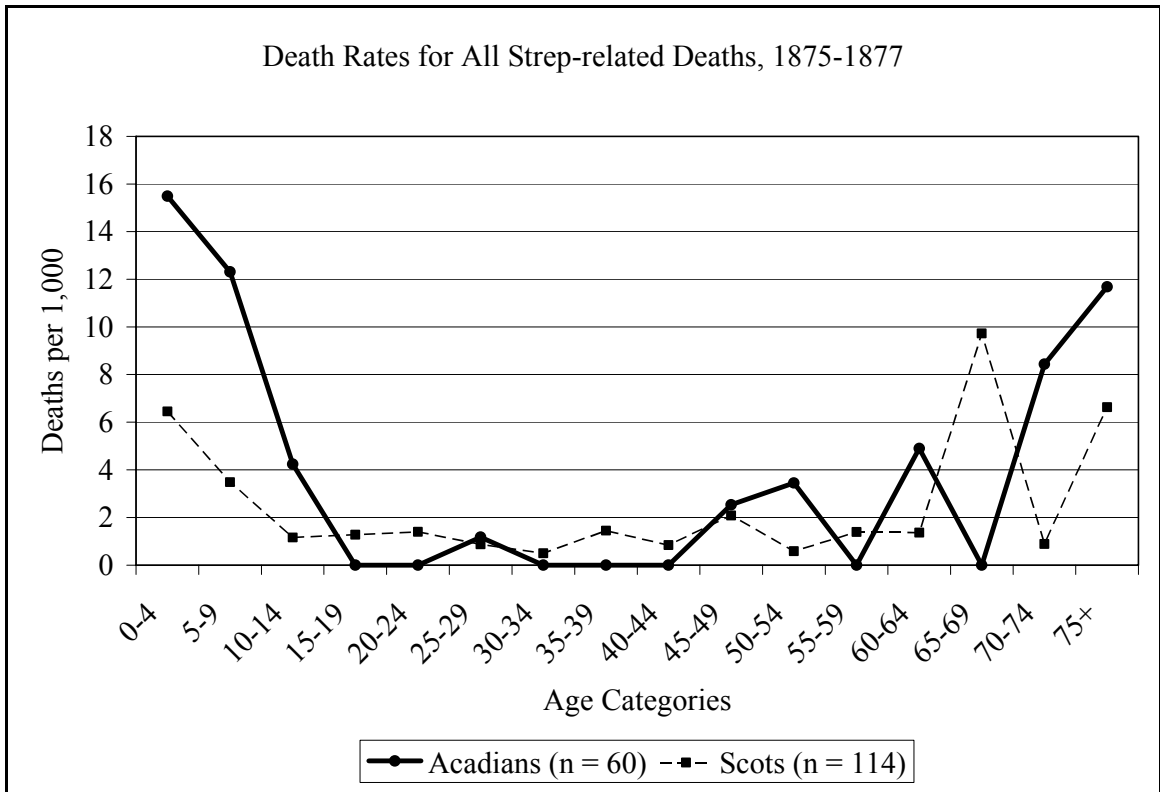


Figure 9.3. Death rates of Acadians and Scots for all streptococcal causes of death, 1875-1877. Based on those for whom ethnicity could be determined.

The disease experiences of the two populations from 1600 on *may* also have been quite similar. The Scots of Cape Breton largely emigrated from the Western Isles and Western Highlands of Scotland (Hornsby, 1992). These are more remote areas of Europe, though they were important industrialized areas by the 19th century because of the wool industry. The Acadians came from various parts of France, though most were from the western coastal provinces (Chiasson, 1998). They arrived in a part of Nova Scotia that

was similarly remote to western Scotland in that it had a low population density and was difficult to reach except by boat and railroad in the 19th century. It too was home to important industries of fishing and mining. Both populations were in remote areas that were parts of densely populated nations. Both were connected to the world economy through trade in their own goods. Though this does not expressly rule out the possibility that their disease experiences were different enough to cause a higher immunity to childhood diseases in the Scots, such is an unlikely scenario. A more thorough study focusing solely on the disease experiences of the two populations for many diseases would be needed and should even be extended to include the original populations or their descendants who stayed in France and Scotland, if at all feasible. To my knowledge, no such comparison study exists.

If the biology of the two populations cannot explain the differences in their experiences with childhood diseases, and with the 1875-1877 streptococcal epidemic in particular, there must be a more complex set of social or cultural factors that are contributing to the difference. To find the answer, we can look at the larger, simpler factors first and work to the more complex smaller factors from there. Within most epidemic models, there are only a few simple, large factors that have a major impact on the direction the modeling equations go. Factors such as the rates of transmission, carrier rates, rates of recovery and death rates have large biological components to them, though the last three also have smaller, complex social or cultural components to them due to previous exposure mediated through social or cultural circumstances. The compartments of the models that contain the susceptibles account for the starting populations and are,

therefore, determined by the underlying demography of the groups. This can certainly have an effect on the way an epidemic spreads, especially one like scarlet fever that kills mainly juveniles. This will be discussed later in the section on sensitivity analysis.

What we are left with are the contact rates. These can be altered by many smaller factors that combine in complex ways to produce differences in the average number of contacts per unit time that an individual is likely to experience. The Scots and Acadians share many social and cultural similarities. They are both largely Roman Catholic. More than 95 of Acadians are Catholics. Those Scots that are not Roman Catholic are usually Presbyterian or some other sect of Protestantism. This common factor means that most of the able population would attend religious services on Sunday. This may be considered an equalizing factor in terms of contact rates. The gathering of most of the population once a week provided an excellent opportunity for any infectious disease to circulate to new hosts until it was able to cycle throughout most of the members of the population who were susceptible to it.

We can also consider the population density on a household basis for the two communities of Chéticamp and Judique as index towns of Acadian and Scots settlements respectively. This is a simple calculation derived from the 1876 summary tables of the 1871 census presented in Table 9.8. These data show that the within-household population density did not differ much between the two populations. The Scots at Judique show a slightly lower average number of juveniles per household at 2.6 but a slightly higher overall number of people per household at 7.27 than the Acadians at Chéticamp. This suggests a higher average number of adults per household among the

Scots at Judique than among the Acadians at Chéticamp. However, these values are not different enough to affect the contact rates of the two groups. The data do support the previous findings of Table 9.7 and Figure 9.1 that show that the Acadian population overall is younger than the Scots with a wider base for its population pyramid than the Scots.

Table 9.8. Population density within households for the sub-districts of Judique and Chéticamp based on the 1871 census summary tables (Canada Dominion Bureau of Statistics, 1876).

	Judique (Scots)	Chéticamp (Acadians)
Juveniles	686	858
Adults	1,233	1,057
Total population	1,919	1,915
Inhabited structures	264	306
Average no. of juveniles per structure	2.60	2.80
Average no. of people per structure	7.27	6.26

In order to examine the size of the families of the epidemic victims, each victim's household was located in the 1871 and 1881 census. Because only the communities of Chéticamp and Judique have been transcribed, only the families of victims from these two communities could be investigated. Furthermore, because many of the deceased were children, they often did not appear in the censuses themselves. Therefore, detailed death records and birth records were required to search for the names of both parents of the deceased so that their family could be pin-pointed in the historic record. These records have only been acquired for the town of Chéticamp. Fortunately, a large number of the victims resided in Chéticamp. Each family was carefully examined for number of parents, number of siblings and number of other adults and juveniles. Older children in the family

at the time of the 1871 census made the task particularly difficult because of their potential to marry (or die) by the time of the 1881 census. Each of these cases was examined carefully and the marriage and death records for Chéticamp were checked thoroughly to determine the fate of each “missing” person in the 1881 census.

Of the 33 victims in Chéticamp, the families of 28 of the victims could be positively identified in both censuses. However, because of multiple members of the same family dying from the epidemic, this only represents a total of 21 families. There were five instances of two children in the same family dying and one instance of three children in the same family dying. The number of occupants represents the number of people present in the house at the time of the victim’s death. Among those families, the average number of occupants of the house was 8.14, slightly higher than the averages for both Chéticamp and Judique. What is more interesting is that the average number of juveniles per household, including the victims, was 5.62. This is more than double the averages of both Chéticamp and Judique. This is of particular note because it suggests that the total number of people in the house is less important than the ratio of adults to children in the house. These data suggest that the higher the number of children in the household, the more likely it was for the members of the household to die from scarlet fever or a related streptococcal illness. This is an expected finding since children have higher susceptibility due to under-developed immune systems and usually come into closer contact with others than adults do.

