AGENT-BASED MODELING OF SEASONAL POPULATION MOVEMENT AND THE SPREAD OF THE 1918-1919 FLU: THE EFFECT ON A SMALL COMMUNITY

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The undersigned, appointed by the Dean of the Graduate School,
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AGENT-BASED MODELING OF SEASONAL POPULATION MOVEMENT

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Presented by Connie Carpenter

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And hereby certify that in their opinion it is worthy of acceptance.

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Chapter 1: Introduction

The 1918-1919 influenza epidemic caused more deaths worldwide than any recorded flu epidemic (Stevens et al., 2004). The people of the small fur-trapping community of Norway House in Manitoba, Canada, were especially hard hit by the epidemic. It has been estimated that 18% of the population died during the flu epidemic in a three-month period (Herring, 1994). The loss of such a large number of people in such a short period must have had a huge impact on the community. The primary goal of this project is to use historical information and computer simulation to study how the 1918-1919 flu epidemic might have spread through the Norway House community.

Archival information from the Hudson's Bay Company (HBC) post journals, the Anglican Church of Canada records, the Norway House Cree First Nation Treaty Annuity Lists, and the Canadian Government have been analyzed by others and made available for the period of time covering the 1918-1919 flu epidemic in the Norway House community. A collaborative effort between Ann Herring and Lisa Sattenspiel, using archival and epidemiologic information in combination with mathematical modeling techniques, has further expanded our knowledge about the Norway House community's experience with the 1918-1919 flu epidemic (Herring, 1994; Herring and Sattenspiel, 2003; Sattenspiel, 2003; Sattenspiel and Herring 1998; Sattenspiel et al., 2000). This information provided wonderful background information and a unique landscape for the development of a new modeling approach to the epidemic.

Agent-based computer simulation provides a new technique for the study of virgin soil epidemics like the 1918-1919 flu. By using this individual-oriented simulation technique, a landscape can be created and populated with a heterogeneous group of agents who move and interact in ways that more closely resemble human behavior than is possible in most other modeling techniques. In this project, various characteristics of the epidemic and the Norway House community were incorporated into the model. These parameters were varied over a range of values and the results analyzed to provide some insight into why the 1918-1919 influenza virus was so virulent and why this epidemic hit this particular community so hard.

This project is based on one major methodological issue and two research questions. The methodological issue was the construction of an agent-based computer model that would simulate the spread of the 1918-1919 influenza epidemic through the Norway House community. Once a computer simulation was constructed and verified, two research questions were tested. The two research questions were based on the influence of changes in population movement patterns on the transmission of disease through a community. First, could the seasonal population movements of the Norway House community influence the spread of the flu through the community? Second, could a winter epidemic differ from a summer epidemic predominantly due to changes in seasonal population movement? In other words, could the change in a single cultural variable, such as seasonal population movement, strongly influence an epidemic?

The construction of the computer simulation required detailed information about the influenza virus, the 1918-1919 Influenza Pandemic, the Swampy Cree-Métis people of the Norway House Community, epidemic modeling, computer simulation, and agent-based modeling. Ethnographic data from the Norway House community was used to create a landscape, people, and rules for behavior of the people within the computer simulation. Current and historical information about the influenza virus and the 1918-1919 Influenza Pandemic were used to create rules for the spread of influenza through the community in the computer simulation. Various techniques of agent-based modeling, epidemic modeling, and computer simulation were integrated to create and run the computer model.

Once the computer simulation was constructed, each component of the model was tested over a range of values. This process was required for the verification of the computer simulation. It was necessary to prove that the simulation was behaving in the way it was intended to behave. After verification of the model was complete, the research questions were investigated using the computer simulation. Summer and winter scenarios were devised which differed only in the pattern of population movement. Assumptions about seasonal population movement patterns were based on ethnographic data. By testing each component of the model over a range of values in the summer and winter scenarios, the influence of a single cultural vector was investigated. This thesis provides the detailed information and methodology used in the model construction and the results obtained from the computer simulation.

Archival and contemporary information about the 1918-1919 influenza epidemic were used to create a computer simulation that could be manipulated in a way that is not possible or ethical in real life. The dynamic nature of the computer simulation allowed for the observation of epidemics over time and at various parameter values. Computer simulation can be a very powerful tool for an anthropologist as long as it is kept in mind that the model is only a simulation of the study population and their actions. The complex environment, culture, and biology of humans cannot be reproduced in a model but vital clues can be obtained from a well-constructed model that will enhance our understanding of the human condition.

Chapter 2: The 1918-1919 Influenza Pandemic

During the 1918-1919 influenza pandemic, an estimated 21-50 million people died from the flu (Taubenberger, 1999). This figure is more than double the number of soldiers killed on the battlefield during World War I (Keegan, 1999). Before one can study the 1918-1919 influenza pandemic, it is necessary to become familiar with the virus that causes influenza and understand how the pandemic was different from the annual flu epidemic seen today. First, the biology, epidemiology, and clinical manifestations of influenza are discussed. Second, a general perspective of the 1918-1919 influenza pandemic is presented. The chapter concludes with a few of the more recent theories about the 1918-1919 influenza virus and the pandemic the virus caused.

Influenza and the Influenza Virus

The influenza virus causes the disease influenza (Isada, et al, 1999). Viruses are intracellular parasites that are completely dependent upon the host cell for all their metabolic and reproductive processes. Viruses consist of a protein coat surrounding a nucleic acid core of RNA or DNA (Crawford, 2000; Beers and Berkow, 1999). The influenza virus is an RNA virus from the Orthomyxovirus family (Isada, et al., 1999). Based on the antigenic differences of internal proteins, there are three types of influenza virus, type A, B, and C (Isada, et al., 1999).

Influenza epidemics and pandemics are usually associated with the type A virus (Chin, 2000). When a sudden increase in the number of cases of a disease occurs locally, the disease is considered an epidemic. When the increase is global, the disease is considered a pandemic (Sattenspiel, 2000).

Type A influenza viruses are further categorized by the antigenic difference in two glycoproteins on the surface of the virus, hemagglutinin (H) and neuraminidase (N). Fifteen subtypes of hemagglutinin and nine subtypes of neuraminidase have been identified to date but only three hemagglutinin subtypes (H1, H2, and H3) and two neuraminidase subtypes (N1 and N2) have been identified as pathogenic or able to cause disease in humans (Nicholson, 2003). The hemagglutinin glycoprotein allows the virus to attach to the host cell membrane prior to entry. The neuraminidase glycoprotein aids in the release of new virus particles from the infected host cell. Both glycoproteins are subject to immunological response in the human body (Beers and Berkow, 1999). The human antibody response is specific for the viral type and subtype and offers little or no protection against a new strain of virus (Couch et al., 1983).

Over time, hemagluttinin and neuraminidase undergo slight structural changes due to mutations that result in different influenza virus strains. The result is an annual change in circulating flu viruses and usually an annual flu epidemic in humans. This process is called antigenic drift (Beers and Berkow, 1999; Chin, 2000).

Another process that influenza viruses undergo is antigenic shift. The RNA from the influenza virus is composed of eight distinct gene segments that can

move independently through reassortment (Taubenberger, 2003), a process by which gene segments can be exchanged between infecting viral particles. A single host cell can be infected with two different strains of influenza virus resulting in a new strain of virus that contains a mixture of genes from the two original viruses. If the new strain of virus contains hemagluttinin or neuraminidase antigen genes that differ significantly from the parent viruses and can invade human cells, the immune system may not be able to recognize the antigens on the new virus and initiate defensive measures (Crawford, 2000). The process of gene segment exchange between human and animal influenza viruses can result in a significant antigenic shift. This process occurs only in the type A influenza virus and is the reason that influenza pandemics occur every 10-20 years (Beer and Berkow, 1999; Isada, et al., 1999; Chin, 2000). The 1918-1919 influenza pandemic was caused by an H1N1 influenza type A virus that underwent antigenic shift from an animal source and entered the human population (Taubenberger et al., 1997).

Aquatic birds are thought to be the natural reservoir for the influenza virus (Taubenberger, 2003). Birds transmit or spread the virus by the fecal-oral route through the water supply (Webster, 2002). The host range for the influenza type A virus is humans, swine, domestic or wild birds, and horses (MSDS, 2001). In humans, the virus is transmitted person-to-person via airborne droplets and occasionally by contaminated items (Isada, 1999; Beers and Berkow, 1999). The airborne transmission of the virus is most common in crowded areas and

enclosed spaces (Chin, 2000). The probability of transmission is very high in family households (Kilbourne, 2003; Longini et al., 2004).

Human susceptibility or risk for infection from a new subtype of the influenza virus is almost universal. Immunity to influenza is specific to a particular strain or subtype of the virus but the virus can change annually (Chin, 2000).

Once the influenza virus is transmitted to a susceptible host, the host does not immediately exhibit symptoms of the flu and is not infectious. The incubation period is defined as the asymptomatic period between transmission of the virus and the point in time when the host becomes symptomatic with the disease. The latent period is defined as the non-infectious period between the transmission of the virus and the point in time when the host becomes infectious to susceptible people (Sattenspiel, 2000; Moser et al., 1979; Cox and Subbarao, 1999; Beers and Berkow, 1999). The incubation period and the latent period are considered the same length of time for influenza viruses with an estimated mean length of 1.9 days (Longini et al., 2004). Once the incubation period ends and the host becomes symptomatic, the clinical disease can last up to two weeks (WHO, 2003). When the latent period ends, the infectious period begins and can last 3-5 days in adults and up to 7 days in children (Chin, 2000). The infectious period begins when the virus can be transmitted to other people susceptible to that strain of the influenza virus and ends when transmission of the virus is no longer possible (Hope Simpson, 1948; Sattenspiel, 2000).

Influenza is a respiratory illness with symptoms such as an abrupt onset of headache, fever, chills, generalized aches and pains, runny nose, sore throat

and an unproductive cough (Isada, et al., 1999). Usually, the illness is uncomplicated and the symptoms will resolve in 1-2 weeks (WHO, 2003). In some cases, a secondary bacterial infection will cause pneumonia following an influenza infection, resulting in worsening of the respiratory symptoms, recurrence of fever, and a productive cough with purulent or bloody discharge (Beers and Berkow, 1999). Typically, *Streptococcus pneumoniae*, *Staphylococcus aureas*, or *Haemophilus influenza* are responsible for the secondary bacterial infection. It has been speculated that there is a higher mortality rate during influenza epidemics because a synergism exists between pathogenic bacteria and the influenza virus, causing more severe disease (Simonsen et al., 2000). In rare cases, a fulminating viral pneumonia can occur due to the influenza virus alone. The viral pneumonia can cause hypoxia with an associated cyanosis and death. Death can occur within a few days (Isada, et al., 1999). Mortality from influenza varies with the subtype of the virus in addition to the age and general health of the person affected (Wright et al., 1980). Normally, influenza has a higher mortality rate in the very old and the very young (Kilbourne, 2003). Annual flu epidemics cause 250,000 to 500,000 deaths around the world (WHO, 2003).

The 1918-1919 Influenza Pandemic

The first wave of the 1918-1919 influenza pandemic began in the spring of 1918 (Taubenberger, 2003). The origin of the epidemic is unknown but the current speculation is that it originated in the United States (Stuart-Harris, 1985;

Taubenberger, 2003; Johnson, 2003,). The pandemic spread rapidly because the disease was highly contagious (Taubenberger, 2003) and the movement of armies during World War 1 resulted in transportation of soldiers and the flu virus to countries all over the world (Stuart-Harris, 1985). The 1918-1919 flu was truly pandemic because it spread throughout the globe (Taubenberger, 2003).

The first wave of the pandemic in the spring caused little mortality but the second wave, which occurred in the fall of 1918, had high mortality rates (Taubenberger, 2003; Stuart-Harris, 1985). The average mortality rate for an annual influenza epidemic is 0.1% in the United States. The average mortality rate for the 1918-1919 influenza pandemic in the United States was 2.5% but exceeded 70% in some isolated communities around the world (Stevens et al., 2004).

An unusual feature of the 1918-1919 flu was that it caused more deaths in young adults than in other age groups. The estimated ages of the young adults range from 15 to 40 years old (Reid et al., 1999; Stevens et al., 2004; Stuart-Harris, 1985; Schoenbaum, 2003). The mortality risk of the 1918-1919 influenza pandemic for a person over 60 years old was the same as the risk for a normal annual influenza epidemic (Stevens et al., 2004). This pattern differs from an annual influenza epidemic, which causes a higher mortality rate in the very old and the very young (Reid et al., 1999). In addition, the 1918-1919 flu had the capability to kill very quickly. A person could be feeling fine in the morning, yet be dead by nightfall (NVPO, 2004). Heliotrope cyanosis (a dusky skin color due to hypoxia), lung congestion, and hemoptysis (blood in the sputum) were

prognostic signs of an early death (French, 1920). These symptoms are much like the symptoms of fulminating viral pneumonia described previously. Secondary bacterial infections were also common during the pandemic, especially in young people (Stuart-Harris, 1985).

The influenza virus was not isolated in humans until 1933 by Smith, Andrewes, and Laidlow (Kilbourne, 2003), so the medical establishment in 1918 had no idea what was causing the pandemic. Fortunately, lung tissue samples were preserved from several soldiers who died rapidly from the 1918-1919 flu. From these lung tissue samples and other samples gathered from arctic graves, segments of viral RNA have been extracted and sequenced from the coding regions for hemagglutinin, neuraminidase, nucleoprotein, and two matrix proteins. The virus has been classified as an H1N1 type A influenza virus. In 1997, it was postulated that the virus belonged to a subgroup of viral strains that infects humans and pigs (Taubenberger et al., 1997). In 2004, further examination brought to light avian components in the hemagglutinin glycoprotein. The current speculation is that raising a large number of domestic animals and fowl in a small area, a highly mobile population, and globalization increase the risk of pandemics from viruses like the influenza virus (Stevens et al., 2004). All of these risk factors were present during the 1918-1919 influenza epidemic (Crosby, 1989).

The 1918-1919 influenza pandemic was caused by a particularly virulent strain of the influenza virus (Ewald, 1991). Virulence, the ability of a virus to invade and damage host cells, involves characteristics such as the ability to

reproduce in the host, host adaptation to the virus, the ability of the host tissue to avoid damage from the virus, and transmission of the virus to the host (Taubenberger, 2003.) One factor that enhanced the virulence of the 1918-1919 flu virus was the novel structural nature of the virus (Stevens et al., 2004). A second factor, which may have influenced virulence during the 1918-1919 flu epidemic, was cultural vectors or mechanisms by which humans enhance transmission of pathogens. The environment of a World War I battlefield involved soldiers under a great deal of stress, crowded into trenches with poor sanitation. Soldiers were frequently in foreign lands and exposed to infectious diseases to which they had no immunity. If soldiers got sick or hurt, they were moved to crowded hospitals or sent home on crowded troop ships (Ewald, 1991). It has been postulated that this wartime environment encouraged not only the rapid spread of the influenza virus but also the evolution of a new, more virulent strain of the virus (Kilbourne, 2003; Ewald, 1991).

In this project, characteristics of the influenza virus and the 1918-1919 epidemic, as described in this chapter, were used in conjunction with cultural characteristics of the Norway House people, as described in chapter 3, to create a computer simulation of the Norway House community during the 1918-1919 influenza epidemic. The next chapter will review the social characteristics of the Norway House people, in order to set the stage for the model to follow.

Chapter 3: The Norway House Community and The 1918-1919 Flu Epidemic

In this project, archival information about the Norway House community in Manitoba, Canada was used to provide a framework for an agent-based simulation of the spread of the 1918-1919 influenza epidemic through a small population. Prior to the construction of the simulation, it was necessary to become familiar with the Norway House people, especially the type of environment they lived in, their economic practices, their social organization, and their seasonal movement patterns. Additionally, information was needed on the specific history of the epidemic in the Norway House community. Knowledge about how the local people interacted with each other was especially important in order to simulate this pattern in a computer model. The simulation was used to attempt to reconstruct the spread of the influenza virus through the community as it might have happened during the epidemic.

The Norway House community is located on the Nelson River at the northeast corner of Lake Winnipeg in Manitoba, Canada (Ray, 1974; Hallowell, 1992) as is shown in Figure 3.1. The community is located in a sub-arctic environment consisting predominantly of coniferous trees surrounding a complex network of lakes and rivers. The soil is unsuitable for agriculture because it is frozen for most of the year and swampy when it is not frozen. The temperature ranges from – 20 ^{o}F in the winter to + 72 ^{o}F in the summer. Traditionally, summer is considered to extend from June to August. Frost and snowy conditions begin in

September and by November; an average of 50-100 inches of snow has fallen. The lowest temperatures occur in February. The ice and snow begin to melt in April but the ice does not break up until early May. Leaves begin to appear on deciduous trees in June and the seasonal cycle repeats. Numerous varieties of birds, fish, and mammals inhabit this sub-arctic ecosystem including many animals important to the fur trade such as beaver, mink, marten, fisher, otter, ermine, fox, and lynx (Hallowell, 1992; Morse, 1989).

The Hudson's Bay Company established Norway House as a fur trading post in 1826 and it became a key center for the collecting and transportation of furs (Morse, 1989). In 1918, although the fur trade was on the decline, Norway House continued to be a Hudson's Bay Company post and a Swampy Cree-Métis settlement. The Hudson's Bay Company post was managed by Europeans but the majority of the trappers were indigenous or of mixed ancestry (Ray, 1974).

The economy of the Norway House community was based, for the most part, on hunting, fishing, and the fur trade. The fur trappers, primarily the Swampy Cree-Métis people, worked on a credit system with the Hudson's Bay Company post. The trappers would obtain supplies on credit from the post prior to their hunting trips and bring furs and other goods back to the post to trade and reduce their debt when they returned. Unfortunately, the natives became increasingly indebted to the post due to a growing reliance on trade goods and the normal cyclic variation in the abundance of fur-bearing animals (Hallowell, 1992; White, 1999).

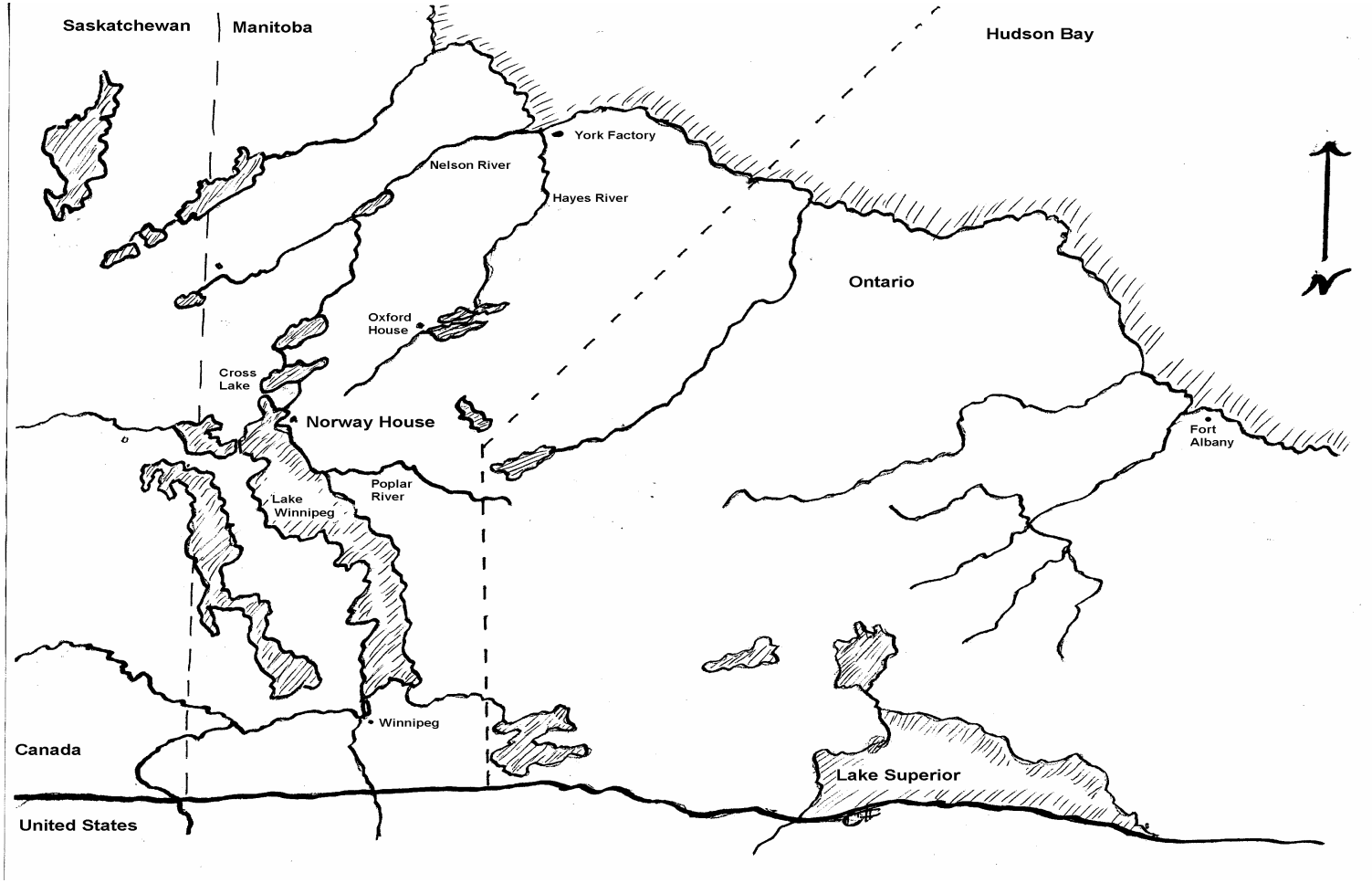

Figure 3.1: Norway House in Manitoba, Canada

The European fur trade, Christian missionaries, and various governmental policies strongly influenced the cultural patterns of the native people in the Norway House area (Hallowell, 1992). Although European influence had many negative effects on the native people and their culture, the fur trade actually reinforced a seasonal movement pattern that was traditional to these people (Long, 1995). Historically, these people were nomadic hunter-gatherers whose entire existence revolved around the seasonal climate and the subsistence available during each season. Using toboggans and snowshoes, they would hunt and trap in the late fall, winter, and early spring when the weather was cold and the fur-bearing animals were most plentiful. In the spring, when the ice began to break up, they would move to favorite fishing grounds to fish and hunt for waterfowl. When the waterways were clear of ice, hunting and fishing continued with canoes as the favored mode of transportation (Flannery, 1995; Hallowell, 1992; Tanner, 1992).