At least one other social factor breaks down roughly along ethnic lines and also affects the contact rates — occupation. If we know what both populations are doing one

day of the week, and it is roughly the same type of event in terms of contact rates, then we must ask what they are doing the other six days of the week that is going to affect their contact rates. The occupations of individuals are documented both in the death records and the censuses. It should be pointed out that the following discussion on this part of the project involves males only. Female occupations are mentioned so rarely in the record that even when a widow is the head of a family, I've chosen to refer only to the males who make up over 99 of those with occupations listed.

Reporting of an occupation for adult males is almost 100% in the censuses. However, this is not the case for the death records. Only 21 of 174 adults (12.07%) suspected of dying of a streptococcal infection have an occupation listed. The data for the dead adults is, therefore, not particularly useful. However, there is another source. Recall that the death registers list both parents' names and a parent's (usually the father's) occupation. This may seem like a spurious association to use since people are free to choose any occupation they want. However, a quick sorting of the 1871 census data for Judique and Chéticamp shows that of the 488 individuals who had an occupation listed who were not the heads of their own household, 375 or 76.84% of them had the same occupation as their fathers.

Farmers and fishermen together make up the majority of occupations listed in both locations, though in different distributions. If we isolate these two occupations and examine the likelihood of a child of a farmer or fisherman pursuing his father's trade, we find an even higher rate of consistency between generations. For Judique and Chéticamp in the 1871 census, over 88% of farmers and over 94% of fishermen had sons that carried

on those respective trades. Combined, over 90% of the farmers and fishermen had sons that carried on these trades. This suggests that using the father's occupation as an alternative to identifying the household type of an adult is reasonable especially where farmers and fishermen are concerned. However, these percentages were not applied in the model for a simple reason – the model uses a population level of modeling and so the population percentages of the two occupations in the population were the important figures to maintain, not the trends of individuals. The differences in the contact rates resulting from different household types is outlined in detail in Chapter 6.

We can statistically examine this trend in the association of ethnicity with household type (Table 9.9). These data are for all streptococcal dead of 1875-1877 who could be identified for both household type and ethnicity. Because one of the cells in the table is less than five, a Yates corrected χ^2 test was used to test the data. The results show a significant χ^2 score of 76.07 with a *p*-value of less than 0.001. The odds ratio indicates that Acadians who died of all possible streptococcal causes were over 293 times more likely to be fishermen than Scots who died of all possible streptococcal causes.

Table 9.9. Comparison of ethnicity to household type for those who died of all possible streptococcal causes from 1875-1877¹⁷.

	Acadians	Scots
Fishermen	40	1
Farmers	9	66

¹⁷ A Yates-corrected χ^2 test was used with the following results: χ^2 score = 76.07 (*p* < 0.001). The odds ratio comparing Acadians to Scots was 293.33.

In order to compare the rest of the living population with that of the dead in terms of household type, a protocol was designed to evaluate the household type of all households based on occupations of the head of the household and any sons where occupation is mentioned. A household that has one member with an occupation listed is considered to be that type of household. For instance, if the father is a fisherman, everyone in the household is considered to be a member of a fishermen household. In cases where there is more than one individual with an occupation listed, the contact rates of everyone in the household are affected. The person with the highest contact rate will “overrule” the person(s) with the lowest. Because the contact rates for fishermen are presumed to be much higher than those for farmers, any fisherman in a household will turn that household into a fishermen household, regardless of other occupations in the household.

Other occupations have been rated as either high or low in terms of contact rates. The list of these is extensive but the protocol for evaluating them can be summarized in terms of contact rates. Any occupation that involved a high number of contacts with people on a daily basis, by means of co-workers or customers, was termed a high contact job. This includes occupations such as butcher, clergyman, joiner,¹⁸ and merchant. Any occupation that involved a low number of contacts with people on a daily basis was termed a low contact job. This includes occupations such as a bookkeeper, accountant, cooper,¹⁹ and miller. These occupations tend to involve many fewer customers on a daily basis because they either don’t deal typically directly with the public *en masse*

¹⁸ A person who makes furniture and light woodwork.

¹⁹ A person who makes barrels.

(bookkeeper, accountant and cooper) and/or they produce large quantities of wares that can be sold through retailers (copper and miller). All of these occupations were grouped separately, though their contact rates may, in reality, be equal to those of the more common occupations of farmer and fisherman.

The 1871 census was examined to see how the living population breaks down by household types and ethnicity. Table 9.10 show that there are striking differences in how the two main ethnic groups break down by household type. The two household types of greatest concern are the farmers and fishermen. Almost 72% of all farmer households were Scots while only 24.55% were Acadian. The picture is completely reversed for fishermen. Over 87% of fishermen households were Acadian while only 12.22% were Scots. Note that only 19 individuals in the sample population could not be assigned to a household type. This suggests that there is a high correlation between ethnicity and household type.

Table 9.10. Percentages of ethnic groups by household types for the combined populations of Judique and Chéticamp based on the 1871 censuses of those sub-districts.

Ethnicity	Household Type									
	Farmer		Fisherman		Other Low		Other High		Unknown	
	n	%	n	%	n	%	n	%	n	%
Acadian	575	24.55	944	87.21	0	0.00	116	30.05	15	78.95
Scots	1,682	71.82	132	12.22	8	100.00	263	68.13	4	21.05
Irish	56	2.39	3	0.28	0	0.00	5	1.30	0	0.00
Mi'kmaq	0	0.00	1	0.09	0	0.00	0	0.00	0	0.00
English	29	1.24	0	0.00	0	0.00	2	0.52	0	0.00
Total n	2,342		1,080		8		386		19	

Recall from Chapter 2 that the Acadians arrived on Cape Breton after The Expulsion in 1755 to set up their own communities anew. They were relegated to the back-lands and coastal locations centered on Chéticamp and Arichat. Despite being largely farmers before The Expulsion in the French homeland and on the Nova Scotia mainland, this relocation forced the Acadians to embrace a fishing lifestyle. This was not a reluctant switch as the Acadians were glad to stay out of English reach in the more remote coastal areas. The Scots were invited over by the British government with the promise of land grants on tracts of arable farm land in the interior and along the rivers of Cape Breton. They were allowed to maintain the farming lifestyle they knew in Scotland. Considering the connection between fathers and sons for these two occupations and the low percentages of out-marrying demonstrated earlier, it is clear that maintenance of lifeways along ethnic lines would be a natural course for these two groups.

We must also consider what percentage of each household type is made up by each ethnic group. This information is presented in Table 9.11 below. These data show that a majority of Scots lived in farmer households (80.52%) while only 34.85% of Acadians lived in farmer households. Fishermen show an even larger difference, with 57.21% of Acadians living in fishermen households but only 6.32% of Scots living in fishermen households. Again, this demonstrates the reliance of the Acadians on the fishing lifestyle while the Scots are more drawn to farming. It is important to note also the representation of fisherman and farmer households in the general population. This sample shows that only 1,080 out of 3,835 (28.16%) of the general population are fisherman households while 2,342 of 3,835 (61.07%) are farmer households. There is

clearly a greater reliance on farming in the general population.

Table 9.11. Household types by ethnic groups for the combined populations of Judique and Chéticamp based on the 1871 censuses of those sub-districts.

Household Type	Ethnicity									
	Acadians		Scots		Irish		Mi'kmaq		English	
	n	%	n	%	n	%	n	%	n	%
Farmer	575	34.85	1,682	80.52	56	87.50	0	0.00	29	93.55
Fisherman	944	57.21	132	6.32	3	4.69	1	100.00	0	0.00
Other Low	0	0.00	8	0.38	0	0.00	0	0.00	0	0.00
Other High	116	7.03	263	12.59	5	7.81	0	0.00	2	6.45
Unknown	15	0.91	4	0.19	0	0.00	0	0.00	0	0.00
Total n	1,650		2,089		64		1		31	

Examining the household types of the scarlet fever dead will help us to make a comparison to the living population. The dead have to be considered differently because of the large number of unknown household types. This results from the fact that if the dead are not shown within a family context as are the living in the census, it is impossible to assign a person to their proper household type if the father's occupation isn't listed. Therefore, in calculating these percentages, unknowns were first removed from the totals. Both extremes of scarlet fever deaths only and those caused by all possible causes were included in this analysis. The data for scarlet fever deaths only is presented in Table 9.12 below. These data show that over 78% of all Acadian deaths were experienced by members of fisherman households while 72.22% of all Scots deaths from scarlet fever were experienced by members of farming households.