In addition, the population's dispersal on the landscape varied seasonally following an aggregation-dispersal pattern (Hallowell, 1992; Herring, 1994). In the fall, the population broke up into winter hunting groups composed of members of an extended family who traveled together to family hunting grounds. The community recognized a family's right to use these winter hunting grounds year after year and to pass those rights down to future generations. The winter hunting group was composed of an average 16 people with one male hunter for every three non-hunting individuals (Hallowell, 1992), but in practice the composition of the group varied and the kinship ties could not always be traced

(Rogers, 1969). Permanent winter bush camps were established in the winter hunting grounds and the family hunting groups dispersed from there (Flannery, 1995). Periodically, after the fur trade was established, the hunters would return to the post to trade their furs and pick up supplies (Herring, 1994). Some hunting groups would have trap-lines up to 300 miles from the post (Stone 1926), so travel to the post was infrequent in the wintertime.

In the summer, the various small winter hunting groups would gather in larger summer fishing settlements. The original extended family groups or winter hunting groups would still be recognized as individual entities in the fishing settlements but would share and cooperate with other hunting groups in close proximity (Hallowell, 1992). In the summer, when the community was gathered, feasts, festivals, marriages, and trade were common (Meyer, 1985; Flannery, 1995). After the fur trading post was established, it became a common place for the community to meet in the summertime (Herring, 1994).

The seasonal aggregation-dispersal pattern of land use and movement allowed these sub-arctic people to adapt to their harsh environment (Hallowell, 1992). In the wintertime when food was scarce and a large population could not be sustained in a small area, the people dispersed on the landscape in small hunting groups. In the summertime when food was more plentiful and a larger population could be sustained, the people congregated in larger settlements.

It is believed that the 1918-1919 influenza pandemic arrived in Canada in the late spring or early summer of 1918 with troops returning from Europe. One out of every six Canadians contracted the flu and 30,000-50,000 people died. The

same characteristics of the disease were seen in Canada as were seen elsewhere. Mortality was very high in young adults and in many isolated populations. Many native settlements and hunting camps were devastated. The flu seemed to strike some families very hard and miss others families completely (Pettigrew, 1983). The warning signs of the flu were very characteristic with symptoms such as semi-conscious stupor, cracked lips due to fever, and incontinence, in addition to the usual flu symptoms. The prognosis was considered terminal if the flu victim's face turned ashy purple due to hypoxia and they emanated a smell of death referred to as an "influenza stench" (Herring, 1994, pp. 81).

The flu did not reach Norway House for several months. Excerpts from the Hudson's Bay Company (HBC) post journals suggest that the flu epidemic came from Clearwater Lake, a community to the north of Norway House, traveled through Cross Lake, and arrived at Norway House with a mail packet on December 4, 1918 (HBCA 1918-1923: fo. 9). This was the last, recorded mail packet from an epidemic-impacted community prior to the Norway House epidemic (Herring, 1994). An HBC chore boy was inflicted with an ailment, possibly the flu, five days after the Cross Lake packet was received. This timing is consistent with the incubation period of the flu (Bannister, 1983), suggesting that the flu may have come with the Cross Lake mail packet.

The flu epidemic arrived at the Norway House post in December and spread throughout the community by the frequent comings and goings of dog-trains between posts and hunting camps (Herring, 1994). The infectious period of the

influenza virus, which is 3 to 7 days (Chin, 2000), allowed infected individuals time to travel to the winter hunting grounds and infect the many families who were out with their winter hunting groups when the pandemic arrived. These families depended on hunting and gathering for food. Frequently, the hunting camps were too far from the post to get help or additional food so they were extremely vulnerable to the flu (Herring and Sattenspiel, 2003). During the epidemic, schools and churches closed and mail delivery stopped because so many people were sick (Herring, 1994).

By February of 1919, approximately six weeks after the epidemic started at Norway House, it was over and an estimated 18% of the population had died (Herring, 1994). The mortality from the flu was much higher at Norway House than the estimated 3% mortality in other indigenous Canadian communities (Graham-Cummings, 1967; Herring, 1994). The actual factors that increased the mortality at Norway House are unknown. One factor that might have increased the mortality at Norway House was the lack of available surplus supplies. The people were hunters and gatherers who depended on the acquisition of meat from the bush on a daily basis (Herring, 1994). If the hunters were unable to hunt due to illness or death, starvation was a possibility for the entire winter hunting group. Starvation would have weakened the immune system and made people more susceptible to the influenza virus and other infections. Other factors that could have influenced mortality would be the presence of pre-existing diseases such as tuberculosis or secondary bacterial or viral infection. Dealing with sickness and death during the epidemic would have put everyone in the

community under a great deal of stress leaving them more susceptible to infection and death. In this sub-arctic terrain, burial of the dead was usually not possible in the winter so the bodies were placed on rooftops or stacked in a place safe from animals until the spring thaw (Herring and Sattenspiel, 2003). Some communities close to Norway House, such as God's Lake and Oxford House, showed no signs of the flu (Sattenspiel and Herring, 1998). Not only did the epidemic hit different communities with different intensities but also at Norway House, some families were nearly wiped out whereas others were untouched (Herring and Sattenspiel, 2003).

It is unknown whether the high mortality was due to the characteristics of the flu virus, the physiology of the people, the sociocultural aspects of the people, or a combination of all three. In this project, I used characteristics of the flu virus, such as disease transmission and infectious period, and cultural characteristics of the Norway House people, such as seasonal population movement, in a computer simulation in an attempt to answer a few of these questions.

Chapter 4: Modeling an Epidemic using Agent-Based Computer Simulation

The simulation of the 1918-1919 influenza epidemic in the Norway House community required the combination of several modeling techniques. This chapter describes the basic concepts of modeling, epidemic modeling, computer simulation, and the agent-based modeling used in this project.

Before the computer simulation was constructed, a visual model of the Norway House community, as shown in Figure 4.1, was created using ethnographic information. This visual model contains the Norway House fort and the four hunting camps that are used by the winter hunting groups in the winter scenario. The arrows represent the paths that are used by the men to travel back and forth between the fort and the camps. The mailman introduces the influenza virus to the fort from an outside source. This visual model was very simplistic, containing only the basic components in the community that might influence the spread of the influenza virus. In order to comprehend a complex system, it is necessary to break the system down into simple components. The importance of these components to the entire system can be determined by the analysis of these individual components. A model aids in this process. It is a simplification of a real life system (Gilbert and Troitzsch, 1999; Sattenspiel, 2003). This simple model was used as the basis for the construction of the environment in the computer simulation.

A second visual model, as shown in Figure 4.2, was constructed to portray the behavior of the epidemic as it passed through the community. This model was based on fundamental epidemiological principles. Four types of people or agents were constructed for this model. The first type of person is the susceptible (S) individual who is at risk of becoming infected with the flu virus. The second type is the exposed (E) individual who is infected with the flu virus but is not currently infectious. In order for a susceptible person to become an exposed individual, the flu virus must be transmitted from an infected person to the susceptible individual. The third type of person is the infectious (I) individual who is infected with the flu virus and has the potential to transmit the virus to susceptible individuals. An exposed person will become an infectious individual after a period of time called the latent period of the disease. The fourth type of person is the recovered (R) individual who has recovered from the flu, is no longer able to transmit the virus to other individuals, and is unable to contract the flu virus from infected individuals. An infectious person becomes a recovered individual after a period of time called the infectious period of the disease. A model with these disease stages is called an SEIR model (Sattenspiel, 2000; Sattenspiel, 2003). This SEIR model was used as the basis for the construction of the agents and the rules in the computer simulation. Demographic information from ethnographic data was used in building the population structure in the simulation.

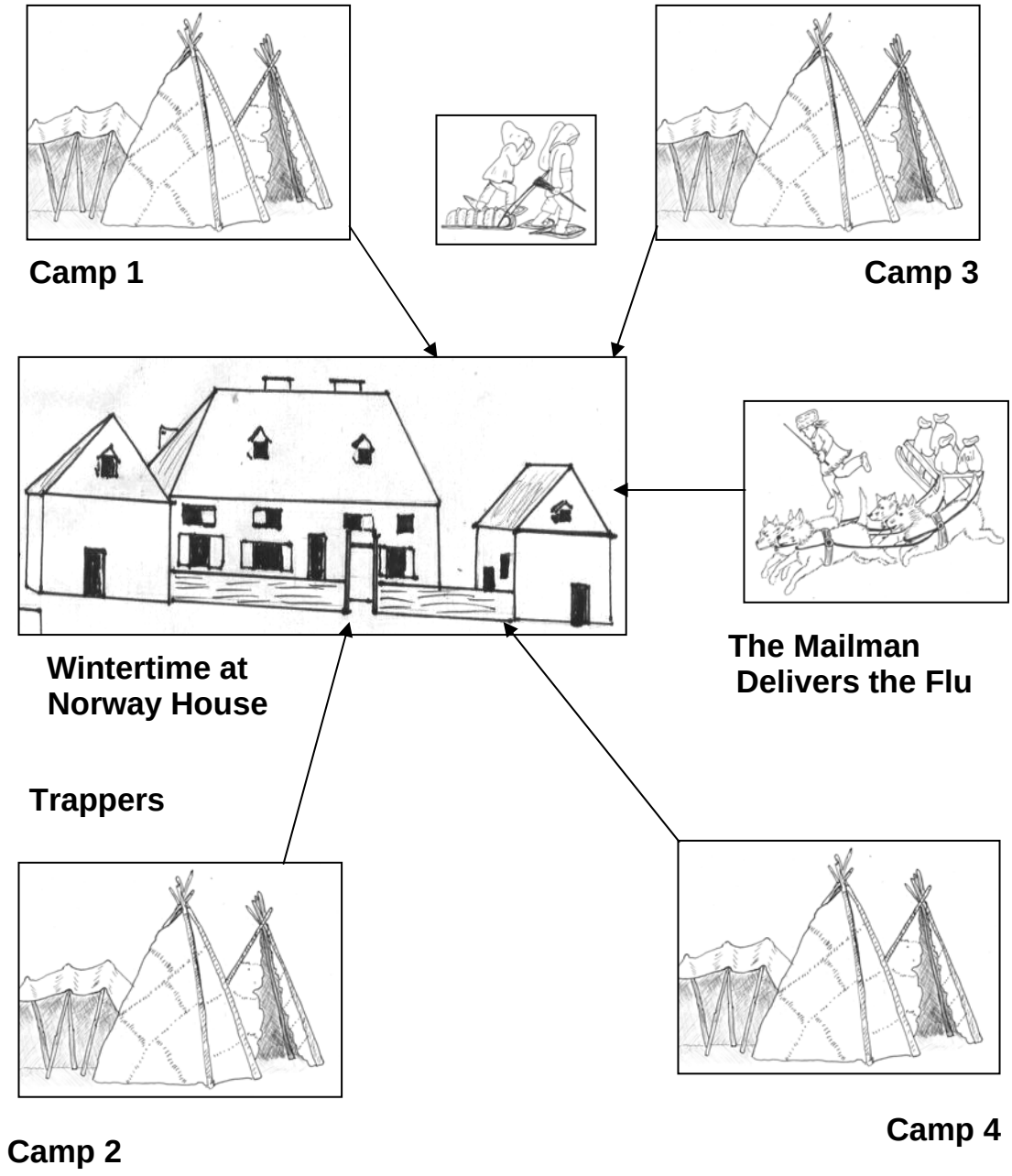

Figure 4.1: A visual model of Norway House in the winter includes the fort, four camps, and the mail carrier who introduces the influenza

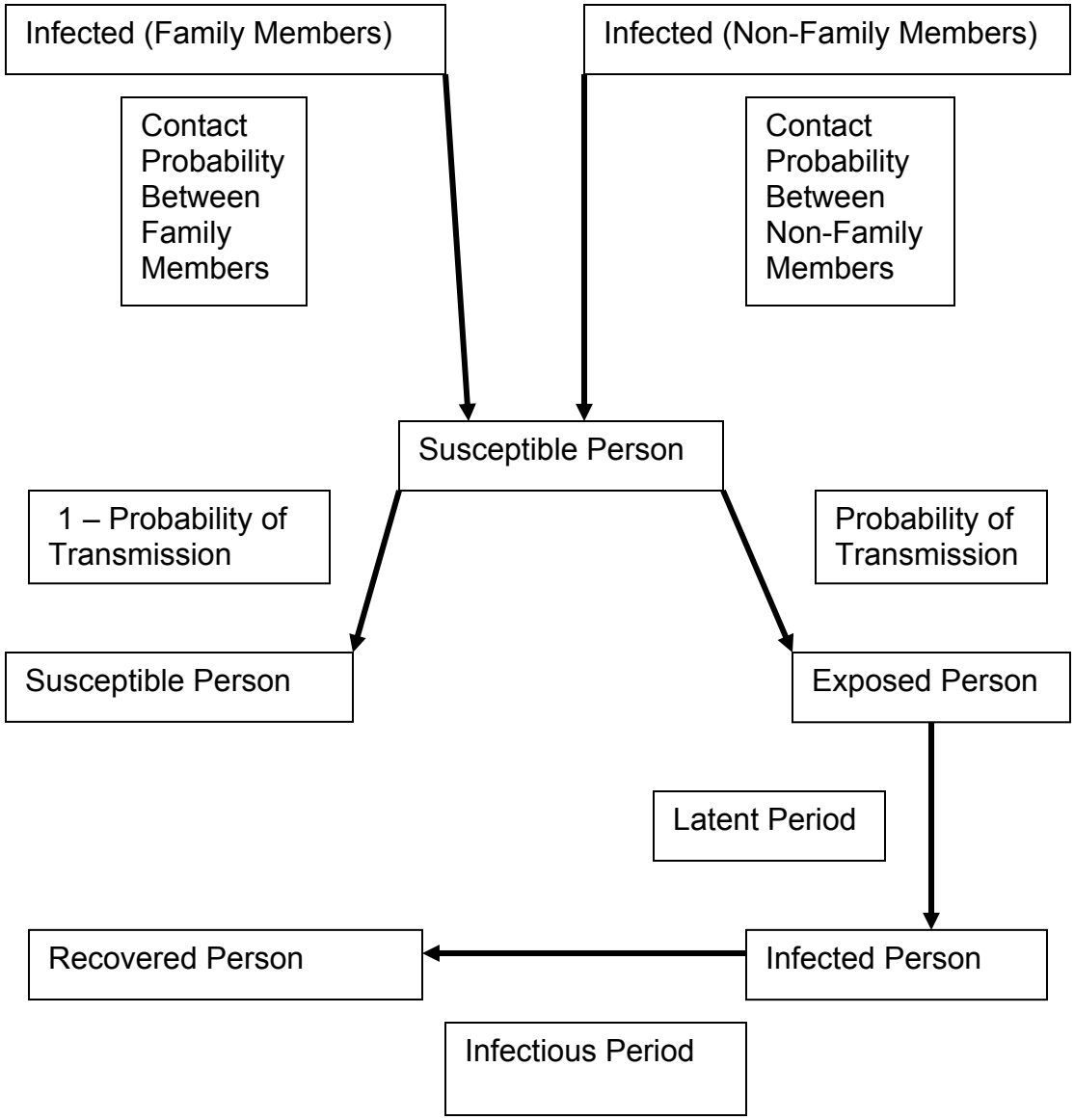

Figure 4.2: A basic visual model of the spread of the influenza virus through the community.

Computer simulation is a type of modeling that can represent the dynamics of a system (Gilbert and Troitzsch, 1999). In this project, the simulation represents the movement of the population and the spread of the epidemic through the population. The input for the simulation is the basic assumptions that have been made about the system. The output of the simulation is the observations made as the simulation runs. The goal is to gain a better understanding of the system by studying the relationships of the components within the system (Gilbert and Troitzsch, 1999). Population movement, population structure, contact time between individuals, and disease characteristics were studied to determine their influence on the spread of the influenza epidemic through the community.

Computer simulation can be used for a variety of purposes including a better basic understanding of a system, predictions, training, entertainment, and experimentation (Gilbert and Troitzsch, 1999). The computer simulation allows for “experimentation” on human populations, when the real experiments would not be ethical or possible (Sattenspiel, 2003). The computer simulation created for the 1918-1919 influenza epidemic in the Norway House community has allowed for experimentation, training, entertainment (at times), and hopefully, a better basic understanding of the spread of the epidemic in that community.

An agent-based computer simulation technique was used for the modeling in this project. Other names for agent-based modeling are individual-oriented, distributed artificial intelligence based modeling (Doran et al., 1994), and artificial societies (Epstein and Axtell, 1996). An agent-based computer simulation is composed of a heterogeneous population of agents dispersed on a landscape

following specified rules of behavior. The individual agents are assigned specific attributes, which can change over the course of the simulation. Agents are separate from the landscape and other agents. Agents can interact with other agents and with the landscape during the simulation. An agent-based model has a "bottom-up" structure because it starts with individuals following their own rules of behavior in a specified environment moving through time, and the output of the model is the emergent behavior of the population (Epstein and Axtell, 1996).

Computer simulation and agent-based modeling are reasonably new to the social sciences (Gilbert and Troitzsch, 1999; Epstein and Axtell, 1996). Kohler et al. (2000) have used computer simulation and agent-based modeling in conjunction with a geographical information system (GIS) to reconstruct a series of paleoenvironments for the Mesa Verde study area in the American Southwest between A. D. 901 and 1287. Ethnographic, archaeological, and environmental data were used to construct landscapes, households, and rules of behavior at different time periods for the model. The resulting computer simulation was used to answer research questions about settlement dynamics over time among the ancient Pueblo people.

The use of agent-based modeling by social scientists for the study of infectious disease is rare (Sattenspiel, 2003). Epstein and Axtell (1996) demonstrated that agent-based computer simulations have the potential to be useful for infectious disease research by modifying their Sugarscape computer simulation to create an immunology-epidemiology model.

One of the advantages of agent-based computer simulation over traditional models is that an epidemic can be introduced into a dynamic social context. The simulation portrays a heterogeneous population of individuals interacting with each other and their landscape over a period of time (Epstein and Axtell, 1996). A second advantage of agent-based modeling is that it is a stochastic modeling technique, which means it considers randomness. This attribute is useful in the study of small populations (Kohler et al., 2000). In the Norway House model, random number generators working in concert with probability functions within the program provide the stochastic nature of the model. A final advantage of agent-based modeling is that a historical population and landscape can be simulated and experiments performed which allow for the examination of contributing factors to specific outcomes (Dean et al., 2000). In the case of the Norway House community, factors can be analyzed which may have led to the high rate of infection and mortality during the 1918-1919 influenza epidemic.

In the next chapter, these basic modeling concepts will be applied to the construction of the agent-based computer model simulating the spread of the 1918-1919 influenza epidemic through the Norway House community. The landscape, the agents and their attributes, the rules of behavior, and the emergent epidemic dynamics for the Norway House model are explained in detail.

Chapter 5: The Construction of an Epidemic Model Using Computer Simulation

This chapter describes the construction of the agent-based computer simulation used in this project. The model is discussed in terms of the human scenario as simulated on the computer. Humans, landscapes, movements, and seasons are expressed as if they were actually part of the Norway House community in 1918-1919. This type of model is easier to construct, troubleshoot, and analyze when a direct connection is made between the people, who are being studied, and the computer simulation. However, it is important to keep in mind that the simulation is only a rough sketch of the people and their activities during the epidemic.

Gilbert and Troitzsch (1999) suggested five basic steps to follow when research involves computer simulation — model design, model construction, verification, validation, and publication. Using these steps as a guideline, I will describe how the model for this project was designed, constructed, and tested in this project. Just as the model is a simplification of the influenza epidemic at Norway House, this description is a simplification of the model construction for this project. The construction of this model was not a straightforward process. Models and methods were evaluated and rejected. Mistakes were made and corrected. Research questions evolved over time.

This project is centered on one major methodological issue and two research questions. The first step was the construction and verification of an agent-based

computer model that would simulate the spread of the 1918-1919 influenza epidemic through the Norway House community. Once this step was completed, the simulation was used to test two research questions. First, could the seasonal population movements of the Norway House community influence the spread of the flu through the community? Second, could a winter epidemic differ from a summer epidemic predominantly due to changes in seasonal population movement? In other words, could the change in a single cultural variable, such as seasonal population movement, strongly influence an epidemic.

Java™ was chosen as the computer language for the model due to its compatibility with agent-based computer simulations (Gilbert and Troitzsch, 1999). Java™ is an object-oriented language, that is well documented, relatively easy to learn, and can cross computer platforms (Flanagan, 1999).

The choice of a framework or tool kit for an agent-based computer simulation was a trial and error process, which required a considerable amount of time to accomplish. Many of the agent-based framework programs were difficult to learn, difficult to run, and ran slowly. The University of Chicago's Social Science Research Computing's agent-based computer simulation toolkit called RePast (Recursive Porous Agent Simulation Toolkit) was chosen on the advice of a colleague who assisted in the construction of this model. RePast utilizes Java™ as a programming language and can be run on a variety of platforms. In addition, RePast is relatively easy to learn, making it a user-friendly tool. RePast provides a framework for constructing and running a simulation, collecting data, and displaying the results.

The first step of model construction was a simple visual model (see Figure 4.1) that represented the Norway House community at the time of the epidemic. This model of the Norway House community included the fort and four hunting camps at varying distances from the fort. The illustration of the mail carrier is included to indicate the introduction of the flu into the community. A second visual model (see Figure 4.2) was constructed to portray the behavior of the epidemic as it passed through the community. This visual model takes into consideration characteristics of the Norway House people and basic epidemiological principles. Using these simple visual models, the basic characteristics of the community and the epidemic were determined for the computer simulation. Documented information about the influenza virus, the 1918-1919 influenza epidemic, and the Norway House people were used in this project whenever possible within the context of the basic principles of epidemiology and agent-based modeling described in previous chapters.

The basic characteristics of the community required for the computer simulation were the population composition, the probability of contact between different members of the community, and the seasonal population movement patterns.

The census for Norway House in 1916 was 734 people (Government of Canada 1917: 18-19). For the purpose of this research, the population of Norway House was estimated at 750 people. The population was subdivided into extended family groups of 15 members, a number consistent with ethnographic data presented in Hallowell (1992). The population was also

divided into age-gender groups of males aged 20-50 years old (Male), females aged 20-50 years old (Female), and a more generalized group which included the young and the old (Child). These categories were chosen because the mortality risk for a person over 60 years old was the same as the risk for a normal annual influenza epidemic (Stevens et al., 2004), while mortality from the 1918-1919 flu pandemic was higher than normal in young adults of both sexes (Reid et al., 1999; Stevens et al., 2004; Stuart-Harris, 1985; Schoenbaum, 2003). Norway House Cree First Nation Treaty Annuity Lists, which provide a type of census information, indicate that the population was comprised of approximately one-quarter “Male”, one-quarter “Female”, and one-half “Child”.

In this project, age-gender groups were used only for population movement analysis. In future projects, these groups will be used to test the effect of differential probability of contracting the flu upon contact and differential probability of mortality. The probability of transmission of the virus and the probability of mortality after infection could be varied in the Male and Female young adult groups to determine what combination of factors would increase the number of deaths in young adults during the epidemic. A higher probability of contact between individuals was assumed for members of the extended family or hunting group. A lower probability of contact between individuals was assumed for members of different hunting groups. The extended family group remained important to these people in both winter and summer (Hallowell, 1992).

In the winter, the population dispersed on the landscape to hunting camps. The men would periodically return to the fort from the hunting camps for supplies

(Herring, 1994). An assumption was made that the men would complete their business at the fort in one day and return to the camp. This seems reasonable since their families depended on the men's assistance for survival in the harsh sub-arctic climate. In the summer, the population congregated around the fort (Hallowell, 1992; Herring, 1994).

The basic characteristics of the 1918-1919 influenza epidemic at Norway House that were used in the construction of this model were the time and place the flu entered the community, the probability of infection upon contact with an infected individual, the infectious period of the flu, the latent period, and the duration of the epidemic. The flu epidemic probably entered the community on December 4th, 1918 with the mail packet from Cross Lake (HBCA 1918-1923: fo. 9; Herring, 1994). This computer simulation introduces a single infected person into the fort from outside the community. The basic structure and assumptions for the model are summarized in Table 5.1.

Once the basic visual model was constructed and the characteristics of the community and the epidemic were established, the programming of the agent-based computer simulation began. The actual computer program, written in Java™, is provided in Appendix A. Following the general principles of agent-based modeling, a heterogeneous population of agents, an environment for the agents, and established rules of behavior for both the agents and the environment were constructed. Agents are the "people" of an agent-based simulation. The environment is the landscape on which the agents move and

interact. The rules of behavior determine the agent-agent and agent-environment interactions during the simulation (Epstein and Axtell, 1996).

Table 5.1: The Basic Structure and Assumptions for the Model

- Basic Structure
    - The grid size for the world is 100 X 100
    - Each simulation runs over 200 days
    - 1000 simulations are run and averaged for each parameter change
    - The output is expressed as characteristics of the epidemic
        - Duration of the epidemic (days)
        - The peak time of the epidemic (days)
        - The peak number of cases during the epidemic
        - The total number of cases during the epidemic
        - The percent of the population that was infected during the epidemic

- Basic Assumptions
    - Population = 750
        - Male (20-50 years old) = 0.25 of population
        - Female (20-50 years old) = 0.25 of population
        - Male and Female (<20 and > 50 years old) = 0.5 of population
    - No birth rate
    - No death rate
    - Everyone recovers from the flu
    - A single person introduces the influenza virus into the fort on Day 20
    - Each person belongs to a family hunting group
    - Summer Scenario
        - Everyone congregates in the fort
        - Everyone moves randomly in the fort
    - Winter Scenario
        - Everyone is divided between the fort and 4 camps
        - Everyone moves randomly within their designated area
        - No Females or Children travel
        - Males in the fort do not travel
        - Periodically the Males in the camps travel to the fort
        - Males travel alone
        - Males remain in the fort for 1 day and return to their camp

Parameters are the input variables for the computer simulation and can be varied over a range of values or set at a specific value. Four types of parameters were established for this project: population, contact, movement, and disease parameters. Population parameters included the total number of people in the Norway House community, the number of people at the Norway House fort, the number of people in the hunting camps dispersed on the landscape around the fort, and the age-gender composition of the population. Contact parameters included the number of people in each family, the number of families in the Norway House community, the location of the families, the probability of contact between family members, and the probability of contact between non-family members. Movement parameters included the distance between each camp and the Norway House post, the probability of staying on the path between the post and the camps, the probability of the "Males" leaving the camp and traveling to the post. Disease parameters included the time of the first infection at the Norway House post, the probability of infection upon contact with an infected person for each age-gender group, the infectious period of the disease, and the duration of the epidemic. The latent period was held constant at a value of one day in this model. Each simulation was run over a 200-day period to allow ample time for the epidemic to run its course. The infection was introduced into the fort on day 20 to allow for mixing of the population prior to the epidemic. The basic parameters used in the computer simulation are summarized in Table 5.2.

Table 5.2: The Basic Parameter for the Computer Simulation

- Population Parameters
    - Number of people at Norway House
    - Number of people in the hunting camps
    - Composition of the population
        - Males (20-50 years old)
        - Females (20-50 years old)
        - Children and Elders
- Contact Parameters
    - Number of families within each camp and Norway House
    - Probability of contact within a family
    - Probability of contact between families
- Movement Parameters
    - Probability of staying on the path between Norway House and the camps
    - Distance between each camp and Norway House
    - Probability of staying in the camp
- Disease Parameters
    - Time of the first infection in Norway House
    - Infectious period of the disease
    - Probability of infection upon contact
        - Males (20-50 years old)
        - Females (20-50 years old)
        - Children and Elders
- Miscellaneous Variables
    - Number of days per simulation
    - Number of simulations performed for each parameter change

Another general parameter that needed to be included in the model was the number of simulations that had to be run for each parameter change. Because the model is stochastic and is initiated with a randomly selected number from a Java™ pseudo-random number generator, each run is different even if the parameters are held constant. In order to analyze a stochastic model, simulations using the same parameters are repeated a number of times and the results are averaged (Kohler et al, 2000). Unfortunately, there does not appear to be any consensus in the literature about the number of times the simulation should be run. In this project, 1000 simulations were run for each parameter change and averaged. The analysis was performed using the average values. An additional test comparing 20 sets of averages of 1, 100, and 1000 simulations was included in the project.

My primary interest was the impact of the flu epidemic on the Norway House community under various conditions. Keeping this in mind, the output of the simulation included the number of susceptible (S), exposed (E), infected (I), and recovered (R) individuals for each time interval in the simulation. Data were collected for the entire population and for the individual hunting camps and the post. The analysis used the acquired data to generate characteristics of the epidemic. These include: the duration of the epidemic, the peak number of infected people during the epidemic, the time when the peak number of people are infected, the total number of people infected during the epidemic, and the percent of the population that were infected during the epidemic. This analysis was repeated for both winter and summer scenarios.

A conscious effort was made to construct a comprehensive model that could be easily modified for future projects. This base model will be available to be modified and used to create simulations for different diseases, different cultural patterns, and different geographic locations.

Verification is the process of confirming that the computer simulation is working as initially planned. This process includes debugging of the computer program, testing the simulation at extremes where the outcome can be predicted, and performing a number of simulations for each parameter change (Gilbert and Troitzsch, 1999). Debugging is a normal function in computer programming and will not be described in this paper. The results of testing extremes with predictable outcomes are shown in the next chapter. The repetition of simulations was described earlier in this chapter. One thousand simulations were run for each parameter change. Additionally, I have found that qualitatively predicting the program output when analyzing the simulation results is extremely helpful in the verification process. Many errors in the program were located and corrected using this simple procedure. For example, as the probability of transmission was increased, I would expect the peak number of cases to increase. If the results from the simulation demonstrated a decrease in the number of cases when probability was increased, I would look for an error in the computer program. If the results from simulation concurred with this hypothetical trend at all but one point, I would recheck the input data for that point and, if necessary, rerun the simulation at that one point to confirm the results. This

process was used to track errors and to confirm true deviations from the predicted trends.

Validation is the process of confirming that the computer simulation is a good model of the behavior that you are trying to simulate (Gilbert and Troitzsch, 1999). In this project, many documented characteristics of the people, the 1918-1919 influenza epidemic, and the influenza virus were included in the model. The verification process established that the simulation behaved in a manner that is consistent with an influenza epidemic. The only quantitative information available from the Norway House epidemic is the duration of the epidemic and the approximate percent of the population who died. The duration of the epidemic can be impacted by many parameters and combinations of parameters in the simulation, so duration alone is not very useful for validation purposes. Mortality was not included in the simulation. The simulation does provide the total number of people infected but the probability of mortality for an infected person is not known for the 1918-1919 flu. It would be advantageous to compare mortality data from the model with the documented mortality in the community during the epidemic. Future research will include extensions of the model in this direction.

The computer simulation used in this project was constructed using many of the basic characteristics of the Norway House community, the 1918-1919 influenza epidemic, and the influenza virus. Once the computer simulation was constructed and verified, it was used to test research questions about the influence of population movement on the transmission of the 1918-1919

influenza epidemic through the Norway House community. The use of an agent-based computer simulation allowed us to ethically experiment with an isolated, historical population during a devastating epidemic in order to learn more about the impact of culturally dictated population movements on the spread of infectious disease.

Chapter 6: The Results from the Computer Simulation

The first test of a newly constructed model is verification that the computer model simulates the appropriate scenario. The agents, environment, and rules must all be tested. Once the model is verified, further analysis is possible. The original hypotheses are tested and new hypotheses are generated. This chapter describes the verification tests performed for this project. Each of the parameters in this study was tested over a range of values for a winter scenario, when the population is dispersed on the land, and a summer scenario, when the population is congregated around the fort. A normal graphical output from the computer simulation for the winter scenario is shown in Figure 6.1. The data output from the simulation provides the number of infected cases over time for the entire population and for specific areas as shown in Figure 6.2. It also provides data on the incidence, which is the number of new cases each day over time (not shown). From this information, characteristics of the epidemic can be determined. These characteristics include the number of days an epidemic lasts (duration); the number of people infected at the peak of the epidemic (peak number); the time when the peak number of people are infected (peak time); the total number of people infected during the epidemic (total number); and the percentage of the total population that became infected during the epidemic (percent infected). Using these characteristics, the effects of each parameter change in a simulated epidemic can be determined.

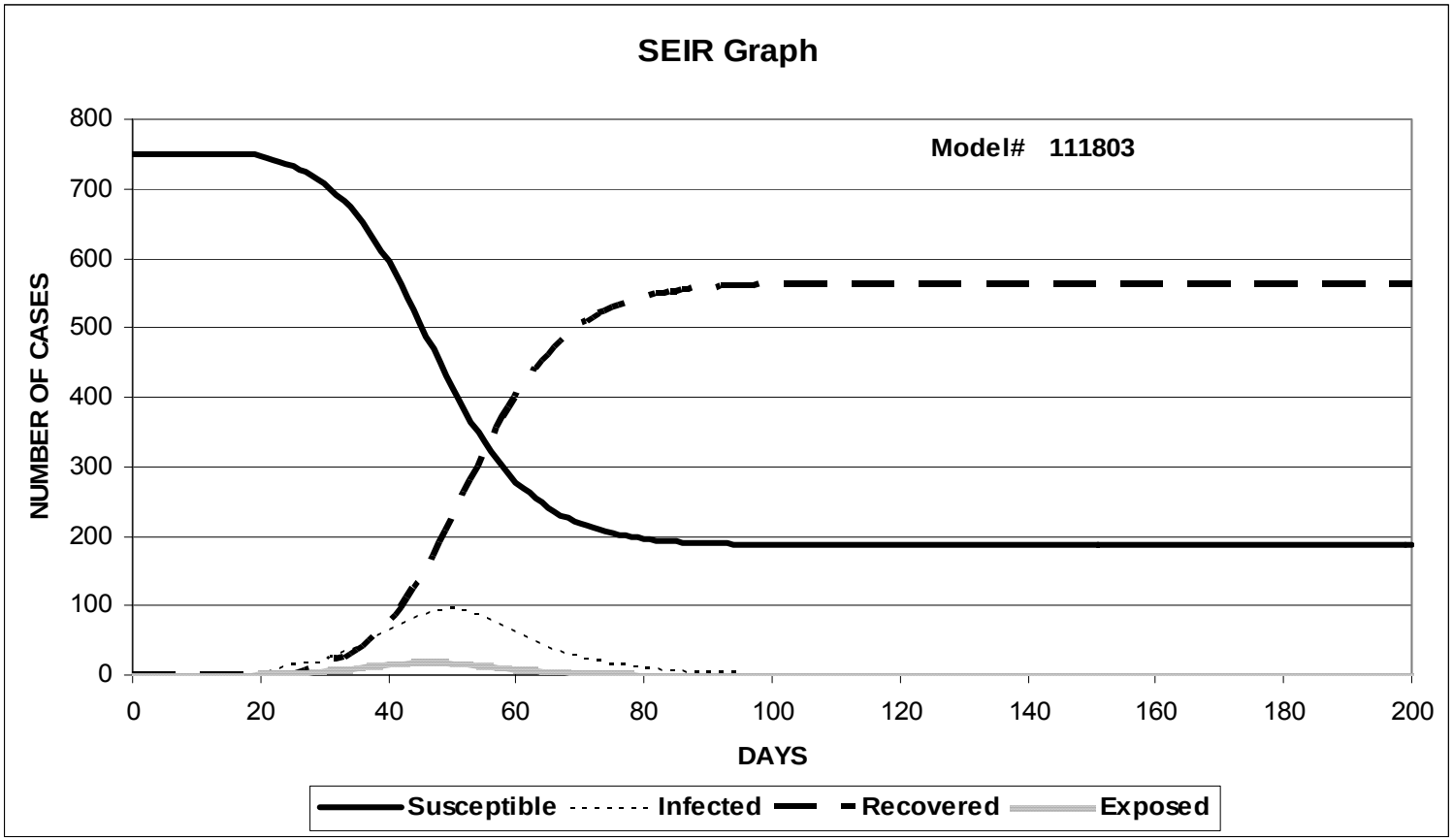

Figure 6.1: A normal graphical output for the winter scenario

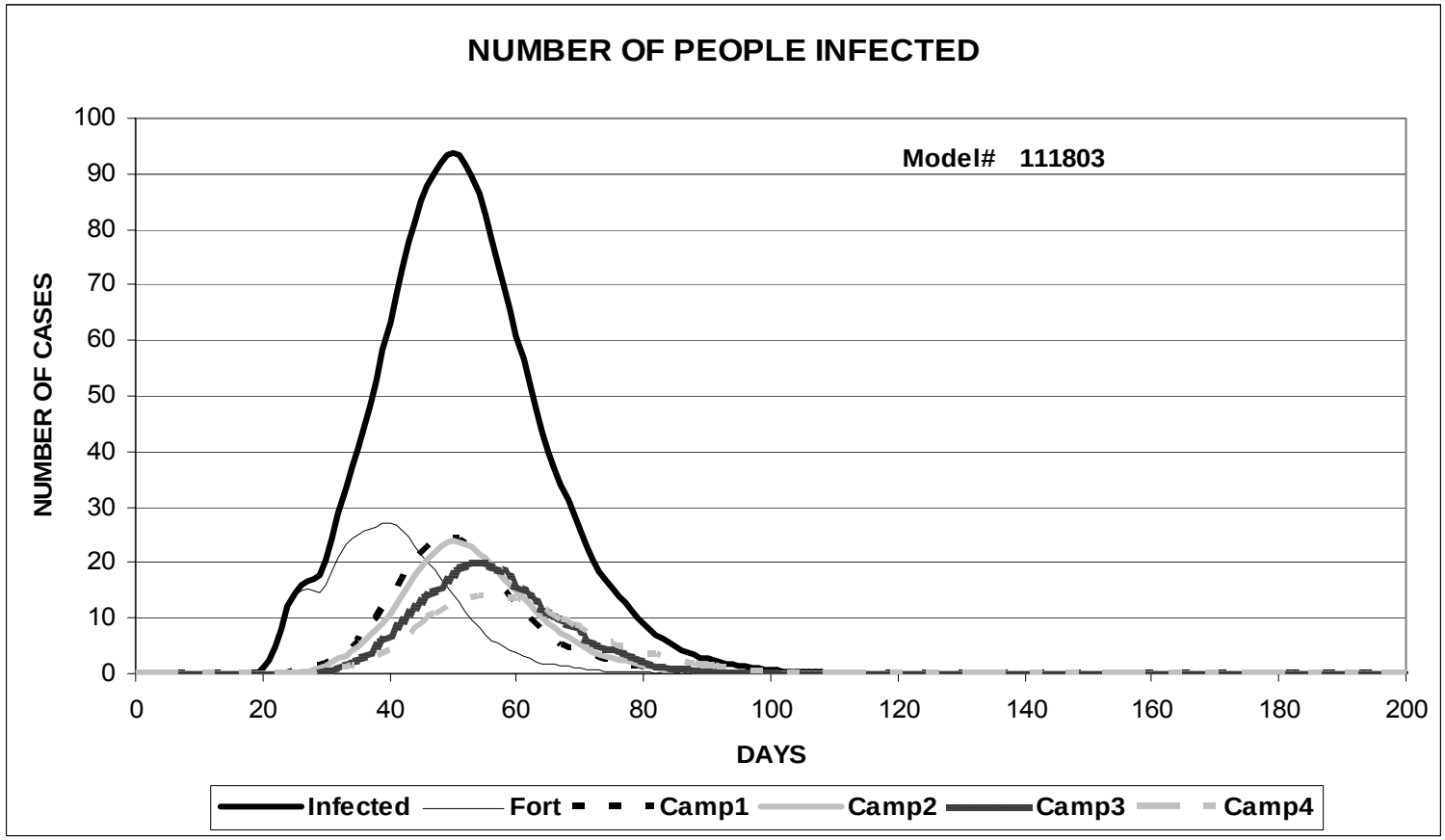

Figure 6.2: The number of infected cases for each area over time

This project consisted of three primary studies: a replication study, a repetition study, and a verification study for each parameter. The replication study established the analytic technique used to compare the curves generated from the stochastic model. The information from this study provided a control range for the epidemic characteristics in the winter and summer scenario. The repetition study was used to establish the number of simulations that needed to be run and averaged for the analysis. This study provided information that was not readily available in the literature and was specific for this model. The verification study was a sensitivity analysis for each parameter used in the computer simulation. This study was performed to determine if each of the parameters was behaving as initially planned during the computer programming stage. A final analysis was performed to study the impact on an epidemic when two of the parameters, probability of transmission and infectious period, are simultaneously varied. A full description of the procedures used and the results obtained from each study will be provided in this chapter.

The Replication Study

The replication study compared 20 sets of 1000 simulations using the same parameters. Each simulation was run 1000 times at the same parameters and averaged. The characteristics of the epidemic, as described previously, were derived from the averages of these runs. This process was repeated 20 times for both the summer and winter scenario. This study was conducted for two purposes. The first purpose was to verify that the model was running in a

consistent manner and the second purpose was to create a normal range of variation between curves generated for identical parameters. When a deterministic model is run repeatedly at the same parameters, it generates the same data. When a stochastic model is run repeatedly at the same parameters, different data are generated. Even when 1000 simulations are averaged and compared to 19 other averages of 1000 in a stochastic model, the data are slightly different. To aid in the analysis of stochastic data, a control range at standard parameters can be used to determine whether a parameter change has an impact on the data. If the data fall outside of the control range with a parameter change, the parameter can be assumed to have an impact on the data. The control range used in this project was useful for identification of the variation between simulated epidemics. The control parameters and the control range for the winter and summer scenarios are shown in Table 6.1, Table 6.2, and Table 6.3. These control ranges were used throughout the project to determine when a parameter change had an impact on the epidemic in a particular scenario.

An analysis of the replication study demonstrates significant differences between the epidemic in the winter and the epidemic in the summer scenario. The epidemic in the winter scenario has a longer duration, longer peak time, lower peak number, lower total number, and a lower percent infected than the epidemic in the summer scenario. This pattern is consistent throughout the project under all parameters. In addition, the winter scenario demonstrates a wider range of results than the summer scenario. The only exception is the peak

number infected which has a similar standard deviation in the winter and the summer. A comparison of the typical winter and summer scenarios at the control parameters is shown in Figure 6.3.