Table 9.12. Scarlet fever deaths for each ethnic group by household type for all counties of Cape Breton Island 1875-1877.

Household Type	Ethnicity									
	Acadian		Scots		Irish		Mi'kmaq		English	
	n	%	n	%	n	%	n	%	n	%
Farmer	7	21.88	13	72.22	1	100.00	0	0.00	0	0.00
Fisherman	25	78.13	0	0.00	0	0.00	1	100.00	0	0.00
Other Low	0	0.00	1	5.56	0	0.00	0	0.00	0	0.00
Other High	0	0.00	4	22.22	0	0.00	0	0.00	0	0.00
Total n	32		18		1		1		0	

We must also consider the deaths that were possibly caused by streptococcal agents combined with those caused by scarlet fever to cover the liberal end of the spectrum of interpretation. As shown in Table 9.13, these data indicate an even more dramatic experience for the Acadian fishermen, where 80% of Acadian deaths occurred in fisherman households while a slightly smaller 70.97% of all Scots deaths occurred in farmer households. Regardless of the slight differences, it is clear that the experiences of the two ethnic groups are polarized towards members of the two main household types. It is also worth noting that in both Tables 9.12 and 9.13, the majority of the remaining Scots deaths are experienced by households of other high contact occupation types, suggesting that Scots are also involved in occupations with high contact rates. However, these are not fishermen. They are a handful of other mixed occupations comprised mainly of carpenters and merchants.

Table 9.13. Deaths from all suspected streptococcal infections for each ethnic group by household type for all counties of Cape Breton Island 1875-1877.

Household Type	Ethnicity									
	Acadian		Scots		Irish		Mi'kmaq		English	
	n	%	n	%	n	%	n	%	n	%
Farmer	9	18.00	66	70.97	3	75.00	0	0.00	0	0.00
Fisherman	40	80.00	1	1.08	0	0.00	1	100.00	0	0.00
Other Low	0	0.00	3	3.23	0	0.00	0	0.00	0	0.00
Other High	1	2.00	23	24.73	1	25.00	0	0.00	0	0.00
Total n	50		93		4		1		0	

It is helpful to review the data from Table 9.1. Only 14.39% of the Cape Breton population is Acadian. When unknowns are excluded, over 1/3 (50/148) of the deaths from streptococcal infections were experienced by Acadians. The Scots comprise 66.39% of the Cape Breton population and 62.84% (93/148) of the deaths from streptococcal infections were experienced by Scots. The data for the Scots suggests, once again, that they had proportional experience of death from this epidemic while the Acadians had a disproportionately high number of deaths from this epidemic.

Finally, the clear association between household type and dying of streptococcal causes can be demonstrated statistically. Table 9.14 shows a χ^2 test for significance of this association. The Mantel-Haenszel χ^2 test results in a significant score of 39.12 with a p -value of less than 0.001. The odds ratio for the test shows that fishermen were over three times more likely to have died from streptococcal causes than farmers.

Table 9.14. Comparison of fishermen and farmers who died of all streptococcal causes to those who died of all other causes, 1875-1877²⁰.

	Fishermen	Farmers
Deaths from all streptococcal causes (1875-1877)	58	85
Deaths from all other causes (1875-1877)	194	883

9.3 Geographic Mapping of Epidemic Cases

All of the preceding data inspired both the geographic analysis and development of the computer-based model to further examine the effects of the epidemic and the factors contributing to it. The geographic landscape of Cape Breton is interesting in that what seemed to be a small, easily navigated place was in fact quite a difficult place to travel through, even up until modern times. The mountains, lakes, and rivers that dot the landscape made travel from place to place difficult. Though port towns were easily reached by sea, overland travel was slow and cumbersome. An anecdote from my own father's childhood serves to demonstrate the extent of this. He reminisced that when visiting his grandparents in Chéticamp, their trip from Sydney, a total of about 185 km (115 miles) would take all day by horse and carriage in the late 1930s /early 1940s. The terrain of this trip is still such that winding roads and massive elevation shifts make for a long journey of three to four hours by car between these two otherwise close locations.

The isolation of many towns from one another likely is what defined the parameters of the epidemic and determined whether or not it could spread from location to location. The cases are shown for all of the counties of Cape Breton Island by census subdistrict in Figure 9.4 below. The map has had the names of the subdistricts removed to

²⁰ A Mantel-Haenszel χ^2 test was used with the following results: $\chi^2 = 39.12$ ($p < 0.001$). The odds ratio for comparing fishermen to farmers was 3.11.

facilitate viewing of the stacked bars. The major cities have been included for orientation. Several trends can be observed with this map. The largest towns have the largest total number of streptococcal deaths. This is an expected feature of the epidemic since the largest towns have the highest population densities and usually the greatest amount of traffic (internal and external) as well. It should also be noted that the areas that were hit hardest by the epidemic are also those that lie along the coastal regions of the ocean and of Bras D'or Lake. These regions also contain the highest concentrations of fishermen. Areas of note that have high numbers of fishermen but do not seem to have been affected badly by the epidemic include the south-eastern regions around Louisbourg and the north-eastern shore of Victoria County. The southern region surrounding Arichat is one of the largest concentrations of fisherman yet only shows a moderate number of deaths in comparison to other regions. These regions were likely not protected somewhat from the epidemic because of their isolation. Though water travel to these regions is easier than land, the window of opportunity for this epidemic to spread via this means may have been narrow enough to have missed some of the major fishing areas. For example, fishing or mariner expeditions that may have regularly provided contact with individuals outside the community may have been out of sync with a chance for susceptibles to interact with an infective. Weather or simple timing could have created opportunities for an infective or group of infectives to be missed altogether and the chance may not have occurred again during the epidemic.

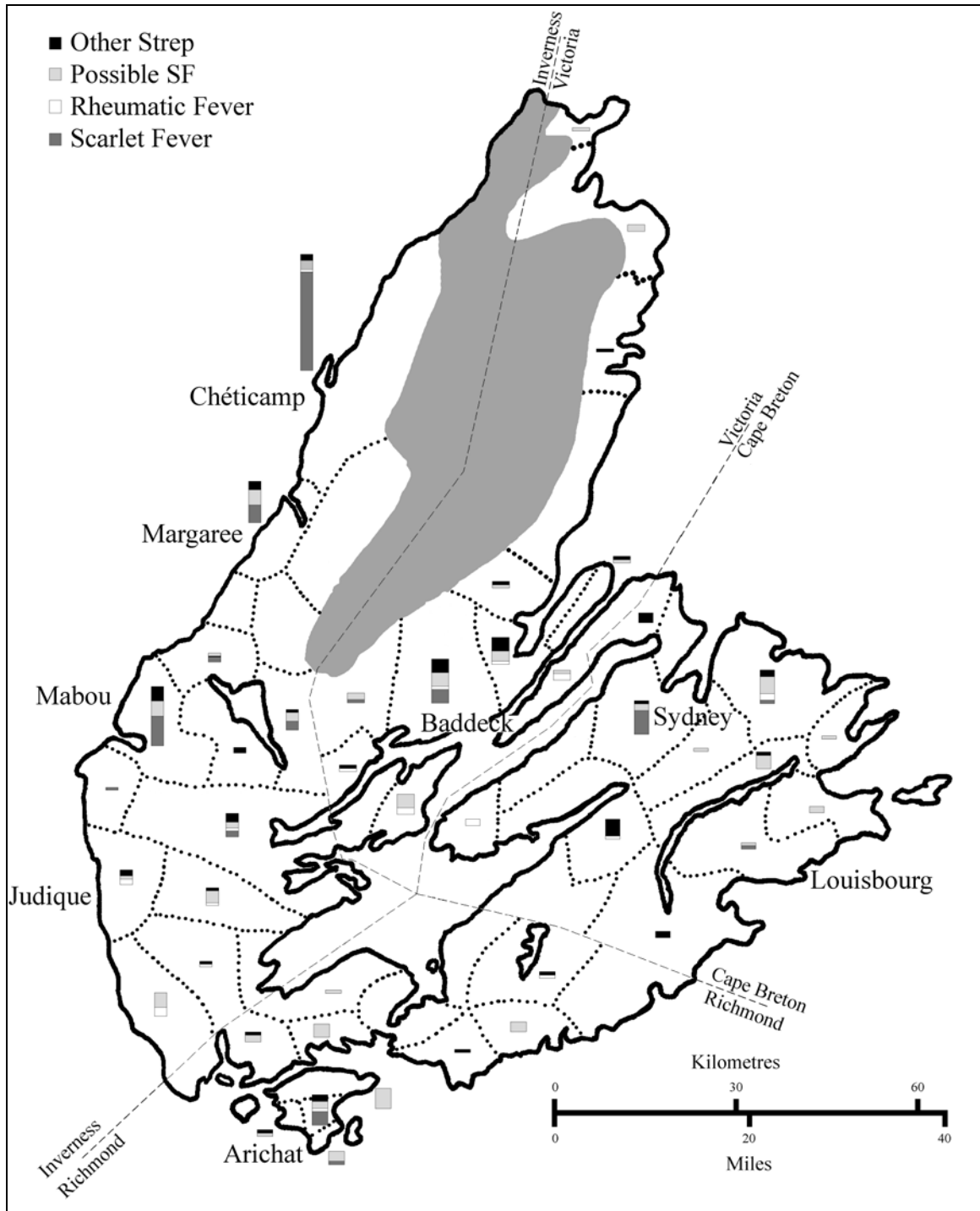


Figure 9.4. Map of Cape Breton Island with the census subdistricts outlined shown with a graphic representation of their corresponding deaths from all categories of streptococcal infection.