Table 6.1: The control parameters for the winter and summer scenarios

Parameter	Winter	Summer
Probability of staying at the camp	0.99	0.99
Time of the First Infection (Days)	20	20
Probability of Staying on the Path	1	1
Distance to Camp 1 (Days)	2	2
Distance to Camp 2 (Days)	3	3
Distance to Camp 3 (Days)	4	4
Distance to Camp 4 (Days)	5	5
Camp Population Size	150	0
Fort Population Size	150	750
Population Proportion - Male	0.25	0.25
Population Proportion - Female	0.25	0.25
Population Proportion - Child	0.5	0.5
Number of Families per Area	10	50
Probability of Contact within Families	0.5	0.5
Probability of Contact between Families	0.001	0.001
Infectious Period (Days)	5	5
Probability of Transmission for Male	0.2	0.2
Probability of Transmission for Female	0.2	0.2
Probability of Transmission for Child	0.2	0.2
Days per Simulation	200	200
Simulations per Batch	1000	1000

Table 6.2: The Summer Control Range

Summer Control Range	Maximum	Minimum	Std. Dev.
Duration (days)	41	32	2.01
Peak Time (days)	17	16	0.22
Peak Number	369	356	3.17
Total Number	749	746	1.16
Percent Infected	100%	99%	

The summer control range includes the maximum, minimum, and standard deviation for each of the epidemic characteristics

Table 6.3: The Winter Control Range

Winter Control Range	Maximum	Minimum	Std. Dev.
Duration (days)	96	80	3.83
Peak Time (days)	31	28	0.96
Peak Number	98	85	3.07
Total Number	574	530	11.44
Percent Infected	76%	71%	

The winter control range includes the maximum, minimum, and standard deviation for each of the epidemic characteristics

In the winter, the people are dispersed on the landscape so fewer susceptible people make contact with infectious people and the contacts that are made are less frequent. Fewer and less frequent contacts between people result in a lower probability of a susceptible person being exposed to an infectious person and contracting the disease. In other words, there is a lower probability that the virus will be transmitted from an infectious person to a susceptible person. The

epidemic is perpetuated by the males traveling back and forth between the camps and the fort. An infectious period of 5 days allows a male infected at the fort to travel to any of the camps and expose susceptible people to the disease. Fewer and less frequent contacts between infectious and susceptible people result in a longer duration and a lower intensity epidemic in the winter scenario. The increased variability in the winter scenario is probably due to the travel time and travel frequency of the males between the camps and the fort. In the summer scenario, the people are congregated at the fort, so the population density is higher, resulting in a higher probability that susceptible people will encounter infectious people. The number of contacts and the frequency of the contacts between infectious and susceptible people will both increase, resulting in shorter and much more intense epidemics in the summer.

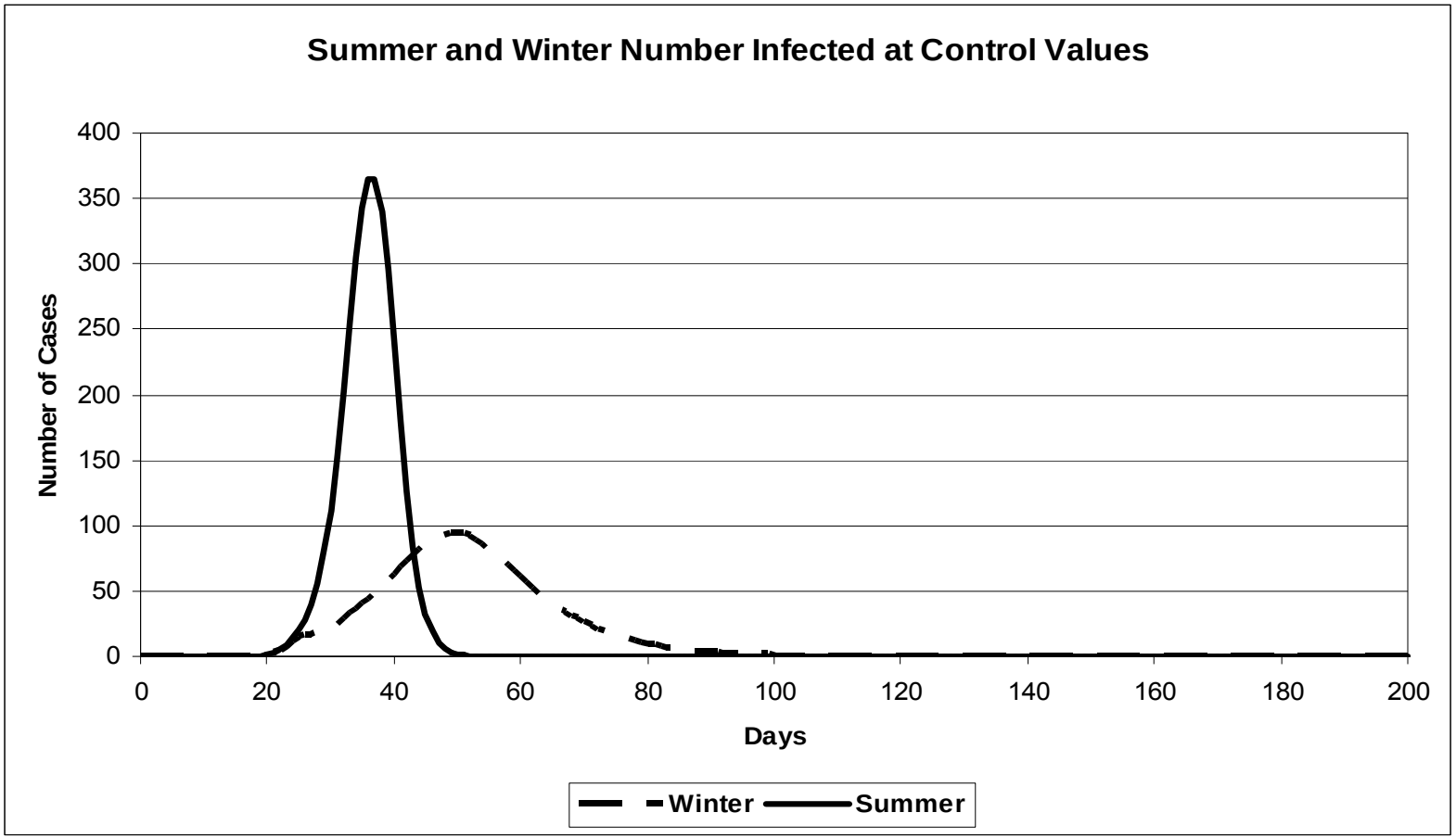

Figure 6.3: A comparison of the typical winter and summer scenarios at control parameters

The Repetition Study

The repetition study was constructed to determine if the average of 1000 simulations are necessary to make a good determination of the basic characteristics of the epidemic in a scenario. To accomplish this study, three tests at identical parameters were run and compared. In the first test, referred to as the Winter 1 test, 20 individual simulations were run and compared as shown in Figure 6.4. In the second test, referred to as the Winter 100 test, each simulation was run 100 times at the same parameters and averaged. This process was repeated 20 times and compared as shown in Figure 6.5. In the third test, referred to as the Winter 1000 test, each simulation was run 1000 times at the same parameters and averaged. This process was repeated 20 times and compared as shown in Figure 6.6. The range of characteristics of the epidemic for each test was derived from the 20 single runs in the Winter 1 test and from the 20 averaged runs in the Winter 100 test and the Winter 1000 test. The epidemic characteristics for each test are shown in Table 6.4. Only the winter scenarios were tested because the winter scenarios are consistently more variable than the summer scenarios due to the males traveling between the fort and the camp in the winter scenario. By testing the more variable of the two scenarios, the highest range of variation between the curves and the widest range of variation for the epidemic characteristics for this project can be determined. With these results, an informed decision can be made as to the number of simulations per parameter change that are required to answer a particular research question.

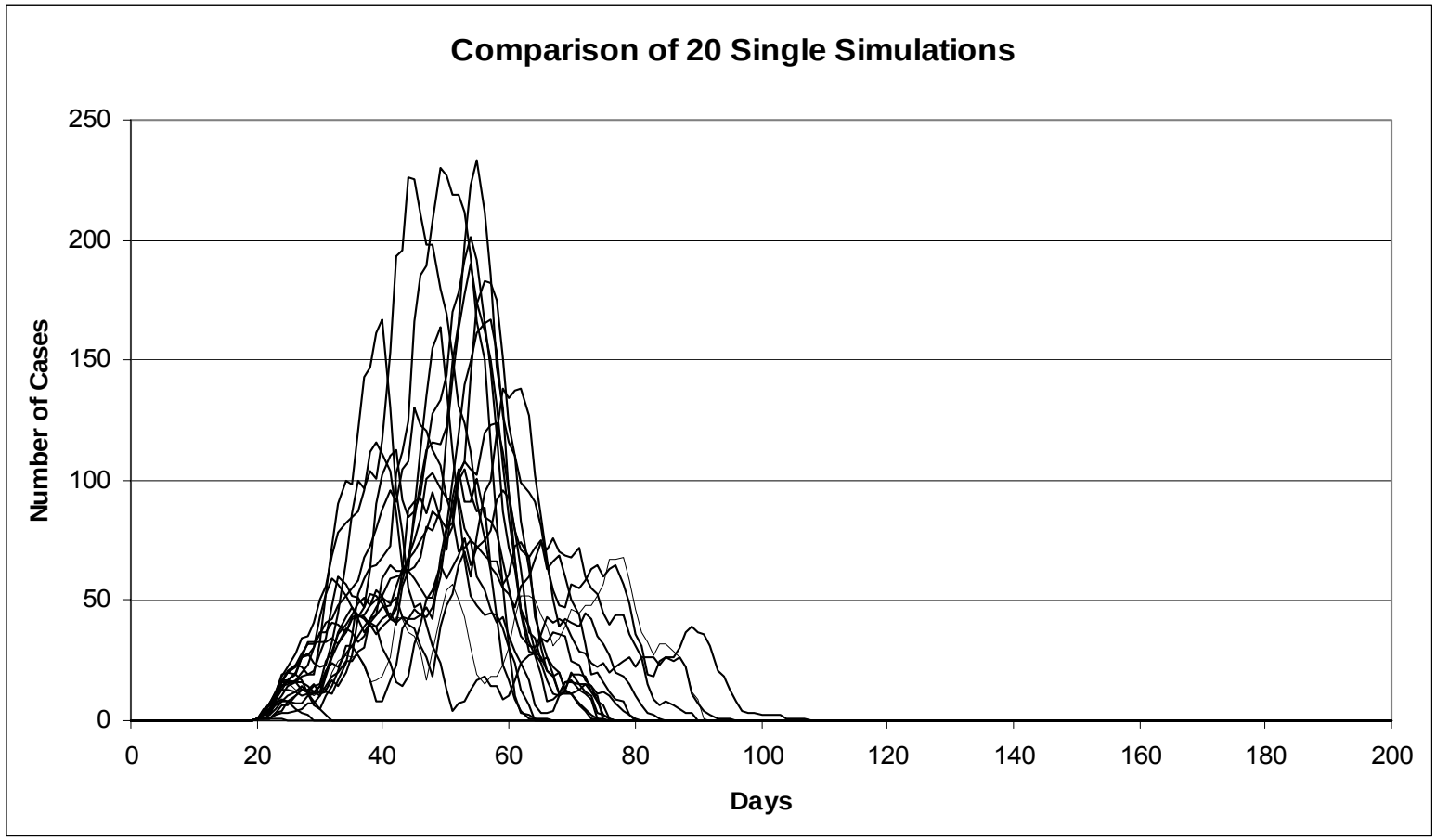

Figure 6.4: The Winter 1 test compares 20 single simulations

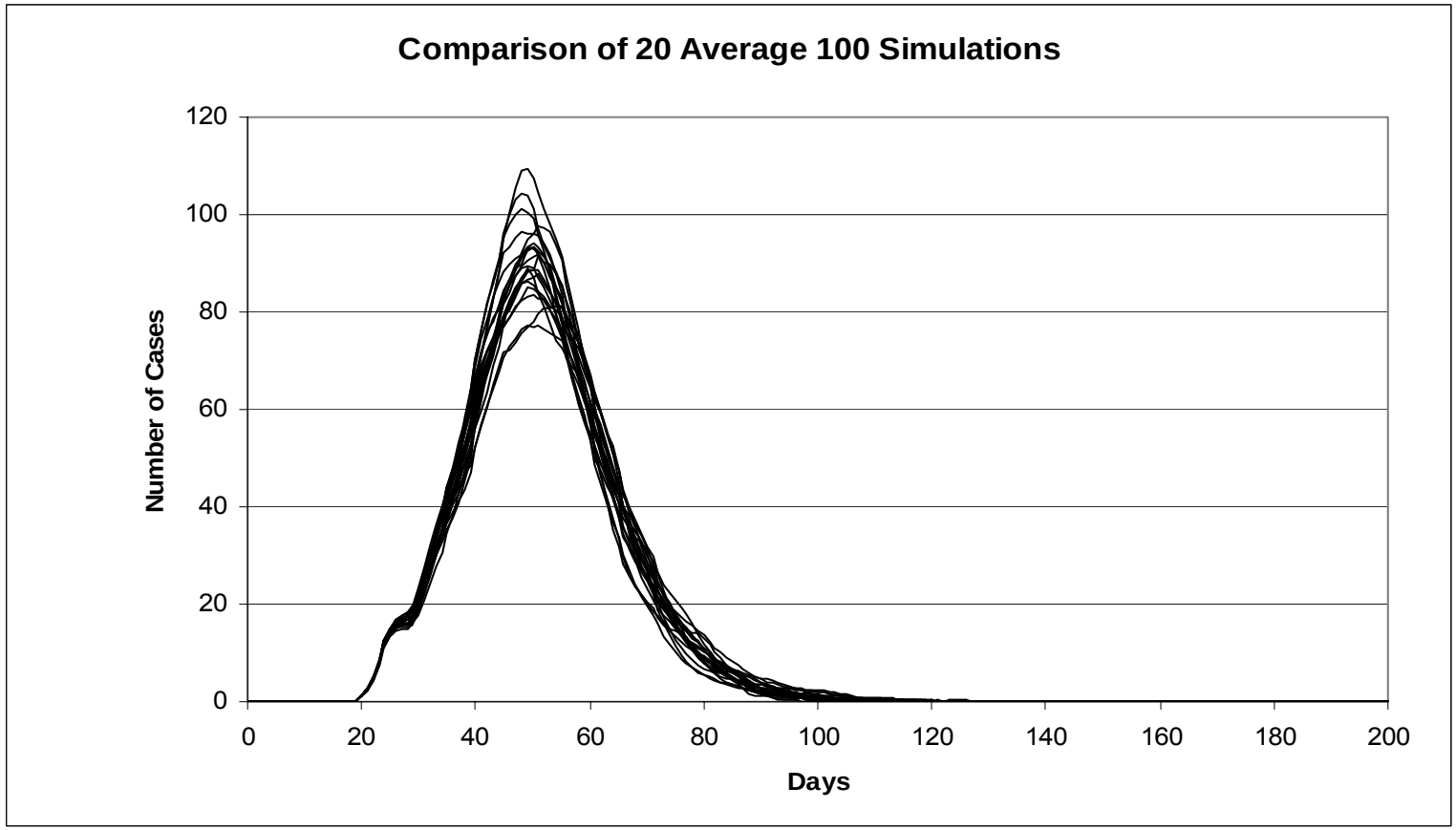

Figure 6.5: The Winter 100 test compares the averages of 100 simulations repeated 20 times

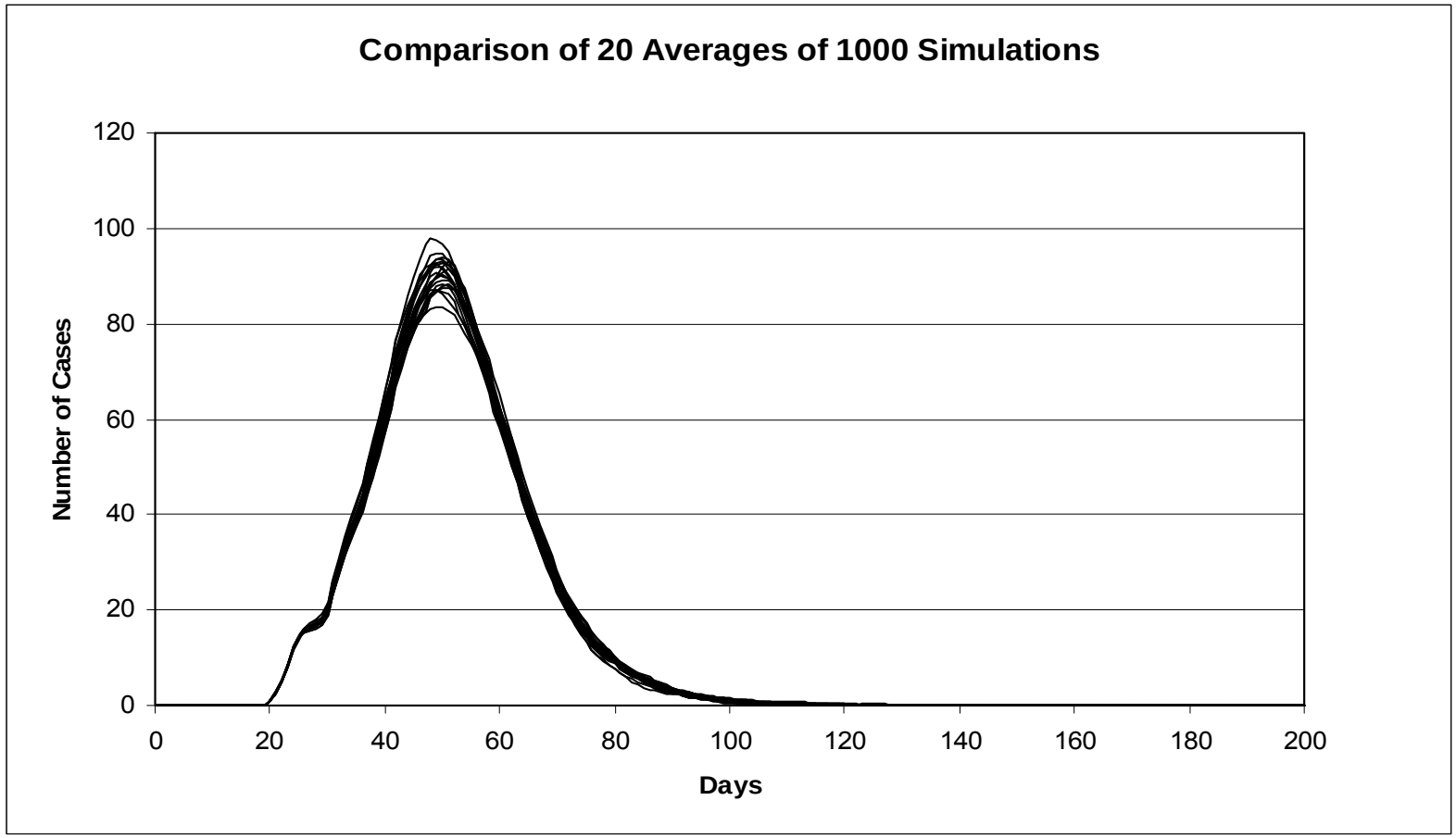

Figure 6.6: The Winter 1000 test compares the averages of 1000 simulations repeated 20 times

Table 6.4: The Epidemic Characteristics for the Winter 1, the Winter 100, And Winter 1000 test

	Winter 1		Winter 100		Winter 1000	
	Range	Std. Dev.	Range	Std. Dev.	Range	Std. Dev.
Duration	5-88	21.00	74-95	5.51	80-96	3.83
Peak Time	1-58	13.10	28-32	1.20	28-31	0.96
Peak Number	1-233	69.58	77-109	7.44	85-98	3.07
Total Number	1-749	230.91	519-603	22.74	530-574	11.44
Percent Infected	0-100%		69-80%		71-76%	

The range and the standard deviation for the epidemic characteristics for the Winter 1 test, the Winter 100 test, and the Winter 1000 test. The Winter 1 test compares 20 single simulations. The Winter 100 test compares the averages of 100 simulations repeated 20 times. The Winter 1000 test compares the averages of 1000 simulations repeated 20 times.

The results from the Winter 1 test yielded a wide range of values, which would be impractical to analyze. The Winter 100 test has a much narrower range of values than the Winter 1 test. The Winter 1000 test has a slightly narrower range of values than the winter 100 test and the general pattern for these tests is very similar. Duration, peak time, and peak number vary less than the total number. The winter 1000 test generated data with a smaller range of variation than the other two tests because random variation is more visible in a test using a very small number of simulations, as in the Winter 1 test, than in a test using a very large number of simulations, such as the Winter 1000 test. These results were expected. What was not expected was the similarity between the ranges of epidemic characteristics for the Winter 100 test and the Winter 1000 test. This comparison demonstrates that the average of 100 simulations for each parameter change would be sufficient for many studies, especially if the time

characteristics of the epidemic are the question of interest. Another factor to consider is the time required to run each simulation. At this point, there are substantial differences in the time needed to run 1000 simulations versus 100 simulations. A more efficient computer program for this model is under development. Because this model was constructed using new computer simulation techniques that were previously untested in this type of epidemic model, a decision was made to err on the side of caution. In this project, all parameters were tested using an average of 1000 simulations.

The Verification Studies for Each Parameter

After the control ranges and the method of analysis were established, each individual parameter was tested over a range of values for both winter and summer scenarios. The other parameters were held constant at control values. The following are the results for each parameter.

- **Camp Stay Probability**

The probability of camp stay refers to the probability that the men stayed in the hunting camps in the winter on any given day. In the winter scenario, the virus could only spread between the fort and the camps by a traveling infectious individual. Further, because the virus was originally introduced into the fort, the only way it could travel to the camps was via the traveling men. The probability of staying in the camp is an important factor to consider in the spread of the virus throughout the entire Norway House community in the winter scenario. The

control value for Camp stay probability is 0.99. The results of simulations varying this parameter are shown in Figure 6.7 and Table 6.5. In the winter scenario, at the camp stay probability of 0 and 0.5, when the men were continuously or frequently traveling, the duration and peak time were shorter than the control values, whereas the peak number, total number, and percent infected were higher. This occurred because the men frequently met a higher number of infectious people; became infectious themselves; and transmitted the virus to susceptible people. The result was a shorter and more intense epidemic. At these probabilities, the epidemic characteristics for the winter scenario approached, but did not equal, the summer scenario characteristics. The winter scenario, at a camp stay probability of 0, differed from the summer scenario because, in the winter scenario, the population was dispersed on the landscape and the men required a period of time to travel from the camps to the fort. This resulted in a longer, less intense winter epidemic. At a probability of 1, the epidemic occurred only in the fort because no one was traveling between the fort and the camps. There was a large change between the camp stay probability of 0.99 and 1 in the winter scenario. This demonstrated that only a few men, traveling between the fort and the camps, were necessary to spread the virus throughout the community at the control parameters. It would be interesting to explore more camp stay probabilities between 0.99 and 1 to determine the minimum probability that is required to transmit the virus to the camps. The longest duration and peak time would probably occur at this probability but that would have to be proven. As expected, the summer scenario was not impacted

by camp stay probability because the entire population was congregated around the fort. There was no travel between the fort and the camps.

Table 6.5: The Epidemic Characteristics for the Probability of Staying in the Camp

Summer				
Camp Stay Probability	**0**	**0.5**	**0.99**	**1**
Duration (days)	33	34	33	34
Peak Time (days)	16	16	16	16
Peak Number	363	362	366	360
Total Number	749	748	749	749
Percent Infected	100%	100%	100%	100%
Winter				
Camp Stay Probability	**0**	**0.5**	**0.99**	**1**
Duration (days)	45↓	45 ↓	83	52 ↓
Peak Time (days)	20 ↓	19 ↓	29	14 ↓
Peak Number	↑ 267	↑ 266	94	22 ↓
Total Number	↑ 739	↑ 738	563	100 ↓
Percent Infected	↑ 99%	↑ 98%	75%	13% ↓

The change in the epidemic characteristics when the probability of staying in the camp is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

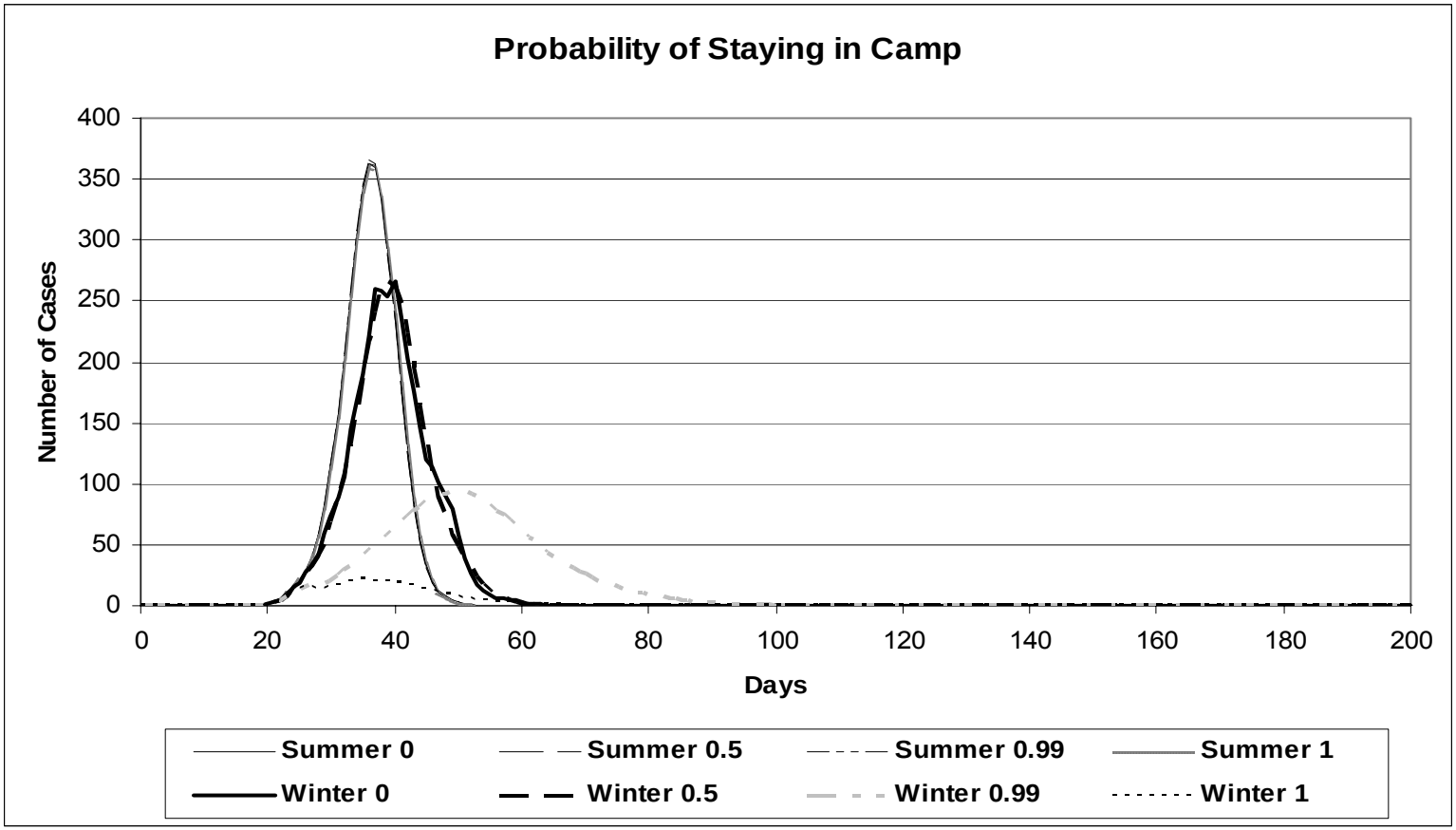

Figure 6.7: The number of infected cases in the winter and summer scenario when the probability of staying in camp is varied

- **The Time of the First Infection (Seed Time)**

The time of the first infection or seed time was the day that the initial infection was introduced into the fort and the epidemic began. This parameter was created and tested to ascertain if the human population was distributed to the camps and the fort prior to the start of the first day of the computer simulation. In addition, the testing determined whether the spatial position of the individual members of the community, which changed daily, influenced the epidemics. The individual members of the community would be in different places on the landscape on different seed time days. The results of simulations varying this parameter are shown in Figure 6.8 and Table 6.6. Although no statistics were done, the seed time appeared to have little or no significant impact on the epidemics for the winter or summer scenarios except at the peak time in the winter at a seed time of 100 days. This value was only 1 day above the control range, which is probably not noteworthy. For this project, the seed time was set at day 20 but, because of the lack of sensitivity of the model to this parameter, day 1 would have sufficed.

The seed time results demonstrate that the population is distributed to the camps and the fort prior to the start of the simulation and that the spatial position of individual members of the community on the day that the virus was introduced had very little impact on the epidemics in either scenario. The impact of introducing the virus into an area other than the fort was not studied in this project. This study was important because it provided information about the actions of the computer simulation, but it is not directly relevant to the real

Norway House community. This is because there are complexities found in real populations, such as immune function of susceptible individuals or the number of people who initially were exposed to the person introducing the virus into the community, which probably altered the course of the actual epidemic that were not incorporated into the model.

Table 6.6: The Epidemic Characteristics for Seed Time

Summer				
Seed Time (days)	**1**	**20**	**50**	**100**
Duration (days)	34	34	34	34
Peak Time (days)	17	17	17	17
Peak Number	369	366	365	363
Total Number	749	749	746	749
Percent Infected	100%	100%	99%	100%
Winter				
Seed Time (days)	**1**	**20**	**50**	**100**
Duration (days)	92	84	85	96
Peak Time (days)	30	30	31	↑ 32
Peak Number	88	94	91	93
Total Number	543	563	552	565
Percent Infected	72%	75%	74%	75%

The change in the epidemic characteristics when the day the influenza virus is introduced into the fort is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

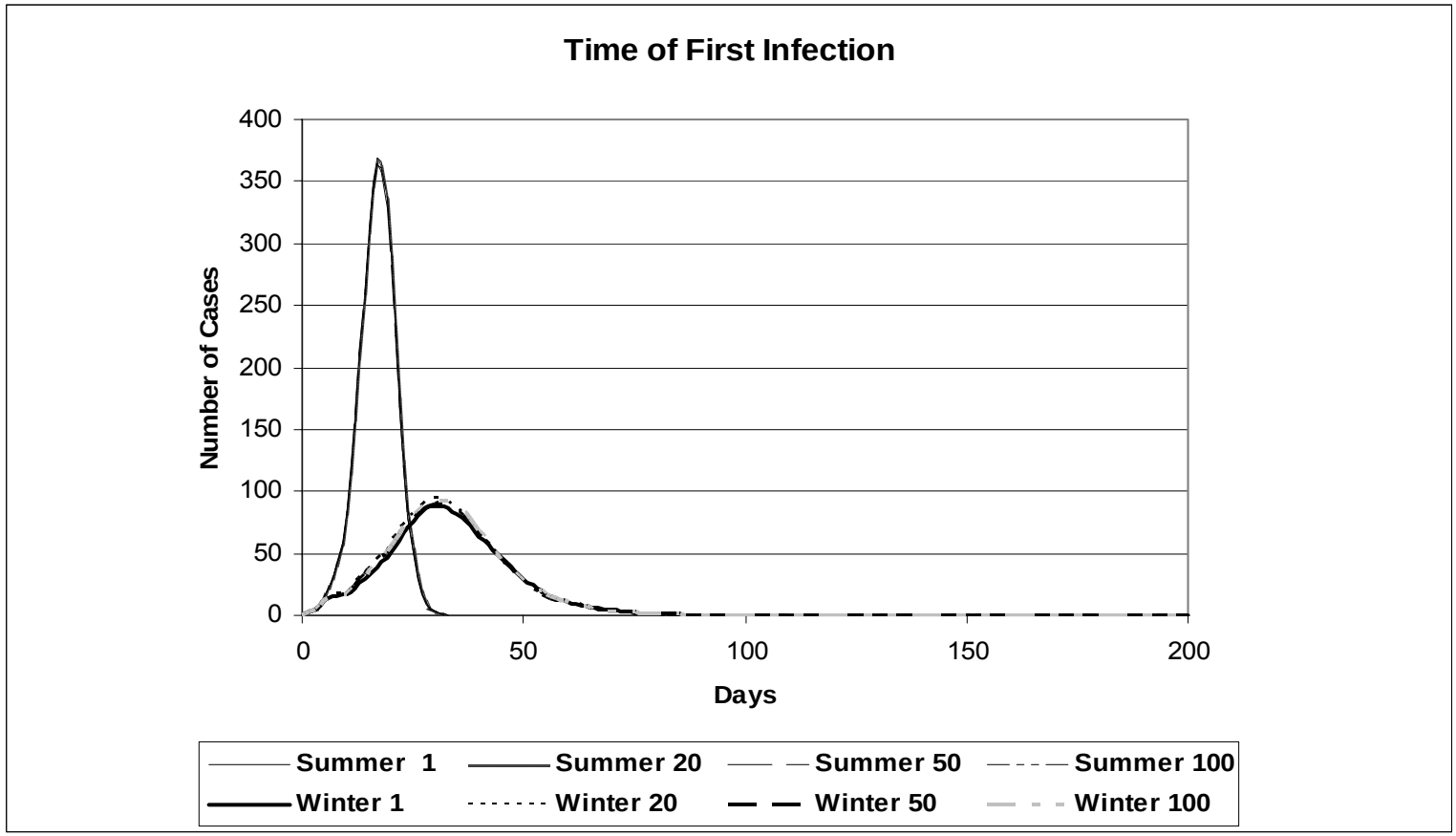

Figure 6.8: The number of cases infected when the day that the influenza virus was introduced into the fort is varied for the winter and summer scenario. Graph legend: Summer 1 is the first day in the summer scenario

- **The Probability of Moving along the Path (Path Probability)**

A set path was constructed between each camp and the fort for the simulation. The probability of moving along the path determined whether a man was moving on the path or wandering off the path. The men were hunters in the Norway House community so it is likely that they left the path for various reasons such as hunting, camping, or visiting a friend. This behavior may have had an influence on the course of the epidemic. Because the results were not consistent for this parameter, the control value for path probability was maintained at 1 for the duration of the project. The men were not allowed to wander off the path. The results of simulations varying this parameter are shown in Figure 6.9 and Table 6.7. The probability of staying on the path had no impact on the summer epidemic because the entire population was congregated around the fort and the paths were not in use. The winter epidemic characteristics only varied from the control range at a path probability of 0 and 0.5. At a path probability of 0, the epidemic only occurred in the fort because the men were not able to reach the fort via the path. At 0.5, the peak number, total number, and percent infected were lower than the control range, but the duration and peak time values were within the control range. At this probability, the men appeared to be able to travel between the fort and the camps but do not have as many or as frequent contact with other people, resulting in fewer people becoming infected. It is unknown why the duration and peak time did not appear to be influenced as significantly as the number of people infected at a probability of 0.5. The model may not be as sensitive to changes in this parameter or not enough data were available to

determine the pattern of change for time components such as duration or peak time. Further testing would be needed to determine the impact of path probability on epidemic characteristics in this simulation.

Table 6.7: The Epidemic Characteristics for the Probability of Staying on the Path

Summer				
Path Probability	**0**	**0.5**	**0.99**	**1**
Duration (days)	36	33	34	33
Peak Time (days)	16	16	16	16
Peak Number	366	363	366	366
Total Number	749	749	749	749
Percent Infected	100%	100%	100%	100%

Winter				
Path Probability	**0**	**0.5**	**0.99**	**1**
Duration (days)	51 ↓	88	88	91
Peak Time (days)	13 ↓	29	29	29
Peak Number	21 ↓	67 ↓	89	95
Total Number	101 ↓	432 ↓	555	572
Percent Infected	13% ↓	58% ↓	74%	76%

The change in the epidemic characteristics when the probability of staying on the path is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

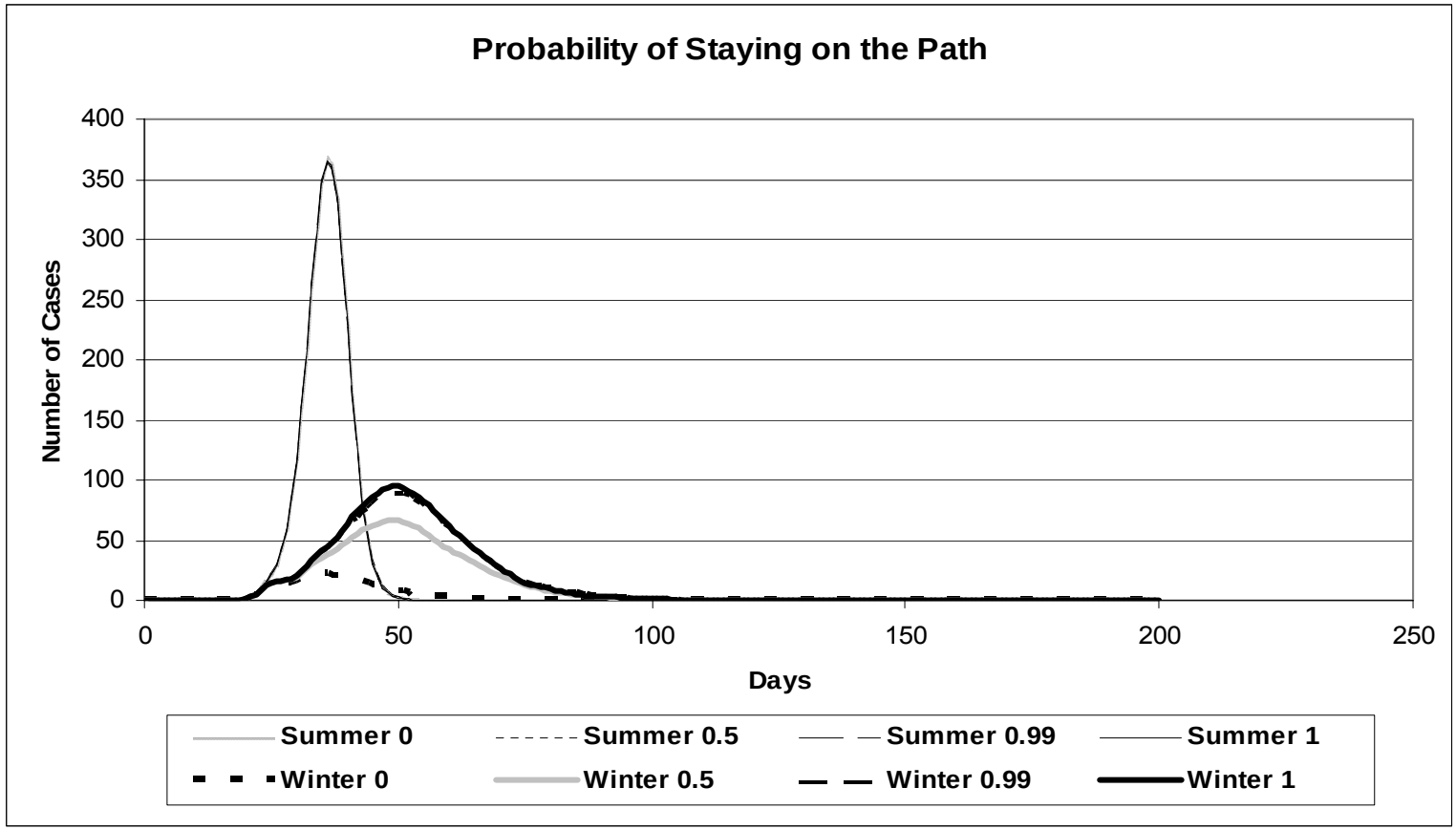

Figure 6.9: The number of infected cases when the probability of staying on the path is varied for the winter and summer scenarios.
Graph legend: Summer 1 is the probability of 1 in the summer scenario

- **The Distance between the Fort and the Camp (Camp Distance)**

The camp distance was the distance in days traveled between each camp and the fort. This parameter was only applicable to the winter scenario when the men were traveling between the camps and the fort. In the summer scenario, people stayed in the area of the fort and did not travel to the camps. Most people in the Norway House community traveled to family hunting grounds in the winter. The family hunting grounds were at various distances from the fort. The four camps are positioned at different distances from the fort as a way to simplify the portrayal of the family hunting ground system seen in the real community. The true family hunting ground system for Norway House was not available in the literature. For this project, the camp-to-fort distances were maintained at two days for camp 1, three days for camp 2, four days for camp 3, and five days for camp 4. The results of simulations varying this parameter are shown in Figure 6.10 and Table 6.8. In this study, the distances that were less than or equal to 5 days were within or close to the control ranges whereas the distances that were greater than 5 days were much lower than the control ranges and only displayed epidemics in the fort. This can be explained by the infectious period, which is 5 days. When the distance from the fort was greater than the infectious period, the men infected in the fort recovered before infecting others in the camp.

There was substantial variability in the results from the distance parameter test. For example, the epidemic characteristics were not the same for camp distances 2,3,4,5 and 5,4,3,2. This could be an idiosyncrasy of the computer program, a lack of sensitivity to the parameter in this model, or simply a by-

product of the stochastic nature of the model. Further testing is required to determine the influence of the various camp distances on the epidemic characteristics.

Table 6.8: The Epidemic Characteristics for the Distance from the Camp to the Fort

Camp Distance (days)	2,3,4,5	1,1,1,1	5,5,5,5	5,4,3,2	10,10,10,10	20,30,40,50
Duration (days)	84	80	92	93	52 ↓	51 ↓
Peak Time (days)	↑ 32	26 ↓	31	30	14 ↓	14 ↓
Peak Number	90	↑ 108	84	103 ↓	24 ↓	23 ↓
Total Number	552	574	532	580 ↓	112 ↓	107 ↓
Percent Infected	74%	77%	71%	77%	15% ↓	14% ↓

The change in the epidemic characteristics when the distance from the camp to the fort is varied. Table legend: 2,3,4,5 means the distance from camp 1 to the fort is 2 days, from camp 2 to the fort is 3 days, from camp 3 to the fort is 4 days, and from camp 4 to the fort is 5 days. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

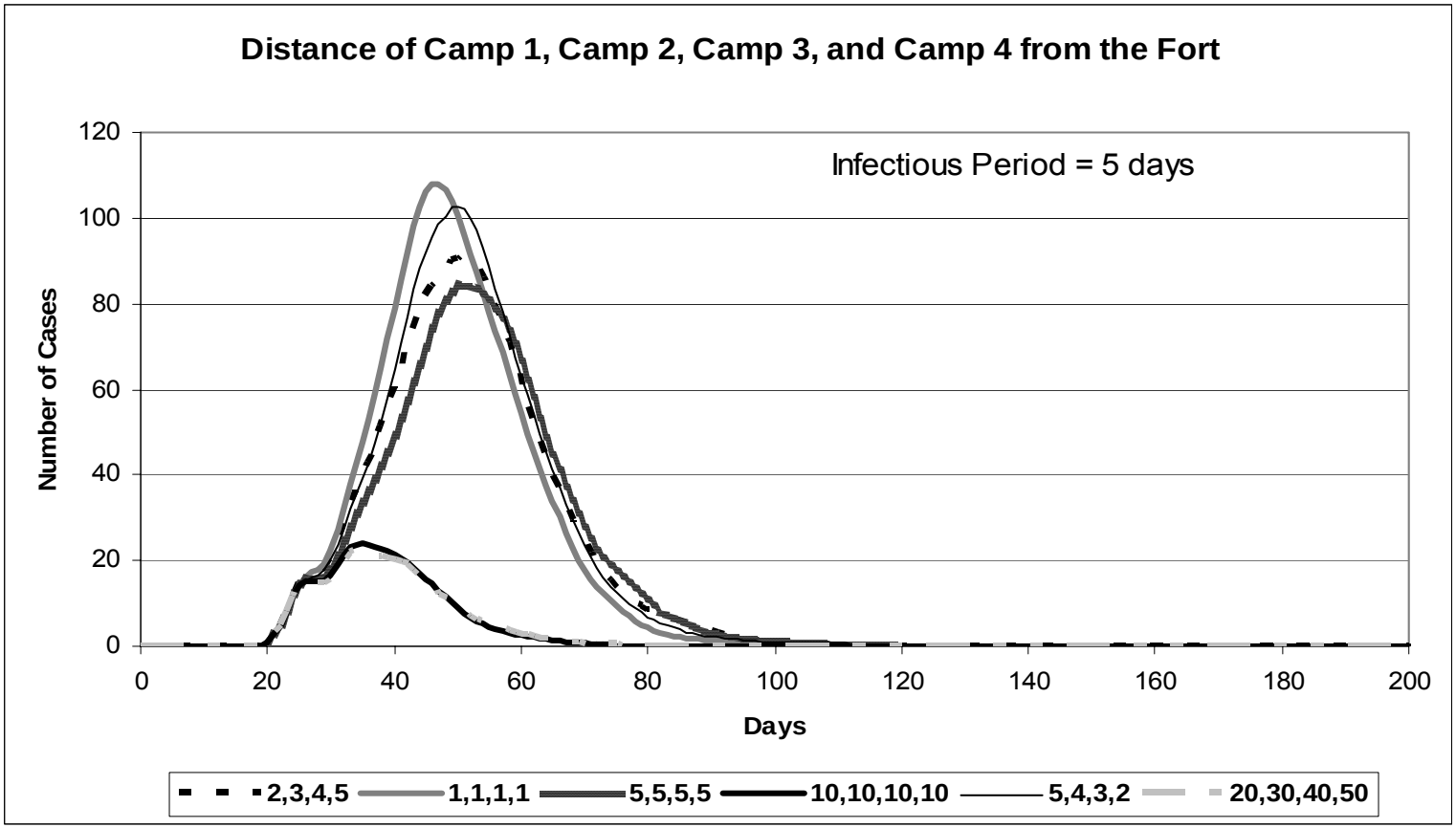

Figure 6.10: The number of cases infected when the distance from each camp to the fort is varied. Only the winter scenario is portrayed.
Graph legend: 2,3,4,5 means the distance from camp 1 to the fort is 2 days, from camp 2 to the fort is 3 days, from camp 3 to the fort is 4 days, and from camp 4 to the fort is 5 days.