9.4 Computer-based Model

Many significant discoveries were made through simulations of the model used to examine the streptococcal epidemic that hit Cape Breton in 1875-1877. A number of questions have been explored with this model but only a few of the more important findings will be presented here.

Sensitivity analysis was used to examine the effects of several factors on model outcomes. Model simulations were run to investigate the effect of changing the number of initial cases and the type of individual that person was, infective or carrier. Figure 9.5 below shows the effects of setting the initial case as an infected adult fisherman and as a carrier juvenile farmer. This factor only slightly affected the timing of the epidemic. When the index case is an infected adult fisherman, the epidemic occurs approximately one month earlier than when a carrier juvenile farmer is the index case. Otherwise, the number of deaths for each group is essentially the same and the curves for the two runs of the model are essentially the same. Because of the lack of effects on the model, other than on the timing of the epidemic, the initial value of the infective adult fishermen is set to one and represents the standard index case for all model runs. It is held constant for sensitivity analysis of all other factors. This is a reasonable assumption given the apparent association between fishermen and the occurrence of the epidemic shown by the other approaches above.

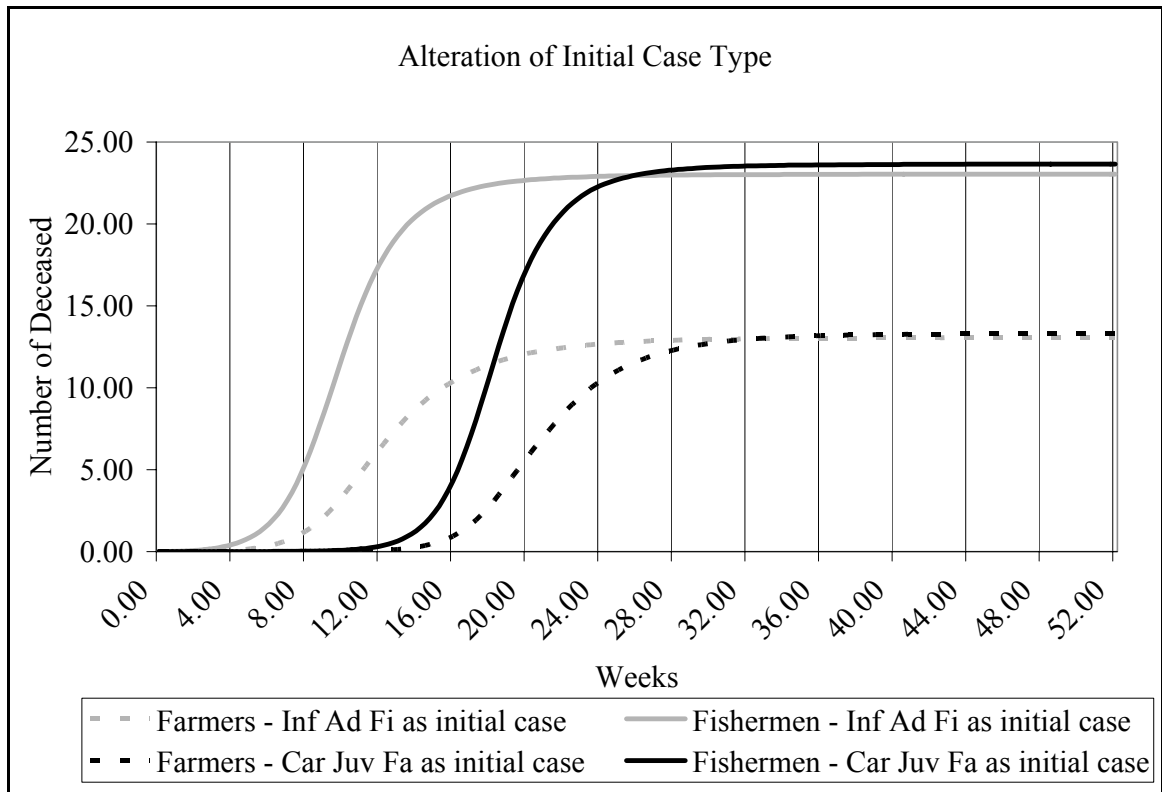


Figure 9.5. Graph showing the effects of altering the type of person who is the initial or index case for the epidemic.

Altering the number of individuals that start at the stock chosen as the initial case does not tend to change the model significantly; even if half the infective adult fishermen are started out in the infective category, the effect on the model is barely detectable. This suggests that it is not the number of people who are infective that is important but rather what type of person they are. During the spread of the epidemic, the number of infectives always follows a bell-shaped curve. This curve is different in height, length and where the peak is located for each group, but it is always the same shape. In other words, there is a pattern to the infectives that is curbed by the recovery rate and the death rate from scarlet fever. Only so many of the infective can die from the disease, the rest have to

become recovered.

Variation in the rates of contact, κ , within the range given in Table 6.1 essentially had no effect on running the model. This suggests not that contact rates are unimportant in the model but that the range of rates used are good estimates. Altering these numbers outside of their ranges does produce a change, especially if high rates are switched with low rates. However, this is not a situation that would likely be encountered given the household types built into the model.

Changing the transmission rate alters the model significantly. If the transmission rate is increased, the number of deaths increases and the length of time it takes for the epidemic to occur decreases. These effects are shown in Figure 9.6 below. This suggests that the transmission rate is a very important factor in changing the results of the model. The highest rate of 0.15 (solid lines) drives the number of dead higher than the historic records show. The lowest rate of 0.05 (dashed and dotted lines) occurs both too slowly and shows a very low number of deaths in comparison to the historic records. These rates show a fairly good range of values for an epidemic model, and the choice to maintain this range for a disease such as scarlet fever or other strep infections seems to match the historic record nicely.

The carrier rate (τ) was also changed to see what its effects might be on the outcome of the model. The normal model range of the carrier τ is set to randomly choose a number between 0.001 and 0.003. There were no significant changes noted even when the carrier τ was set to 0.01. This simply raised the number of juvenile farmers who died from 13 to 16 but left the number of juvenile fishermen who died the same. This suggests

that although the role of the carriers is important, the rate of transmission is not the most important aspect of their role.