- **The Camp Population and The Fort Population**

The camp population was the number of people in each camp. The fort population was the total number of people in the fort at the initiation of the simulation. The number of people in each family remained 15 members throughout this study. This study was performed to determine the impact of increasing the total population on the epidemic characteristics. For the summer scenario, the population number was set for 750 individuals in the fort and 0 in the camps. For the winter scenario, the population number was set for 150 individuals in the fort and 150 individuals in each of the camps resulting in a total population of 750. The results of simulations varying this parameter are shown in Figure 6.11 and Table 6.9. This limited study demonstrated that the epidemic did not spread through the entire community in the winter scenario when the total population was 225 or less. This low population number resulted in a low population density in each area. In turn, the low population density resulted in a low probability and frequency of contact between infectious people and susceptible people, hence, fewer people were infected. The epidemic died out faster for the same reason. When the population was increased to 2250 in the winter scenario, the duration and the peak time were shorter and the peak number, total number, and percent infected were much larger than the winter scenario control range. The duration and peak time were longer than the summer control range. The number of people infected was increased and the epidemic lasted longer because the population density was higher for each area

resulting in the increased probability and frequency of contact between infectious and susceptible individuals. The extended length of the winter epidemic as compared to the summer scenario was probably due to the population's dispersal on the landscape and the men's travel time between the camps and the fort. In addition, this study highlighted a quirk in the computer simulation. When the population in the fort was zero, the initial infection occurred in the nearest camp, which in this case is camp 1. Because the men did not travel to camps other than their own camp, the epidemic remained in camp 1.

The summer scenario was used only for comparison at control values. This scenario was not tested at various population levels although, in retrospect, that comparison should have been made.

Table 6.9: The Epidemic Characteristics for the Population in Each Camp and the Fort

Fort Population	**0**	**15**	**45**	**450**	**150**	**750**
Camp Population	**150**	**15**	**45**	**450**	**150**	**0**
Total Population	**600**	**75**	**225**	**2250**	**750**	**750**
Duration (days)	61 ↓	25 ↓	41 ↓	65 ↓	88	33
Peak Time (days)	14 ↓	5 ↓	6 ↓	26 ↓	30	16
Peak Number	21 ↓	12 ↓	14 ↓	↑ 657	93	366
Total Number	109 ↓	25 ↓	50 ↓	↑ 2234	561	748
Percent Infected	18% ↓	34% ↓	22% ↓	↑ 99%	75%	100%

The changes in the epidemic characteristics when the population in each camp and the fort are varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

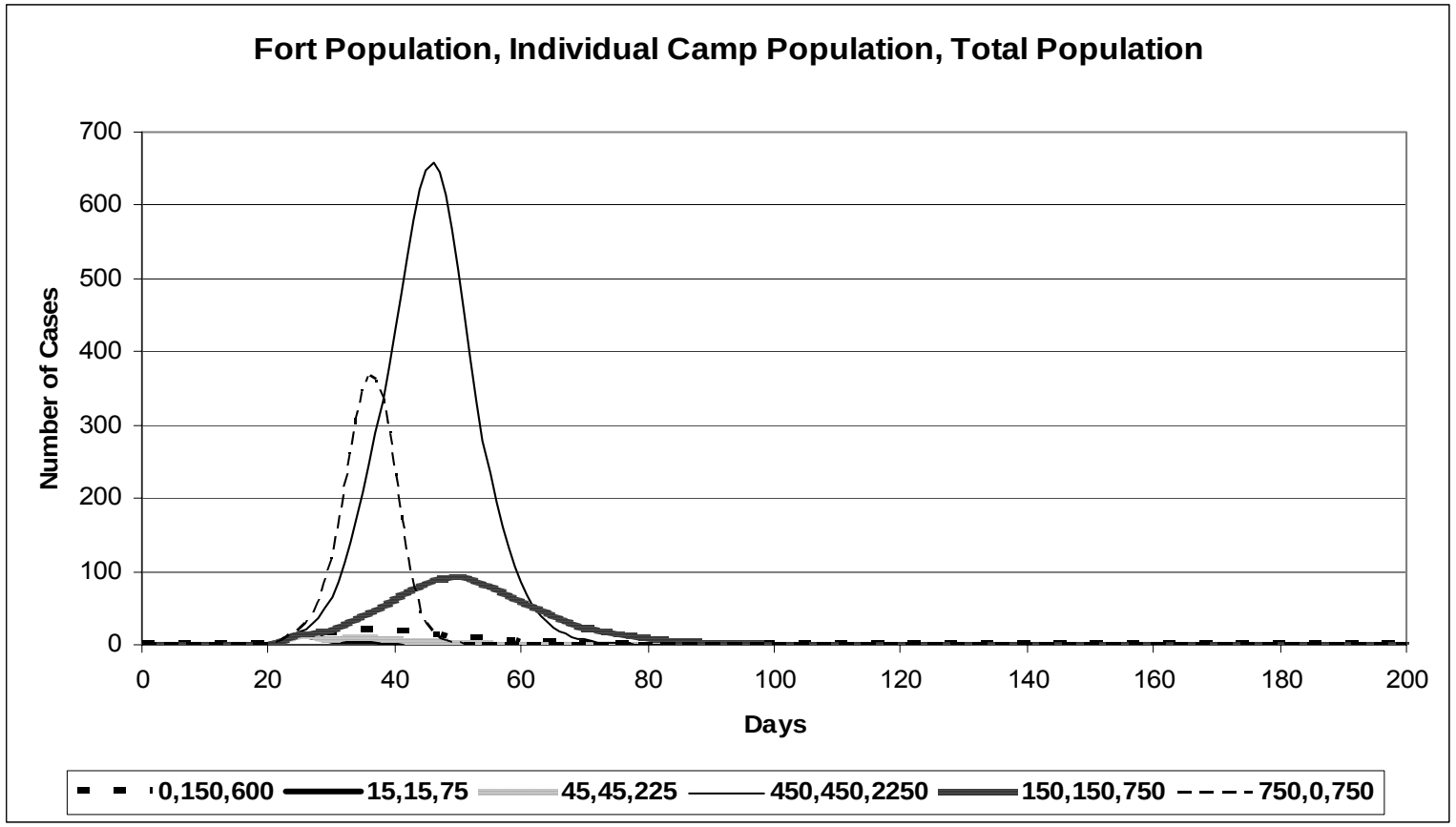

Figure 6.11: The number of infected cases when the population in the fort and camps is varied. Graph legend: 150, 150, 750 refers to 150 people in the fort, 150 people In each camp, and a total population of 750 people

- **The Male, Female, and Child Proportion in the Population**

This parameter was the proportion of the population that falls into each of the three age and sex categories. The Male group included all the males that are 20-50 years old. The Female group included all the females that are 20-50 years old. The Child group included both males and females that are younger than 20 years old or older than 50 years old. For this project, the proportion of the population for the Male and Female group is 0.25 and for the Child group is 0.5 of the total population. This proportion was chosen because it most closely represents the known population proportions in the Norway House community at the time of the 1918-1919 influenza epidemic.

The behavior of the Female and Child groups was assumed to be the same in that they did not travel between the fort and the camps. The Male group from the camps could travel between the fort and the camps. The Male group from the fort did not travel. This parameter was constructed to test the impact of groups with different behavior patterns on the characteristics of the epidemic. This was accomplished by establishing a behavior pattern for one group and varying the proportion of the population that belonged to each of these groups. This parameter was included in the simulation because different age and sex groups in the Norway House community performed different tasks resulting in different behaviors that might have influenced the course of the epidemic.

The results of simulations varying this parameter are shown in Figure 6.12 and Table 6.10. The summer epidemic was not influenced by changes in the population proportion because the entire population was congregated around the

fort and there was no travel between the fort and the camps. In the winter scenario, the only population proportion that influenced the epidemic characteristics was the male proportion of the population. When there were more males traveling, the epidemic was more intense but the duration and peak time were not as predictable. The increased proportion of Males in the population increased the number of people traveling, in turn increasing the number and frequency of contacts between infectious and susceptible people. The result was that more people became infected.

The reason for the observed variability of duration and peak time was not known. Once the proportion of the Male group reached 0.1, the duration and the peak time fluctuated slightly. More data points would be required to demonstrate a pattern in these time characteristics of the epidemic. When there were no individuals in the Male group, no males were traveling and the epidemic remained in the fort. This occurred because only the males traveling could transmit the virus from the fort to the camps.

This test demonstrated that a behavior such as travel had an impact on the course of the epidemic. This parameter will become very important for future projects when specific transmission probabilities are assigned to different age and sex groups and the number of behaviors for each group is increased.

Table 6.10: The Epidemic Characteristics for the Proportion of the Population that are Male, Female, and Child

Summer					
Male Proportion	**0**	**0.1**	**0.25**	**0.5**	**0.5**
Female Proportion	**0.25**	**0.45**	**0.25**	**0.5**	**0**
Child Proportion	**0.75**	**0.45**	**0.5**	**0**	**0.5**
Duration (days)	32	32	33	32	36
Peak Time (days)	16	16	16	17	16
Peak Number	363	367	366	362	358
Total Number	747	749	749	747	749
Percent Infected	100%	100%	100%	100%	100%
Winter					
Male Proportion	**0**	**0.1**	**0.25**	**0.5**	**0.5**
Female Proportion	**0.25**	**0.45**	**0.25**	**0.5**	**0**
Child Proportion	**0.75**	**0.45**	**0.5**	**0**	**0.5**
Duration (days)	51 ↓	84	83	77 ↓	81
Peak Time (days)	14 ↓	31	29	26 ↓	26 ↓
Peak Number	22 ↓	45 ↓	94	↑ 138	↑ 141
Total Number	103 ↓	322 ↓	563	↑ 666	↑ 678
Percent Infected	14% ↓	43% ↓	75%	↑ 89%	↑ 90%

The change in the epidemic characteristics when the proportion of the population that is Male, Female, and Child is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

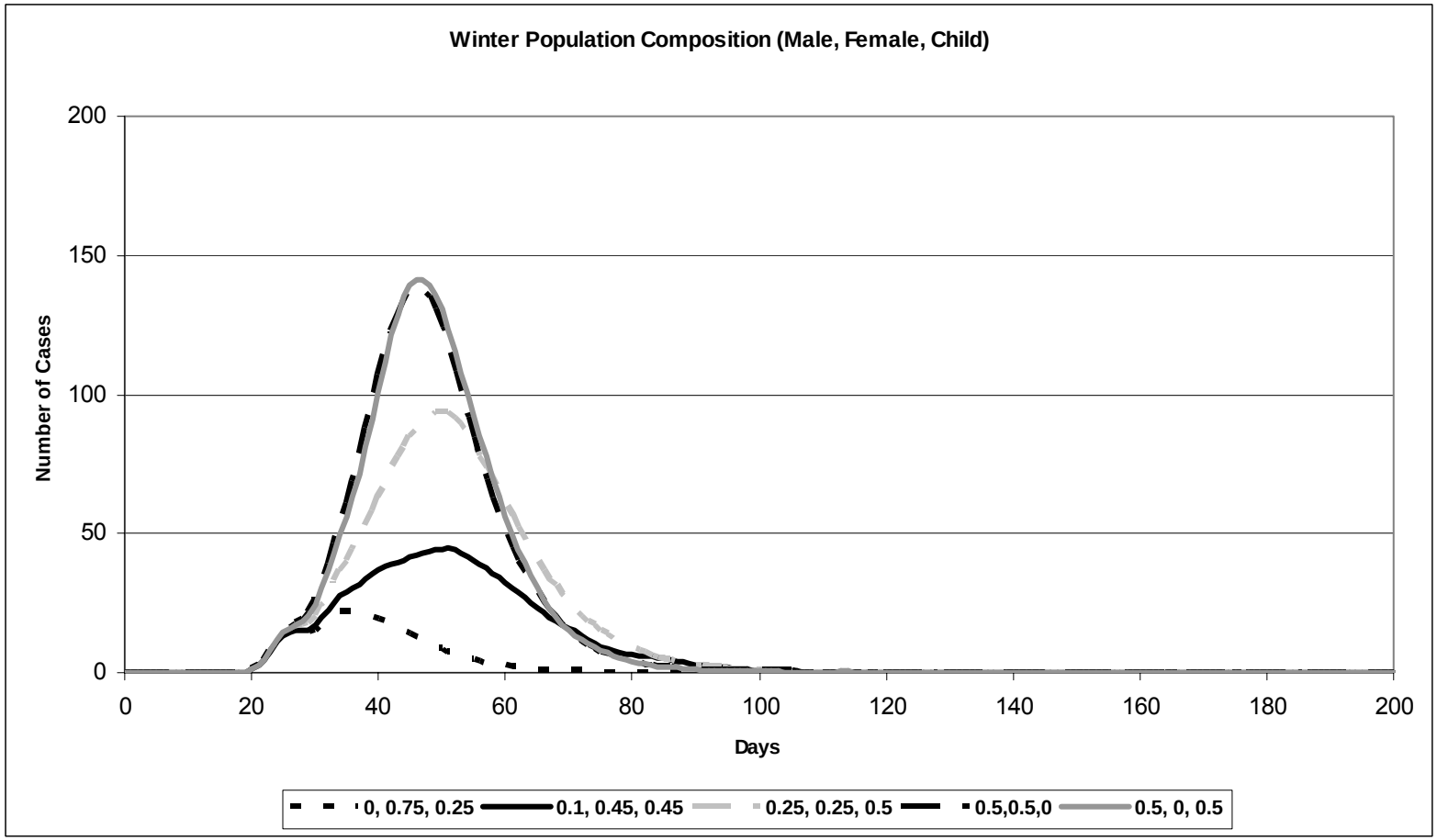

Figure 6.12: The number of cases infected when the proportion of the population for Male, Female, and Child are varied.
Graph legend: 0, 0.75, 0.25 refers to 0 proportion of the population is Male, 0.75 is Female, and 0.25 is Child.

- **The Number of People per Family (The Number of Families per area)**

The number of people per family parameter determined the number of extended families or hunting groups in each area. This parameter was adjusted to the population in each area to obtain the desired family size. The purpose of this parameter test was to determine the influence of family size on the course of the epidemic. In this project, the number of individuals per family was maintained as 15. This family size was chosen because it approximates the number of individuals in the winter hunting groups in the Norway House region. The results of simulations varying this parameter are shown in Figure 6.13, 6.14, and Table 6.11. In the summer scenario, when the family size was increased, the duration and the peak time shortened and the peak number, total number, and percent infected increased. This was very likely due to the increased probability of contact between family members. When the number of family members increased, the contact between the family members occurred more frequently and increased the probability that the virus would be transmitted. The winter scenario responded in the same manner except when the number in each family decreased to 2 or 3 individuals, and then the epidemic remained in the fort. This was probably due to the low probability of contact between non-family members. The males still traveled to the fort but they had a very low probability of meeting an infectious person.

Table 6.11: The Epidemic Characteristics for the Number of Families per Area or the Number of People per Family

Summer				
Families per Area	**1**	**10**	**50**	**100**
People per family	**750**	**75**	**15**	**8**
Duration (days)	8 ↓	16 ↓	33	↑ 53
Peak Time (days)	3 ↓	9 ↓	16	↑ 23
Peak Number	↑ 750	↑ 633	366	215 ↓
Total Number	↑ 750	↑ 750	749	694 ↓
Percent Infected	100%	100%	100%	93% ↓
Winter				
Families per Area	**1**	**10**	**50**	**100**
People per family	**150**	**15**	**3**	**2**
Duration (days)	27 ↓	86	19 ↓	10 ↓
Peak Time (days)	10 ↓	29	2 ↓	3 ↓
Peak Number	↑ 413	84	2 ↓	2 ↓
Total Number	↑ 733	542	6 ↓	3 ↓
Percent Infected	↑ 98%	72%	1% ↓	0% ↓

The change in the epidemic characteristics when the number of families per area (the number of people per family) is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

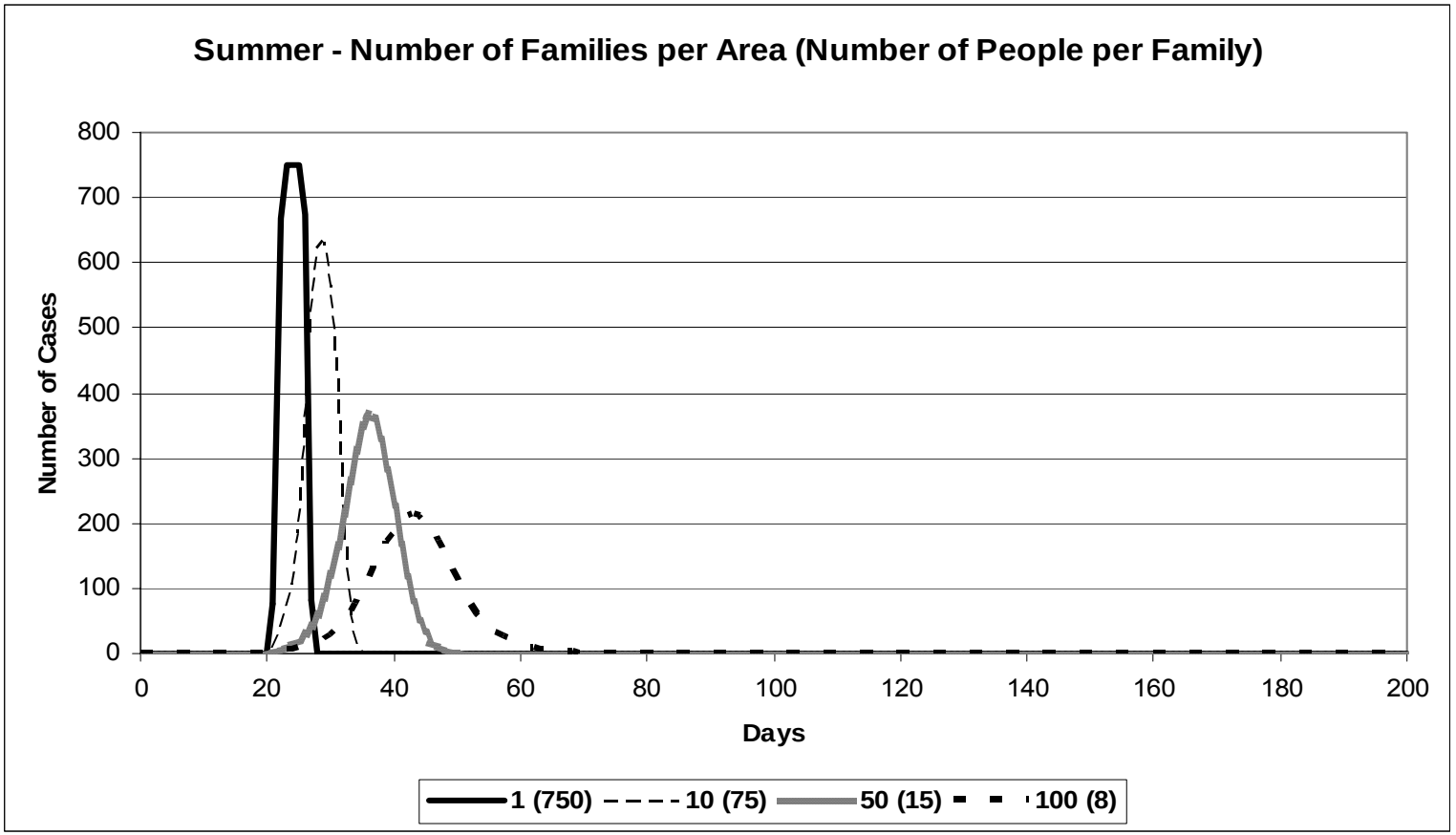

Figure 6.13: The number of cases infected when the number of families per area (the number of people per family) is varied in the summer scenario. Graph legend: 1(750) refers to 1 family in each area and 750 people in each family

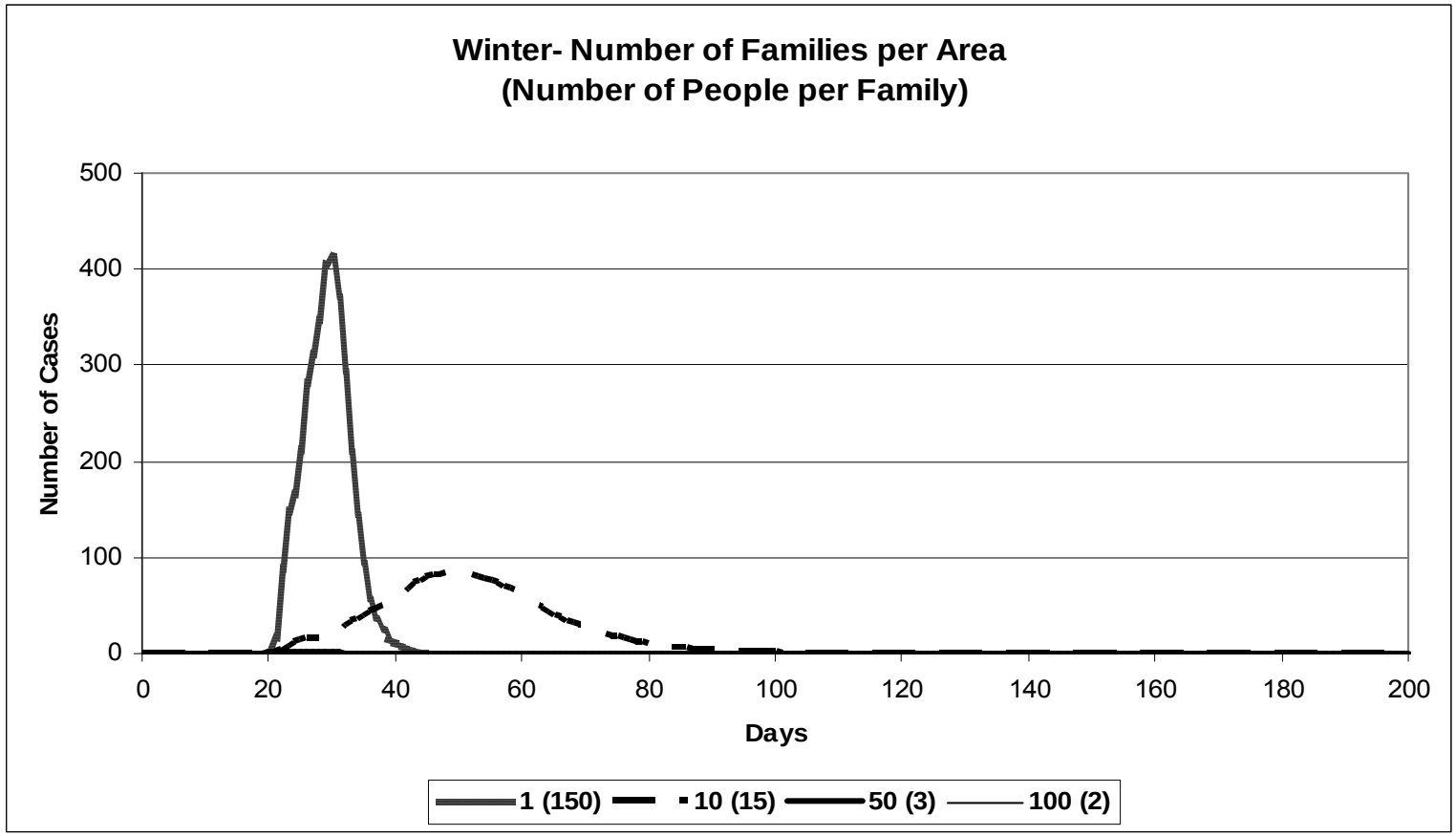

Figure 6.14: The number of cases infected when the number of families per area (the number of people per family) is varied in the winter scenario.
Graph legend: 1(150) refers to 1 family in each area and 150 people in each family

- **The Probability of Contact within Families**

This parameter determined the probability that one family member would be in contact with another family member. This probability was set higher than the probability of contact between non-family members based on the assumption that family members are more likely to be in contact routinely. The extended family group remained important to the people in both winter and summer (Hallowell, 1992). In this project, the probability of contact between family members was maintained at 0.5. The results of simulations varying this parameter are shown in Figure 6.15, 6.16, and Table 6.12. In both, the winter and summer scenario, the peak number, total number, and percent infected increased when the probability increased. As the contact probability increased, the probability of meeting an infected person was increased, hence, more people become infected. Duration and peak time lengthened to a maximum at the probability of 0.25 in both the winter and summer scenario. The duration and peak time probably shortened after the maximum because so many people were becoming infected so rapidly that the epidemic burned itself out early resulting in a shorter epidemic

Table 6.12: The Epidemic Characteristics for the Probability of Contact between Family Members

Summer					
Contact within Families	**0.00**	**0.25**	**0.50**	**0.75**	**1.00**
Duration (days)	14 ↓	↑ 44	35	29 ↓	27 ↓
Peak Time (days)	4 ↓	↑ 20	16	15 ↓	14 ↓
Peak Number	2 ↓	273 ↓	360	↑ 400	↑ 433
Total Number	4 ↓	719 ↓	748	↑ 748	↑ 750
Percent Infected	1% ↓	96% ↓	100%	100%	100%
Winter					
Contact within Families	**0**	**0.25**	**0.50**	**0.75**	**1.00**
Duration (days)	5 ↓	↑ 113	80	79 ↓	69 ↓
Peak Time (days)	1 ↓	↑ 38	30	25 ↓	24 ↓
Peak Number	1 ↓	44 ↓	93	↑ 110	↑ 125
Total Number	1 ↓	396 ↓	574	↑ 580	↑ 600
Percent Infected	0% ↓	53% ↓	77%	77%	↑ 80%

The change in the epidemic characteristics when the probability of the contact between family members is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

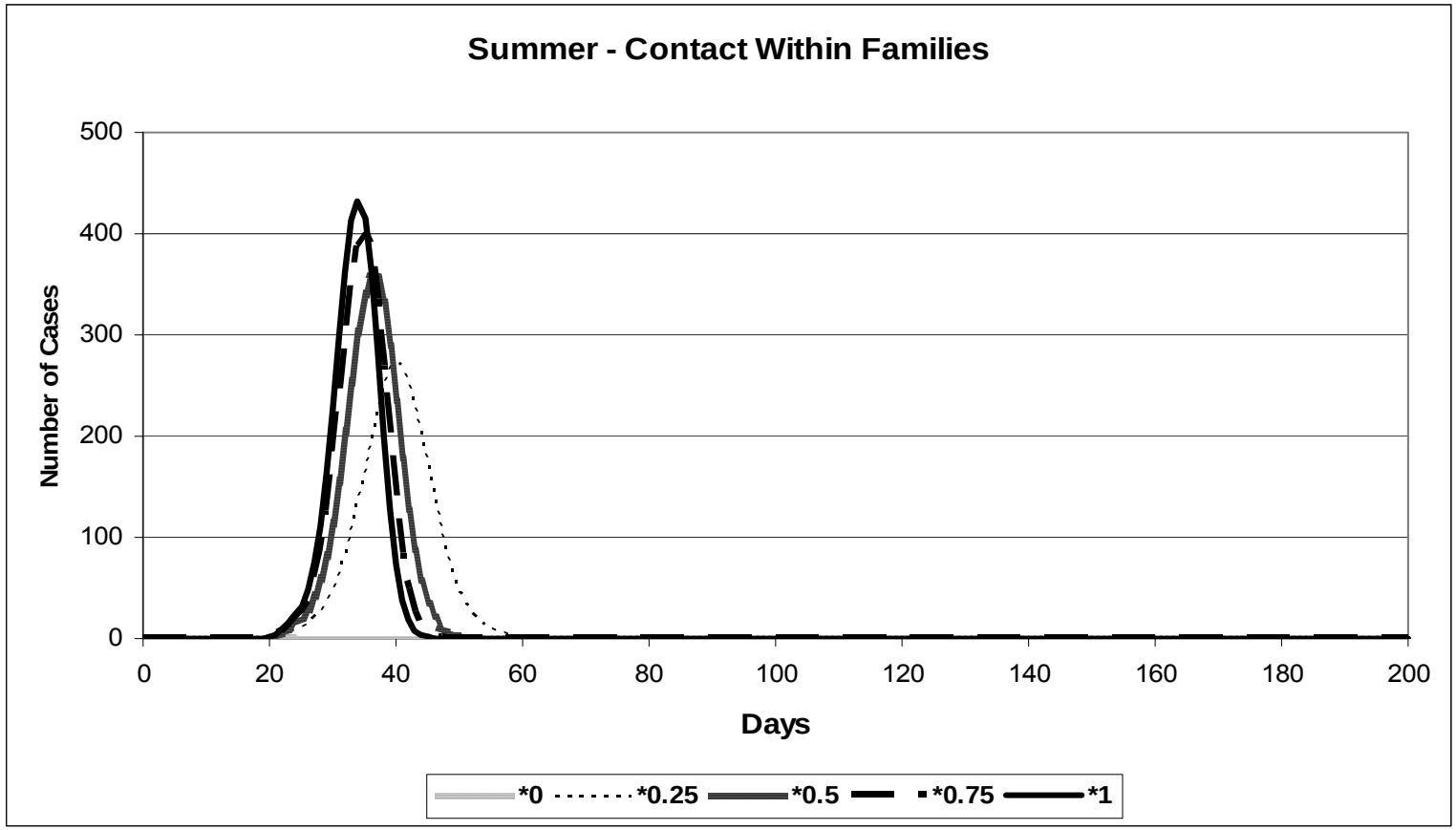

Figure 6.15: The number of cases infected when the probability of contact between family members is varied in the summer scenario.

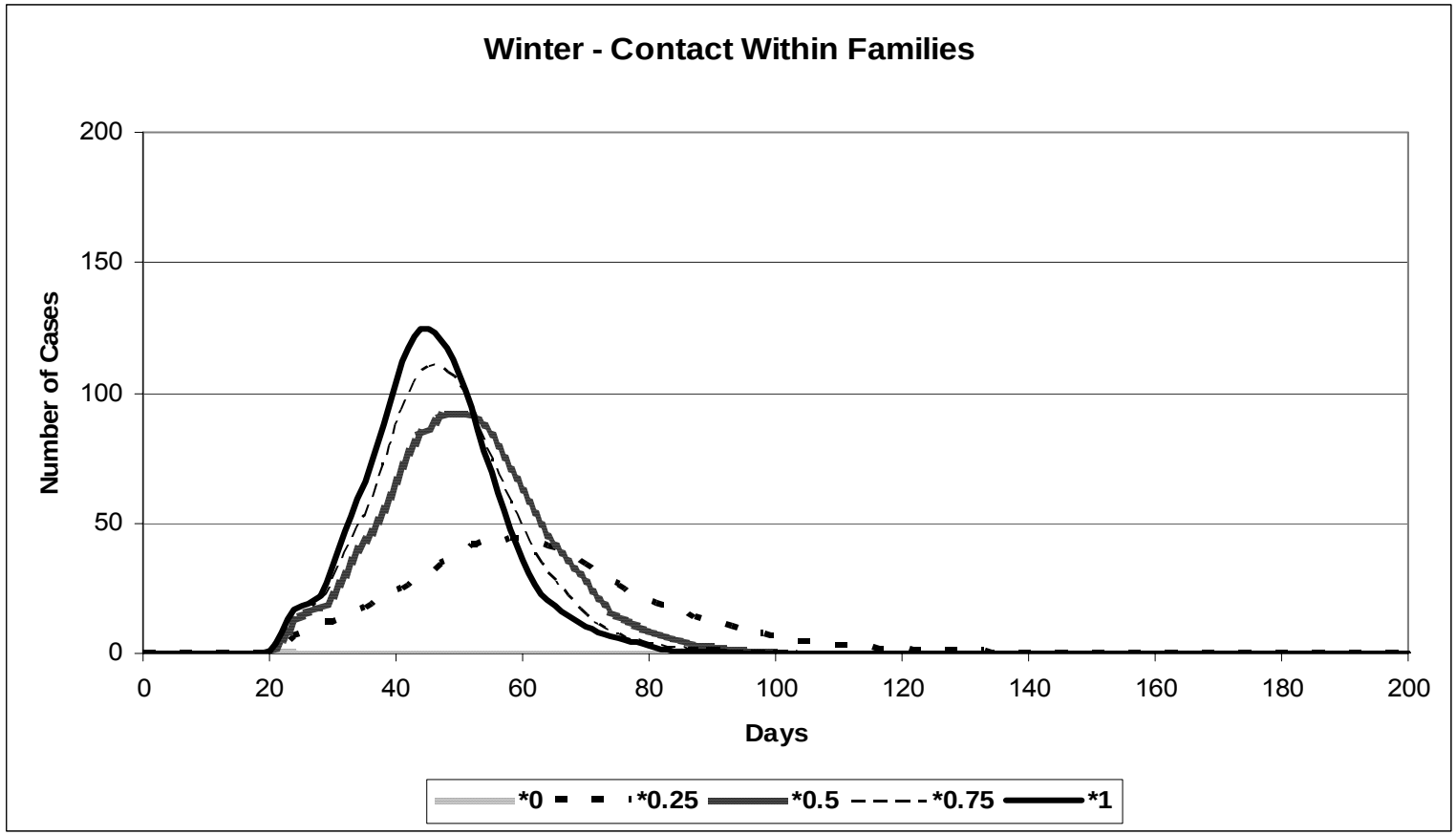

Figure 6.16: The number of cases infected when the probability of contact between family members is varied in the winter scenario.

- **The Probability of Contact between Non-Family Members**

This parameter determined the probability that an individual from one family would come into contact with an individual from another family. This probability was lower than the former parameter because of the assumption that non-family members were less likely to be in contact routinely. In this project, the probability of contact between non-family members was maintained at 0.001. The results of simulations varying this parameter are shown in Figure 6.17, 6.18, and Table 6.13. The pattern of change for the epidemic characteristics was the same as seen with the contact probability between family members. The number infected increased as the probability increased. The duration and peak time lengthened to a maximum at a probability of 0.001 in the winter and summer scenario. After that point, the epidemic burned itself out early resulting in a shorter epidemic. This phenomenon was similar to the epidemic burn out seen in the previous parameter.

Table 6.13: The Epidemic Characteristics for the Probability of the Contact between Non-family Members

Summer					
Contact Between Families	**0**	**0.0001**	**0.001**	**0.01**	**0.1**
Duration (days)	12 ↓	12 ↓	33	16 ↓	10 ↓
Peak Time (days)	5 ↓	5 ↓	17	9 ↓	5 ↓
Peak Number	13 ↓	13 ↓	363	↑ 667	↑ 749
Total Number	16 ↓	16 ↓	749	↑ 750	↑ 750
Percent Infected	2% ↓	2% ↓	100%	100%	100%
Winter					
Contact Between Families	**0**	**0.0001**	**0.001**	**0.01**	**0.1**
Duration (days)	24 ↓	23 ↓	88	45 ↓	33 ↓
Peak Time (days)	5 ↓	5 ↓	29	19 ↓	12 ↓
Peak Number	13 ↓	13 ↓	93	↑ 242	↑ 377
Total Number	27 ↓	26 ↓	560	↑ 699	↑ 733
Percent Infected	4% ↓	3% ↓	75%	↑ 93%	↑ 98%

The change in the epidemic characteristics when the probability of the contact between non-family members is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

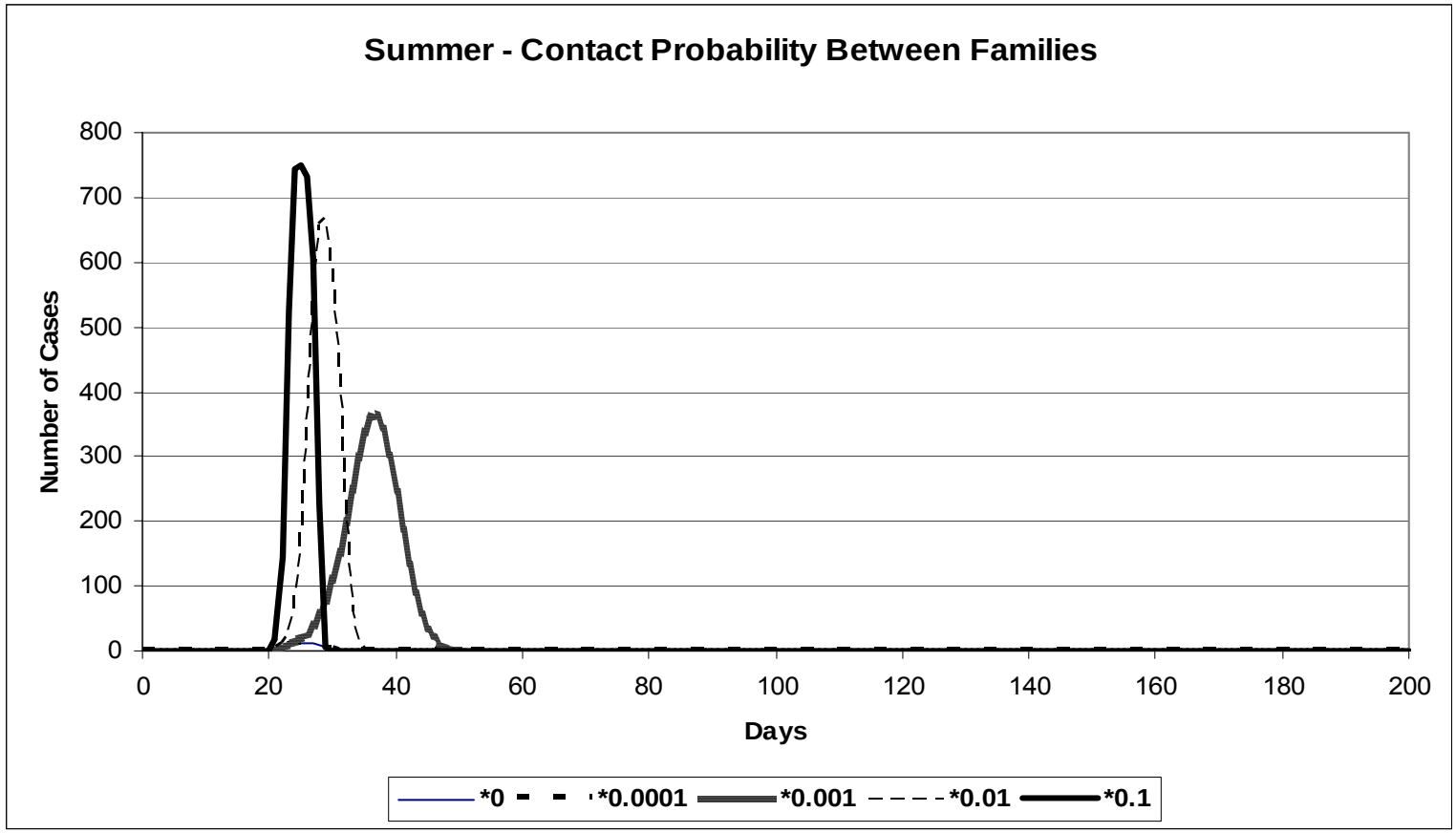

Figure 6.17: The number of cases infected when the probability of contact between non-family members is varied in the summer scenario

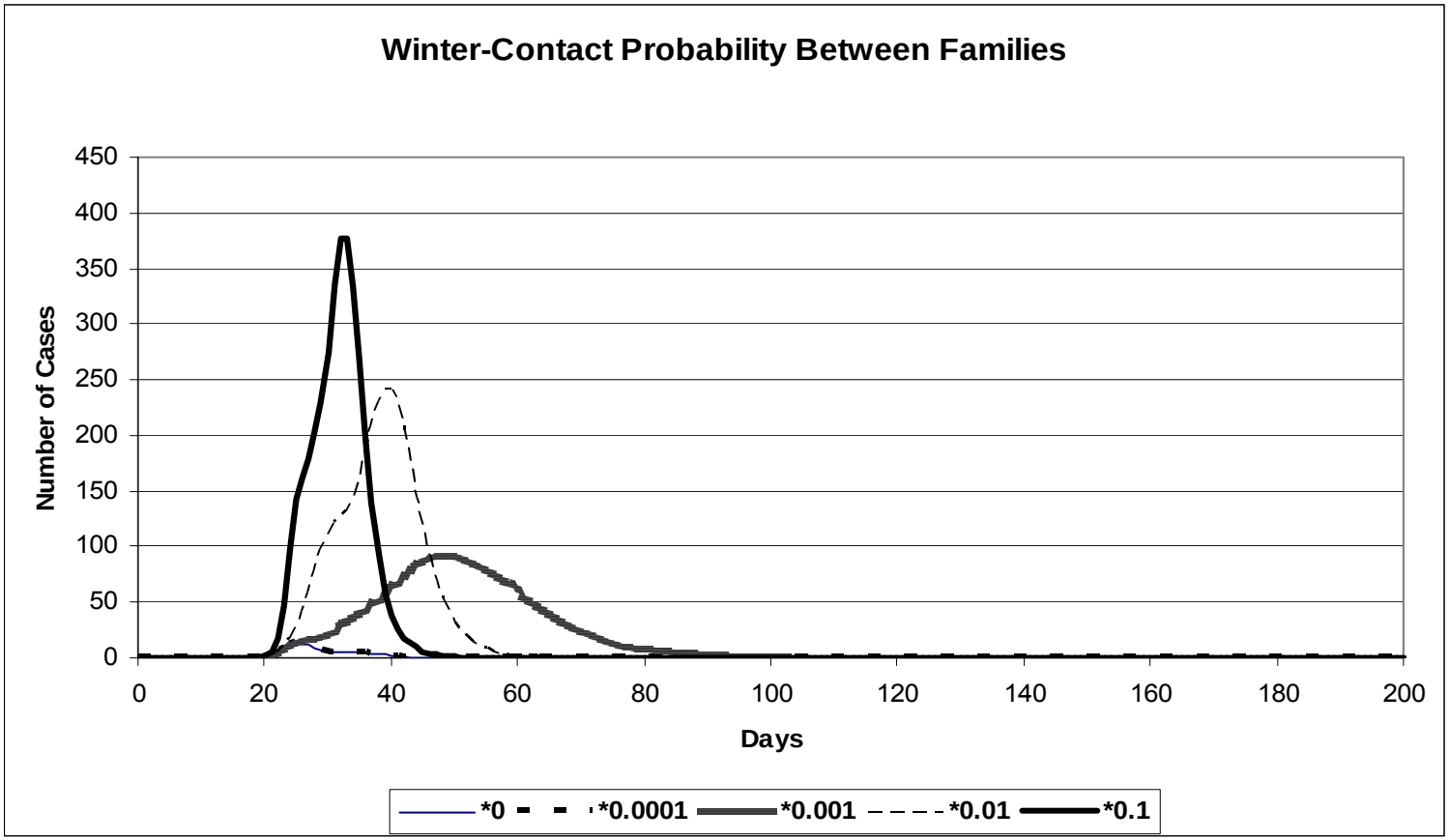

Figure 6.18: The number of cases infected when the probability of contact between non-family members is varied in the winter scenario.