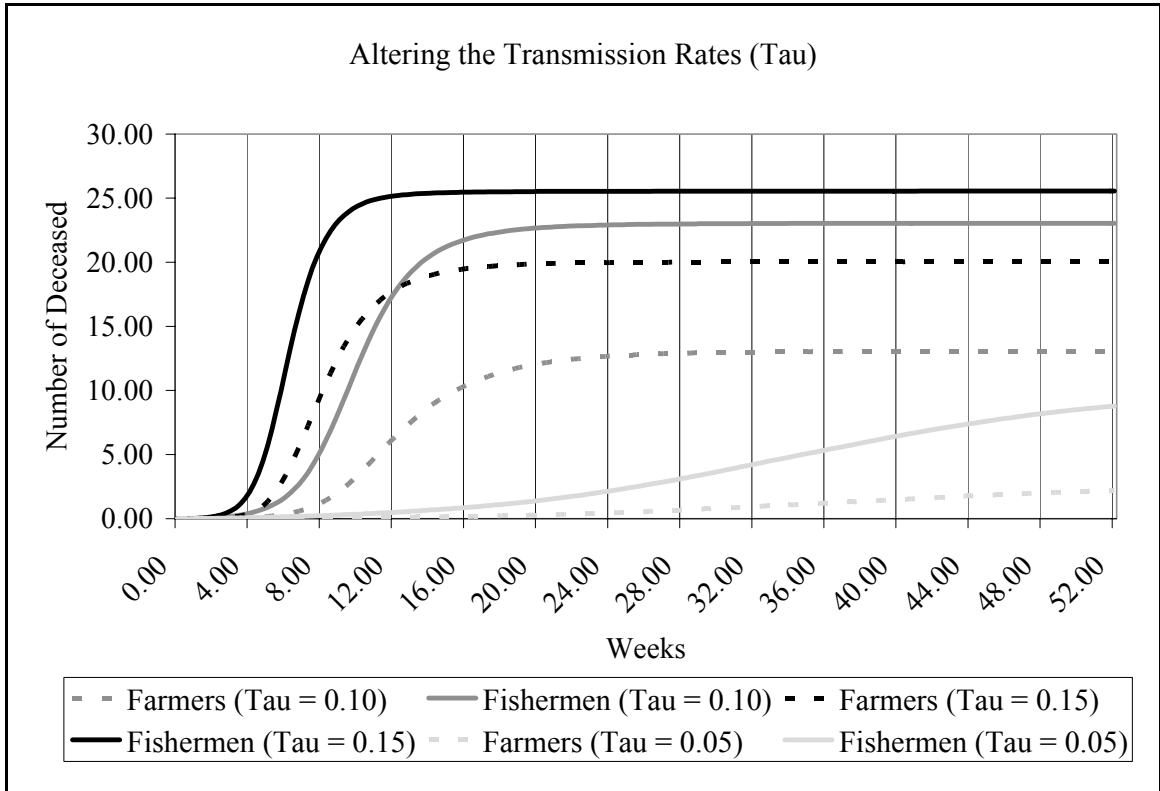


Figure 9.6. Graph showing the effects of altering the transmission rate (tau). This represents the probability that an infective individual will transmit the disease to a susceptible.

The death rate from scarlet fever is one of the more potent factors in the model. It determines the rate at which the infective juveniles will die from the disease. Altering this factor has dramatic effects on the outcome of the model, as shown below in Figure 9.7. The range of this factor in the model, and found to be the extremes of scarlet fever epidemics in the literature, runs from 5% to 30%. These values were used to analyze the alteration of this factor in the model. Interestingly, this factor does not affect the timing

of the epidemic but certainly does have proportional effect on the number of deaths. A low rate of 5% kills a total of about 31 juveniles while a rate of 30% kills about 145 juveniles. The former represents a mild epidemic while the latter represents a major one. The ability to alter this factor is quite helpful in determining the possible virulence of the epidemic that hit different parts of Cape Breton. If all other parts of the model are correct, a death rate of 5.7% was found to approximate the epidemic that hit Chéticamp with a total of about 35 victims. However, this figure is closely tied to the interaction modifier.

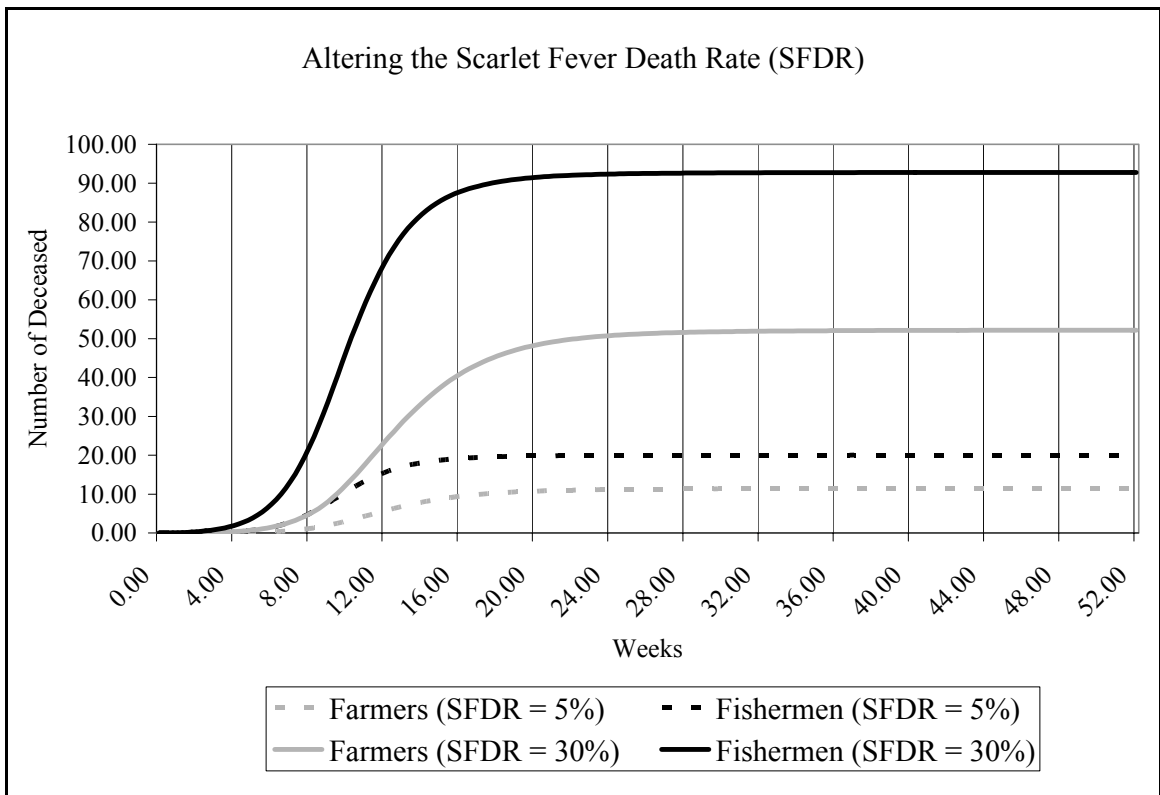


Figure 9.7. Graph showing the effects of altering the scarlet fever death rate that affects juvenile infectives.

The most significant effect of the interaction modifier, and the effect that it was designed to have, was altering the ratio of deceased juvenile farmers to fishermen. Figure 9.8 shows this effect. A low interaction modifier of 1.0 represents the model in the absence of the interaction modifier. This essentially means that each group is equally likely to interact with each of the other groups. A high interaction modifier of 1.9 means that 95% of one's interactions are within one's in-groups (juveniles and adults of the same household type) and 5% are with members of one's out-groups (juveniles and adults of the other household type). Recall from Chapter 6 that the interaction modifier ranges from 0 to 2 since there is one in-group and one out-group. Therefore, 95% multiplied by 2 gives 1.9. An interaction modifier of 1.875 was found to produce the exact number of deceased juvenile farmers and fishermen that died in Chéticamp during the epidemic, 10 juvenile farmers and 23 juvenile fishermen.

The model suggests that 93.75% of contacts in Chéticamp capable of transmission may have been made within one's in-groups and the remaining 6.25% may have been made with members of one's out-groups. This does not suggest that 93.75% of the people one would have had *as* contacts was comprised of one's in-groups but rather that the percentage of contacts capable of transmission of the disease would have been equal to this number. One could conceivably have 75% of contacts being members of the in-group while the percentage of contacts capable of transmission of the disease was 93.75%. This seeming paradox can exist because one is more likely to spend more time with members of one's in-groups (parents, siblings, cousins, etc.) than with members of one's out-groups while still maintaining a certain percentage of out-group contacts.

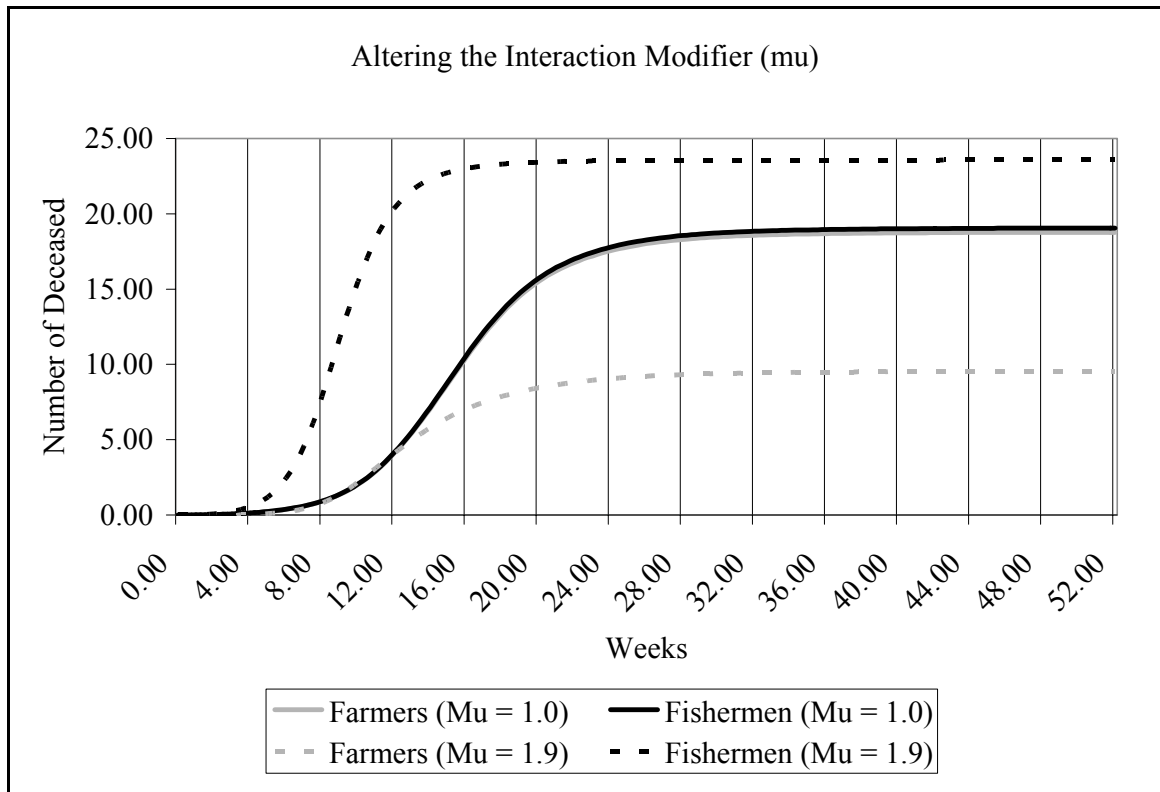


Figure 9.8. Graph showing the effects of altering the interaction modifier.

The carrier rates for adults and juveniles were the final pair of factors to be investigated. Recall that these rates describe the transition from the infective to the carrier state. This rate has been split into separate rates for adults and juveniles because of the differences in the disease experiences of the two. Juveniles are more likely to become carriers than adults because of their less developed immune systems (Moses et al., 2002). The inclusion of this factor was designed to affect the proportion of adult to juvenile carriers. Data for this proportion are drawn from Duben et al. (1978) and Moses et al. (2002). As discussed in Chapter 5, results of these studies suggest that during an epidemic approximately 75% of the carriers are juveniles. The total percentage of carriers

in the population averages 11% with a range of 6.2% to 20.1%. In order to monitor these proportions in the model, a graph was created that reported both figures (Figure 9.9).

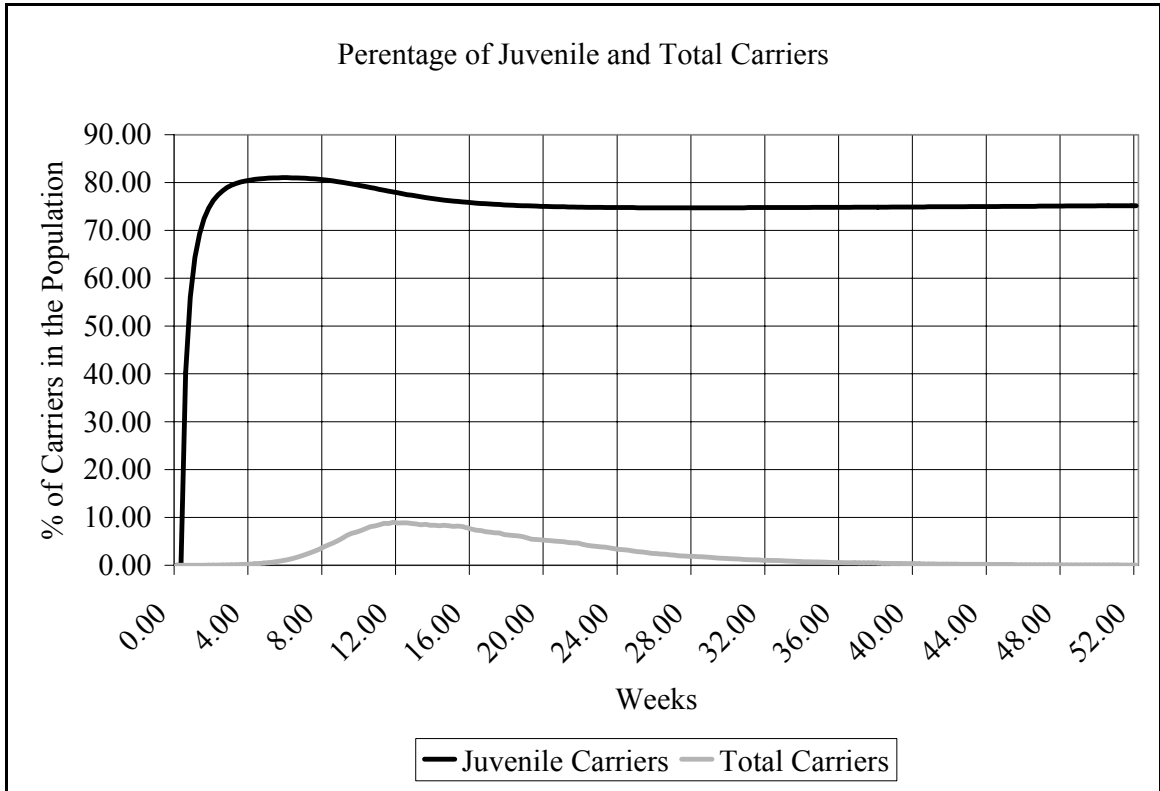


Figure 9.9. Graph depicting the percentage of total carriers in the population and the percentage of those carriers who are juveniles.

Through sensitivity analysis it was determined that the ideal numbers for these carrier rates were 0.44% for the juvenile contact rate and 0.055% for the adult contact rate. These rates maintain the correct number of dead while producing the differences in the ratio of juvenile to adult carriers. The carrier rates are difficult to balance because they affect the number and proportion of carriers as well as the number of dead. If there are too many carriers from the epidemic, the numbers of deceased will be too low. It also

makes the numbers untenable in reality because a high rate suggests that most people carry the disease after their infective period.

Many other factors can be explored but the ones that have a major effect on the outcome of the model have been reviewed here. It is emphasized again that the preceding descriptions concern a model that was built to explore the impact of an epidemic in a single locale in Cape Breton. Applying the population numbers, ratio of farmers to fishermen and ratio of juveniles to adults does not create a “master model” of all of Cape Breton Island. In other words, the outcome of the model does not reflect the outcome of the actual epidemic that took place. The number of dead produced by a run of the model underestimates the actual impact of the epidemic across the island. The proportions of fishermen to farmers also do not match. The implications of this are explored further in Chapter 10.

Chapter 10: Discussion, Conclusions and Future Directions

The results of this project have interesting implications that merit discussion. The initial finding of a high percentage of scarlet fever victims in Acadian families prompted further research into the nature of this epidemic. It was found that the degree of difference in the experiences between the Acadians and Scots was actually much smaller than initially perceived. This was demonstrated by interpreting the historic record to include all possible causes of death resulting from a possible streptococcal infection. However, even after this reinterpretation, Acadians still died in disproportionately higher numbers than the Scots. Answers to this were sought using traditional demographic methods, mapping the disease experience on the Cape Breton landscape, and through the use of a computer-based mathematical model.

The population pyramid in Figure 9.1 shows notable differences in the ages of the Acadian and Scots populations of the index towns of Judique and Chéticamp. The Acadian side of the pyramid reveals a more established population with many juveniles, creating a wide base. This younger Acadian population is partially responsible for higher death rates in that population because of infant and child mortality from childhood diseases. The population with the most children will naturally have higher rates of death because of this phenomenon. It is expected that these rates would even out as the Scots population became more established, say in two to four generations or by the times of the 1891 or 1911 censuses.

Interestingly, the average number of occupants within the households of the two groups is essentially the same, with there being slightly more total people in the houses in Judique. However, a much higher average number of children per household were found in the scarlet fever epidemic victims. This suggests that although household size does not differentiate the ethnic groups, household composition *is* a determining factor in the spread of the epidemic. It should be warned that these conclusions are based on only 28 deaths representing 21 households and that this small number may not be representative of all households on the island with victims from the epidemic. Nonetheless, it is an interesting feature that should be investigated with more analysis. This will require transcription of all 1871 and 1881 censuses as well as all birth and death vital records for all subdistricts and counties on Cape Breton. This feature could not be investigated with the computer-based model because of the aggregate level of organization of the model.

The differences between farmers and fishermen and their lifestyles are perhaps the most telling feature of the study. The differences in contact rates because of these two occupations, and the household types that result from them, are striking. There is a fairly clear association of higher risks for becoming infected with a streptococcal agent if one is a fisherman than if one is a farmer. A high correlation between ethnicity and occupation was also observed. These occupation choices were maintained between generations, likely because of geography and tradition. This creates a situation where ethnic groups maintain specific life-ways and occupations. As a result of this association, any features that come with one's occupation also become part of belonging to an ethnic group. For instance, there are likely fewer cases of drowning amongst the farming Scots than there

are amongst the fishing Acadians.

The geographic distribution of the epidemic deaths is more difficult to summarize. The map in Figure 9.4 shows some trends. The hardest hit areas seem to be coastal or to have ready access to the large Bras D'or Lake in the centre of Cape Breton Island. The subdistricts in the interior are much less affected, some not reporting a single case. This suggests something about the landscape of the island and its importance in the spread of this disease. The geography of Cape Breton Island is mountainous in many regions, especially on the northern arm that juts out into the Gulf of the St. Lawrence in the Atlantic Ocean. This prevents easy travel to and from locations that are otherwise relatively close to one another. This rugged landscape likely created specific patterns of movement throughout the island in a manner that greatly determined the outcome of all infectious disease epidemics. Movement was more easily facilitated by water travel and was the likely mode of arrival of the epidemic in the first place.

Cliff and Haggett (1980; 1984) and Cliff et al. (2000) have found similar results in their studies of measles epidemics in Iceland. They showed that internal travel was hampered by weather in the winter months for the period of 1896-1944. Seasonal distributions of the epidemics reflect this barrier to contact between communities. Epidemics were more likely to spread from community to community in the summer months. Iceland's measles experience showed no endemic period between epidemics due to the low population size. New epidemics had to arise from an incoming outside source. This was supported by evidence that showed port towns, especially the large capital of Reykjavík, were most likely to receive the epidemics first; the epidemics then spread

from these. Interestingly, fishermen were often implicated as the index cases in new epidemics.

The model used in this project was developed to examine the factors, both biological and social, that were suspected to have the most impact on the outcome of the epidemic. The model was designed to demonstrate the disease experience in single locations that shared discrete characteristics due to the concentration of the population in a certain locale.

The model does not perform accurately when the initial numbers in each population group (e.g., adults and juveniles; fishermen and farmers) are increased to reflect the entire island's population rather than just the population of Chéticamp. This suggests something very significant about the actual epidemic, assuming that the model is correct. The inability of the model to summarize the disease experience of the entire island of Cape Breton may indicate that one or all of the contact rates, the transmission rates, the interaction modifier and the scarlet fever death rates in addition to the ratios of dead juvenile farmers to fishermen are different in the different locales around the island. The *ratio* of juvenile to adult contact rates may change but is probably a biological factor of all populations that, when all things are considered equal, are similar from group to group. That is, the differences in immune system responses between adults and juveniles are likely a feature of biology rooted in development, rather than social or cultural factors. The transmission rates are also a biological factor that is likely not facilitated or hindered by much other than individual circumstances. The contact rates are a social reality tied to the household types themselves.

The interaction modifier is almost certainly the only factor of these four that is affected by the social structure of the population. This can be influenced by geography in terms of population density increasing interactions with members of out-groups and certainly by the introduction of different household types to the model. The interaction modifier may be altered depending on the geography and population density of the area.

The most powerful of the factors influencing the patterns of epidemic spread on the island is probably the scarlet fever death rate (SFDR). The SFDR may hold the key to the problem of differing experiences seen around the island in Figure 9.4. Perhaps there was more than one strain of this disease cycling at the same time. The preceding literature review suggests that this is the normal situation of populations experiencing a streptococcal epidemic. These different strains can have completely different virulences and, in the complex case of *Streptococcus*, may even manifest in different forms. The model is not conducive to testing a situation of more than one strain but this is a possibility that needs to be investigated further.

A scenario that might show evidence of multiple strains acting in concert with each other would have a few identifiable features. For one, the epidemics would be interspersed with a continuous minimum number of morbid cases in the population. Theoretically, this could be as low as one but would probably be much higher in order to sustain the presence of the strains in the population. There might also be overlapping cycles of epidemics that are out of phase with one another. These cycles might be identifiable by the exact timing of their peaks, the duration of the epidemic or the height of the peaks in relation to surrounding ones. In other words, the differing virulence of two

strains would alter the SFDR (e.g., one low, one high) and, thus, the number of dead resulting from each strain. However, there is much background noise and other confounding factors that could make this difficult to identify. In the historic record, there may also be a shift in how the causes of death are reported because of differing manifestations caused by different strains. For instance, those affected by one strain may be listed as dying from rheumatic fever while those affected by a different strain may be listed as dying from scarlet fever. The potential for multiple strains to sustain repeated infections in a location is high (Gunn and Griffith, 1928; Moses et al., 2002; Perks and Mayon-White, 1983; Martin and Høiby, 1990). Multiple strains may keep a continuously renewed susceptible population supplied with new challenges to their immune systems. Because the immunity conferred by surviving one strain is not transferred to the experience of another, there is a higher chance of death with multiple strains, even if the virulence of one strain is low. These are just some examples of what multiple strains may look like in the historic record and in a model of such a situation.

The model was shown to have potentially high explanatory power and can be compared to any epidemics of streptococcal infections given that enough information on the deaths of the individuals is available. Morbidity data could even be helpful because of the ability of the model to set initial numbers of infectives and carriers. However, the model is limited to analyzing just two household types and only two age groups in a population. This may be sufficient for some epidemics but not for others where selective infant mortality, or elderly mortality are also issues.

Several conclusions can be drawn from this study that inform us about the potential of historic records research. The first is that we must never take the information at face value. Understanding the cultural and temporal context of the records, their recorders and the people to whom the events in the records happened is essential in piecing together an accurate picture of what happened. Interpreting what the study population recorded as the temporally colored reality of medicine and disease experience requires painstaking research into the lives of these people and the times in which they lived. Each person in each time has an individual perception that shares in a larger group perception of what they have experienced. We have to understand the steps involved in how a true event becomes a recorded event and eventually a transcribed event passed down to us. This involves appreciating the social reality of disease and death and the language and terminology used to talk about it. It helps to be able to read a range of cursive hands as well.

A number of approaches and sources were used in this study because it is believed that this best facilitates the analysis of a complex chain of events such as those involved in the spread of an epidemic. Approaching a data set from different angles with different tools allows one to draw several interrelated conclusions which support one another better because they use different aspects of the data. For instance, the importance of island geography in the spread of the disease would not have been noticed if a map was not created to show the areas of greatest impact.

The project aimed to answer four research questions. The first was, *“Is there a difference in the patterns of spread and timing of scarlet fever epidemics between the two*

major ethnic groups on Cape Breton Island?” The obvious simple answer is “yes”. An initial surface reading of the historic record suggests a misleadingly high proportion of Acadians died from the scarlet fever epidemic of 1875-1877. However, a revised, more liberal interpretation of the historic record shows that Acadians still suffered a higher than expected number of deaths from the streptococcal epidemic than the Scots, though not as severe as a more literal interpretation would suggest. Despite making up only 14.39% of the 1871 census population, Acadians account for 22.45% of deaths due to all streptococcal causes from 1875 to 1877. Scots comprise 66.39% of the 1871 census population but only account for 53.31% of deaths due to all streptococcal causes from 1875 to 1877.

The second question was, “*Does the inclusion of cause of death information for diseases reported as something other than scarlet fever, but which are nonetheless equally likely to have been caused by streptococcal bacteria, present a different picture of epidemic spread than analysis of scarlet fever deaths alone?*” Again, the answer to this question is “yes”. The picture is quite different when other causes are considered. It appears almost as if the Scots and Acadians interpreted the manifestation of the same epidemic differently. The Acadians used the phrase “scarlet fever,” or “*scarlatine*” in French, almost exclusively when referring to the causes of death. The Scots tended more to use the phrase “rheumatic fever” or simply “fever” without further indications of its specific nature. This supports the idea that disease reporting is linguistically and culturally specific. It may even reflect different past experiences of the two populations. The Acadians likely experienced other epidemics of scarlet fever that were documented

by doctors as such while the Scots may not have encountered the name before either because of a lack of experience with the disease (not likely) or a lack of knowledge of this term (more likely).

The third research question asked was, “*If there are differences in the patterns of spread and timing of scarlet fever epidemics between the two major ethnic groups on Cape Breton Island, as I expect there will be, what are the behavioral or cultural factors that may be contributing to these differences?*” The analysis showed that a combination of factors contributed to this difference. The Acadians had more juveniles in their population, lived in busy port towns and had maintained a lifestyle of fishing that resulted in increased daily contact rates. The families that had members who died from the epidemic were also shown to have higher than average numbers of juveniles living in the household at the time of the victim’s death. All of these suggest that a complicated set of interactions were present upon the arrival of the epidemic that predisposed the Acadians to a worse experience of deaths and illnesses. The age composition of households and their occupations, not their biological relatedness or ethnic identity, doomed the Acadians to suffer the worst of the epidemic’s effects.

Finally, the fourth question asked in this project was, “*What is the effect of multiple strains of streptococcal infection on the course of an epidemic?*” The model designed was unable to test the effects of such a situation. However, the power of the biological factors in the model to effect change in the numbers of dead the model produced suggested that multiple strains may have been the reason for different experiences of death at different locales. This is especially true of the scarlet fever death

rate, which reflects the virulence of the disease. Despite the deaths of 33 juveniles (5% of all juveniles) in the community of Chéticamp, which numbered just over 2,000 people in 1875, the model suggests that the virulence of the epidemic was low (5.7%). Most epidemics in the literature suggest death rates between 5% and 30%. Were the epidemic more virulent, the effects on the population may have been catastrophic. This suggests that only one strain was likely present in the 1875-1877 streptococcal epidemic of Cape Breton Island.

In the widest sense of this project, the important role of cultural and social factors in the spread of epidemics has been demonstrated. The inter-relationships of people are equally as important as the biological mechanisms of the infecting agents that colonize them. We are the larger environment of micro-organisms and so the interactions within that environment must be understood in order to fully appreciate the intricacies of the system. Studies of disease systems *should not* be limited to the biology of the micro-organisms that cause them. We need to know about the fluctuations in their environments. Though we cannot look at the individual biology of every infected person in history, we can examine the cultural, social and resulting demographic behaviors of the populations at large. The choices people make based on these behaviors ultimately influence that wider environment of the infecting micro-organisms we seek to understand.

In a more specific sense, this project increases understanding about the behavior of group A streptococcal infections in historical populations. The disease is so varied, as evidenced in the huge number of strains and the mechanisms it has evolved to bypass our

bodies' defense systems, that it is likely to continue to stay with us for a very long time. Though scarlet fever seems to have largely disappeared as a specific manifestation of the infecting agent, GAS infections are still responsible for a large number of cases of disease and death worldwide (Beres et al., 2002; Katz and Morens, 1992; Moses et al., 2002; Quinn, 1989). The CDC reports 3.2 cases per 100,000 for 2002 with a disturbing rise in all cases, but especially in severe manifestations (streptococcal toxic shock syndrome and necrotizing fasciitis) since the 1980s and 1990s (CDC, December 2003). It will continue to plague us in the near and distant future. However, if we can understand the cultural, social and demographic behaviors that promote its spread, perhaps we can take measures to avoid potentially large epidemics and pandemics in the future. Factors such as trade, migration and class can be affected by our actions and may provide clues to help defend us from future problems with this disease.

The anthropological significance of this project is to emphasize the role of demographic, social and cultural factors in analyzing any epidemic. This approach needs to be emphasized more strongly in standard epidemiological investigations. People are social beings and any social being is going to react to epidemics with social and cultural behaviors in addition to their biological responses. We must continue to analyze the interactions of biological and socio-cultural factors in epidemic studies.

For Nova Scotia, this project is a unique investigation. Despite the well-known ethnic migrations to Cape Breton Island and the unique example this provides, the area's disease history is poorly documented. This region is an excellent setting for such historic case studies. The data are rich and extensive and are easily accessible. The history of the

area is well-documented and published in both popular and academic media (Brasseaux, 1991, 1996; Caplan, 2001; Chiasson, 1998; Donovan, 1990; Doucet, 1999, 2003; Forbes, 1979; Griffiths 1973, 1981, 1992; Hornsby, 1992; M^{ac}Donald A., 1990; M^{ac}Donald PM, 1999; M^{ac}Donell, 1982; M^{ac}Kenzie, 1979; M^{ac}Millan, 2001; Moore, 1982; Newton and Robertson, 1984; Planetta 1980; Plank, 2001; Ryan, 1992). Such areas should be taken advantage of if we are to understand disease history in general.

The quality of these data has good and bad points. In 1864, the British Empire passed a law requiring the registration of all vital events with a federal registrar. In 1877, this law was dropped or revoked for unknown reasons. This window of 1864-1877 for the vital records has unfortunate timing for this epidemic in particular. It is likely that individuals continued to die from the epidemic after the recording of vital events was taken out of Canadian legislation. To remedy this problem, transcription of parish records for many locations on the island would be an ideal way to supplement the current data. Not all parishes in Cape Breton have made these available to the public and not all have been transcribed. The original parish records of Chéticamp are not available in full to the public. One of the volunteer staff members of *Les Trois Pignons* cultural centre is available for consultation. In addition, serious researchers are often able to convince curators to allow a transcription if their project is deemed important and beneficial.

The under-reporting of occupations and causes of death in particular is also an unfortunate limitation of this study. It is not known if the actual death certificates of each individual survive and contain the missing information. More research in this area needs to be undertaken to ensure that all possible causes of death that are available have been

accounted for. Occupations are more easily obtained but will require marriage records from the vital records to be transcribed. Again, this is limited by the 1864 to 1877 window. Nonetheless, the fact that these records have survived and come down to us is greatly appreciated. The registrars did the best that they could to ensure the recording of vital events and the census records are in good shape as well.

Though the order of recording in the censuses is elusive, a more detailed examination may prove useful. Correlation of census records to the A. F. Church maps will require the transcribing of all census subdistricts on the island for the 1881 census and perhaps the 1891 census as well, since the maps were produced in the decade between these two. The families can then be located in the census records and cross-referenced to the map. This will facilitate a detailed geographic plotting of this epidemic. The A. F. Church maps themselves are an absolute wonder to have, but for the current project they contain too much information to be managed properly at this time.

In order to examine the movement of the disease across the Cape Breton landscape, full geographic plotting, based on the A. F. Church maps, should then be attempted. This would involve GIS software that could calculate the distances between locales and also record the roads, railways and water routes available to people on the island. The potential for such a project is great and will likely be pursued with the aid of other investigators.

The demographic parameters of the data set will only improve with the transcription of more vital records, censuses and parish records. These will facilitate the creation of genealogical databases that can link families to one another. This would allow

for biological as well as social analyses of any number of factors including occupation trends, disease trends, family size trends, etc.

The model used in this study, while having potential to provide very useful information about the nature of streptococcal infections, will likely be abandoned in the near future. The reason for this is the inability of the modeling approach used to reflect individual variability and the flexibility and complexity that can be achieved with the use of agent-based and other individual-based modeling techniques, which can explicitly model the lives and movements of individuals. An individual-based model is presently under development with a colleague. Such a model will allow for the creation of other household types that have different movement patterns, contact rates and locations, the analysis of household sizes and the automatic inclusion of an aging process that eliminates the need for different stocks divided along age group lines.

Follow-ups on the generations of survivors of epidemics would make for an interesting study. This would require good coverage of the island in terms of parish records because of the stoppage of vital events recording in 1877. The loss of this source of information until the practice was re-legislated in 1908 is unfortunate but may be able to be worked around.

Overall, there is much potential with this project. An island location presents a somewhat closed system but is still a participant in the events of world pandemics. Interestingly, the scarlet fever epidemic of 1875-1877 was probably the least impacting of the childhood epidemics seen elsewhere in the record. Epidemics of croup, diphtheria, measles, typhoid fever and whooping cough are all found in the record. Adult epidemics

of consumption, pleurisy and phthisis are present as well. Continued analysis of these data will provide many future research opportunities. It is hoped that future studies can benefit current policy on disease reporting and documentation as well as protocols designed to protect society in the event of large outbreaks. If we are, as a society, to survive the more serious illnesses that linger on our doorstep, it behooves us to examine the past so that it is not repeated.

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