- **The Infectious Period**

The infectious period is the period of time, in days, that an infected person can transmit the influenza virus to susceptible people. The infectious period was maintained at 5 days for this project. The results of simulations varying this parameter are shown in Figure 6.19, 6.20, and Table 6.14. In the summer scenario, the intensity of the epidemic increased as the infectious period increased, until 100% of the population was infected. The duration and peak time reached a maximum at an infectious period of two days. In the winter scenario, the same pattern was seen with the exception that the duration increased to a maximum at an infectious period of six days. This phenomenon was similar to the epidemic burn out seen with both contact times. The intensity of the epidemic increased with increasing infectious periods. An increased infectious period provided more time for a susceptible person to become exposed to an infectious person. This point is further emphasized in Figure 6.21. In this figure, the difference between the winter and summer scenarios was typical but the number of people infected increased in both scenarios as the infectious period was increased. Figure 6.22 is a comparison of summer and winter scenarios when the duration of epidemic was plotted against the infectious period. The summer epidemic was very intense so the maximum duration occurred at a very short infectious period. After this point, so many people became infected so rapidly that the epidemic burned itself out and the epidemic ended earlier. The same phenomenon occurred in the winter scenario but the

maximum occurred at a higher infectious period. The epidemic burn out phenomenon was seen in both contact time and infectious period.

Table 6.14: The Epidemic Characteristics for the Infectious Period

Summer										
Infectious Period (Days)	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Duration (days)	41	↑ 44	36	35	33	33	33	33	35	35
Peak Time (days)	1 ↓	↑ 18	17	16	16	17	17	17	18	18
Peak Number	2 ↓	76 ↓	186 ↓	283 ↓	366	↑ 433	↑ 498	↑ 547	↑ 591	↑ 628
Total Number	64 ↓	610 ↓	721 ↓	739 ↓	749	747	↑ 750	↑ 750	↑ 750	↑ 750
Percent Infected	9% ↓	81% ↓	96% ↓	99%	100%	100%	100%	100%	100%	100%
Winter										
Infectious Period (Days)	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Duration (days)	8 ↓	35 ↓	66 ↓	79 ↓	83	90	89	88	84	84
Peak Time (days)	1 ↓	4 ↓	18 ↓	29	29	30	30	31	31	31
Peak Number	1 ↓	6 ↓	14 ↓	46 ↓	94	↑133	175	209	251	290
Total Number	10 ↓	47 ↓	168 ↓	395 ↓	563	↑ 642	678	693	718	731
Percent Infected	1% ↓	6% ↓	22% ↓	53% ↓	75%	↑ 86%	↑ 90%	↑ 92%	↑ 96%	↑ 97%

The change in the epidemic characteristics when the Infectious period is varied. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

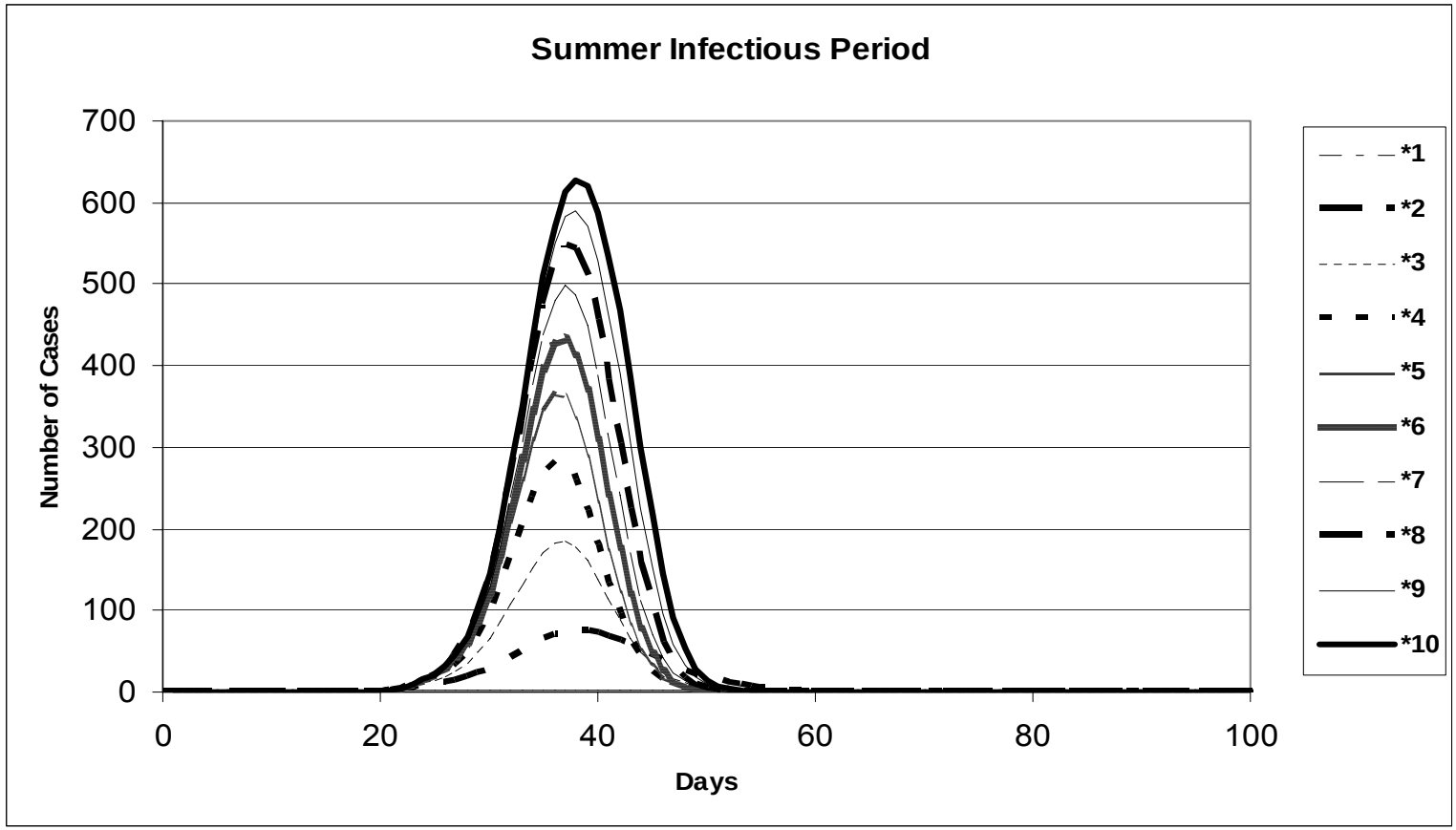

Figure 6.19: The number of cases infected when the infectious period in days is varied in the summer scenario.

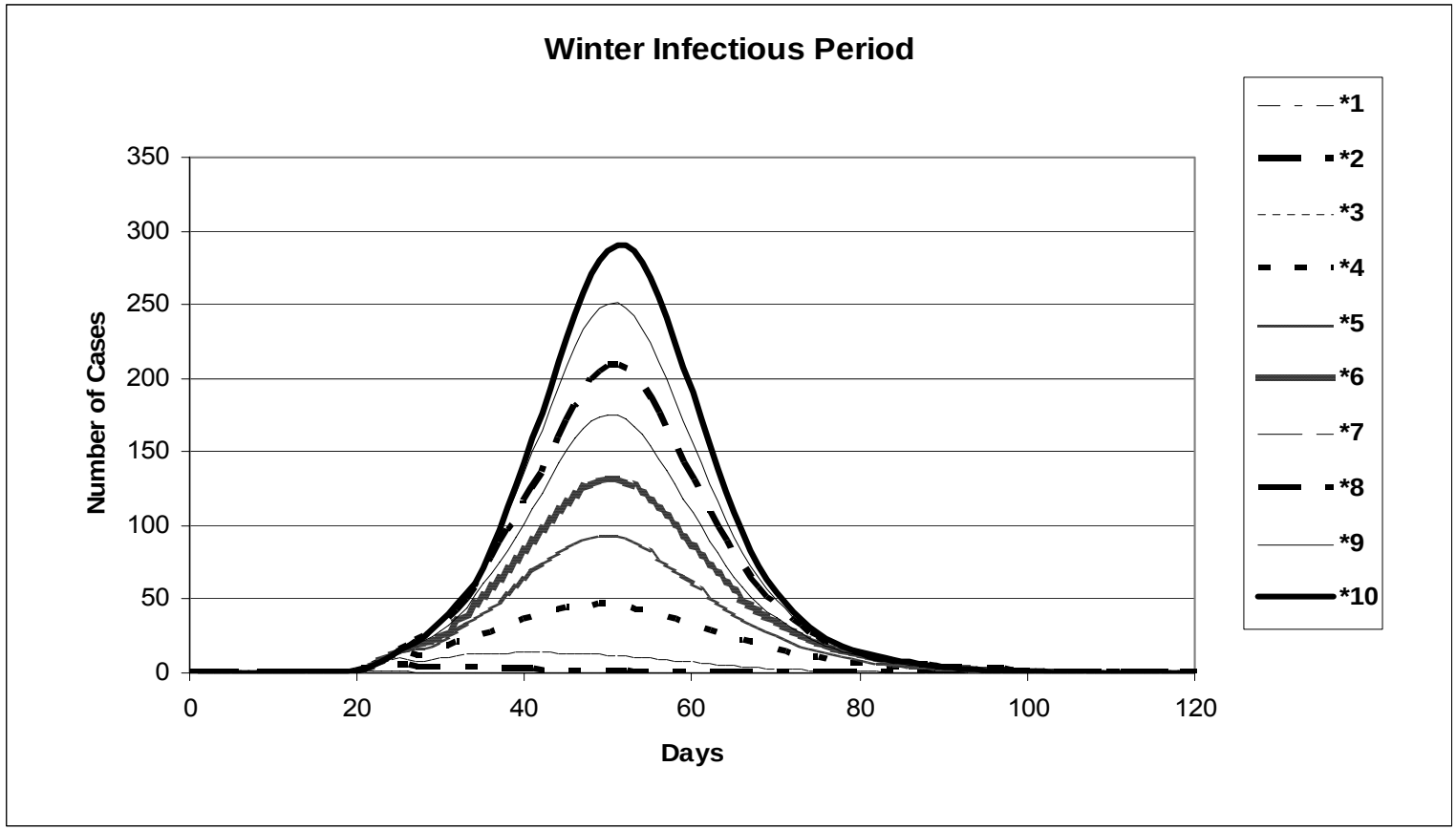

Figure 6.20: The number of cases infected when the infectious period in days is varied in the winter scenario.

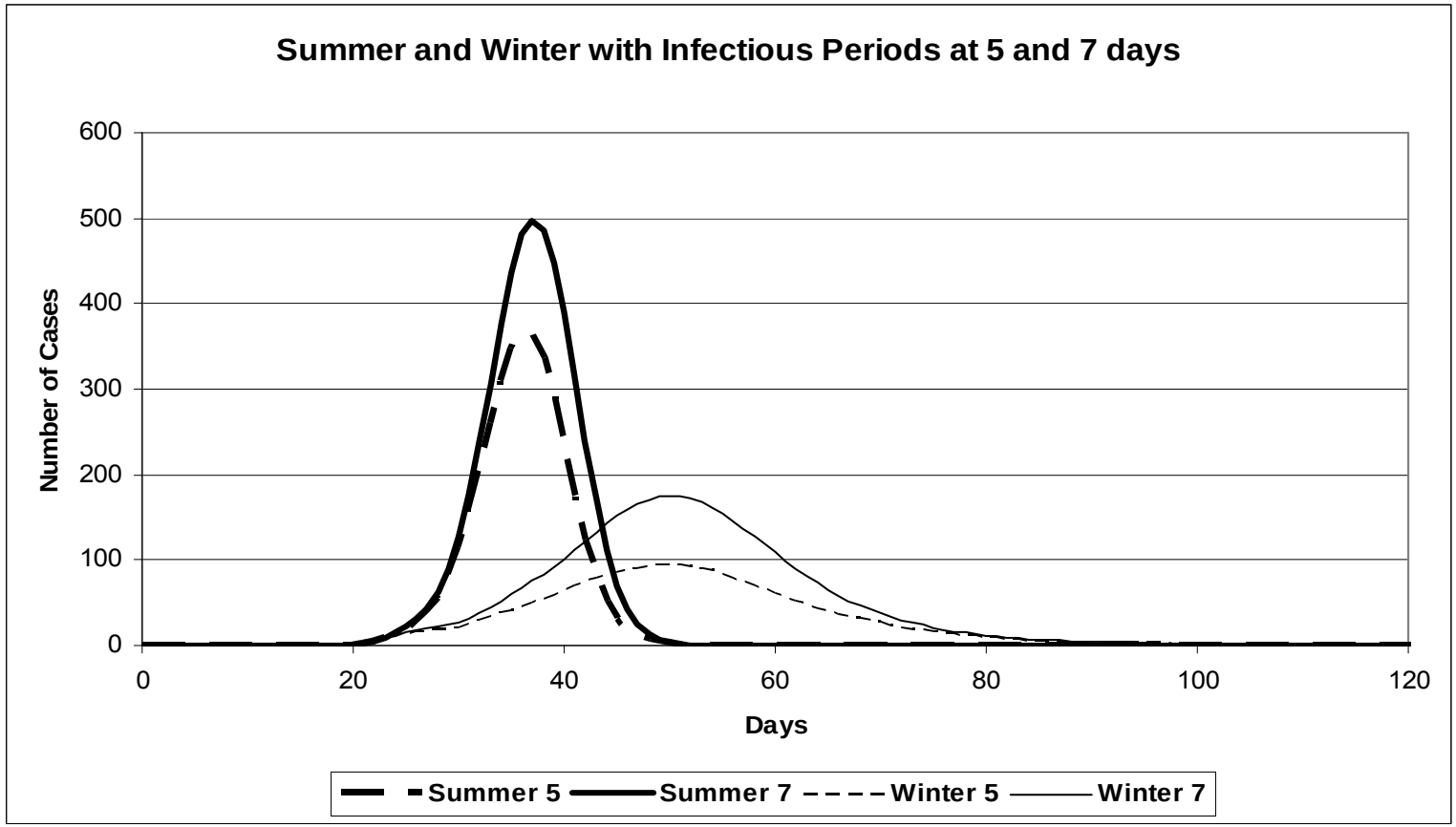

Figure 6.21: The number of cases infected when the infectious period in days is varied in the summer and winter scenario.

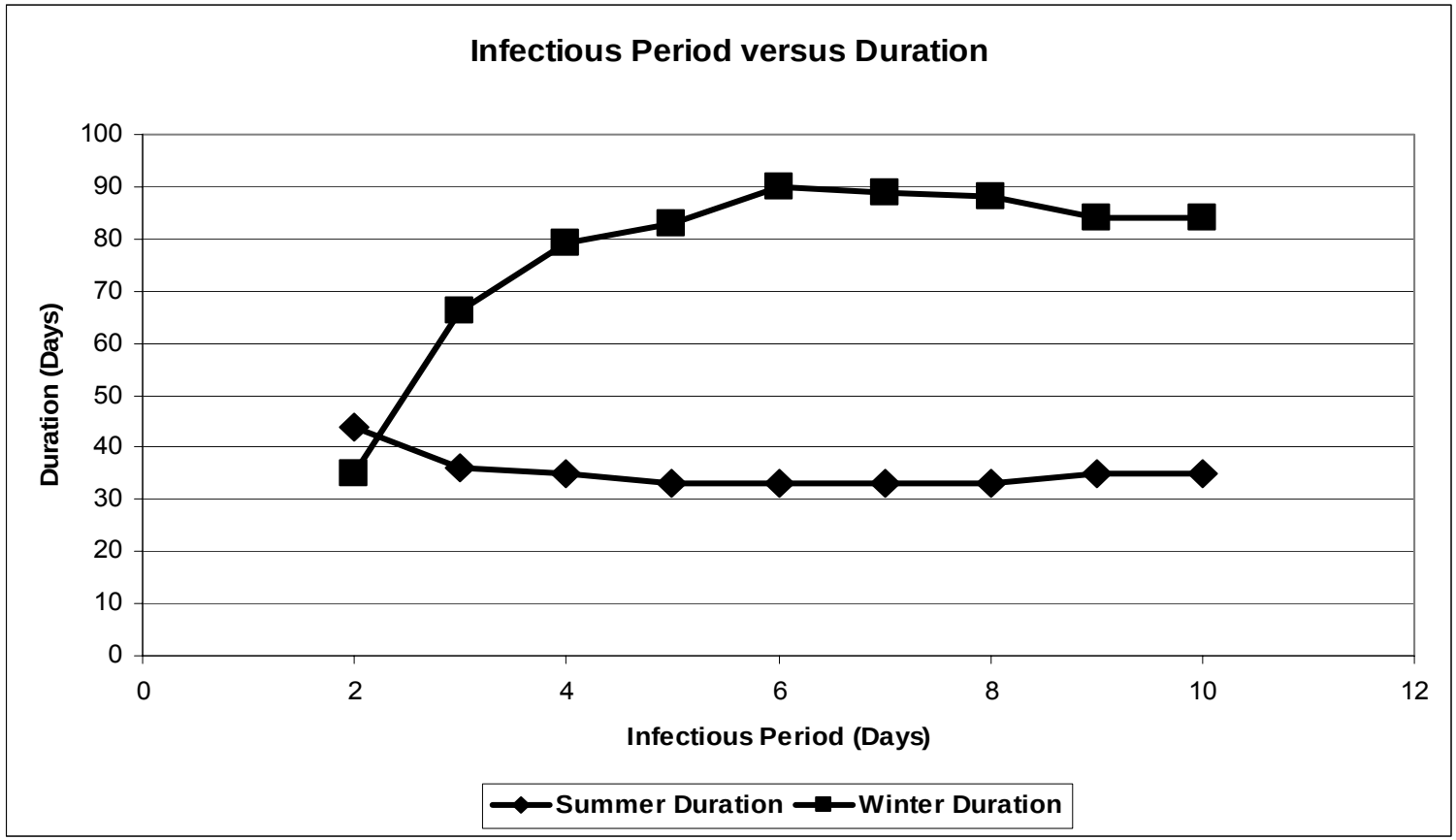

Figure 6.22: The duration of the epidemic in days when the infectious period in days is varied in the summer and winter scenario.

- **The Probability of Transmitting the Virus**

This parameter was the probability that a person would be infected with the disease if they were exposed to an infected person. The computer simulation was constructed with the potential to vary this parameter differently for different groups of people. This will be a very useful parameter in future projects especially when mortality parameters are added to the model. In this project, the entire population was assigned the same probability of transmission. The transmission probability was maintained at 0.2 for this project. The results of simulations varying this parameter are shown in Figure 6.23, 6.24, and Table 6.15. In the summer scenario, the intensity of the epidemic increased as the probability increased. At a probability of 0.2, 100% of the population was infected. Duration and peak time reached a maximum at a probability of 0.1 and by a probability of 0.3, the duration decreased below the control range. The winter scenario followed the same pattern. As the probability of transmission increased, the intensity of the epidemic increased by increasing the peak number, total number, and percent infected. The duration and peak time decreased after the maximum at a probability of 0.1. The change in intensity was due to an increased likelihood that a susceptible person would contract the disease from an infected person. The change in duration and peak time probably occurred because the epidemic burned itself out, resulting in a shorter epidemic. Future research will include testing of the probabilities of transmission between 0 and 0.1 to see where the true maximum value lies for duration and peak time.

Table 6.15: The Epidemic Characteristic for the Probability of Transmitting the Virus

Summer										
Transmission	**0.1**	**0.2**	**0.3**	**0.4**	**0.5**	**0.6**	**0.7**	**0.8**	**0.9**	**1**
Duration (days)	↑ 70	33	24 ↓	21 ↓	19 ↓	18 ↓	17 ↓	16 ↓	15 ↓	15 ↓
Peak Time (days)	↑ 26	16	13 ↓	11 ↓	10 ↓	10 ↓	9 ↓	9 ↓	8 ↓	8 ↓
Peak Number	166 ↓	366	↑ 476	↑ 537	↑ 592	↑ 616	↑ 651	↑ 656	↑ 682	↑ 691
Total Number	692 ↓	749	↑ 750	↑ 750	↑ 750	↑ 750	↑ 750	↑ 750	↑ 750	↑ 750
Percent Infected	92% ↓	100%	100%	100%	100%	100%	100%	100%	100%	100%
Winter										
Transmission	**0.1**	**0.2**	**0.3**	**0.4**	**0.5**	**0.6**	**0.7**	**0.8**	**0.9**	**1**
Duration (days)	↑ 101	83	65 ↓	57 ↓	52 ↓	49 ↓	41 ↓	43 ↓	40 ↓	38 ↓
Peak Time (days)	↑ 36	29	24 ↓	22 ↓	20 ↓	19 ↓	18 ↓	17 ↓	16 ↓	16 ↓
Peak Number	17 ↓	94	↑ 154	↑ 187	↑ 214	↑ 232	↑ 256	↑ 264	↑ 280	↑ 288
Total Number	173 ↓	563	↑ 668	↑ 693	↑ 710	↑ 703	↑ 714	↑ 713	↑ 720	↑ 719
Percent Infected	23% ↓	75%	↑ 89%	↑ 92%	↑ 95%	↑ 94%	↑ 95%	↑ 95%	↑ 96%	↑ 96%

The changes in the epidemic characteristics as the probability of transmitting the virus changes. The (↑) symbol indicates that the value is above the control range for that characteristic. The (↓) symbol indicates that the value is below the control range for that characteristic. The control ranges are available in Table 6.2 and Table 6.3.

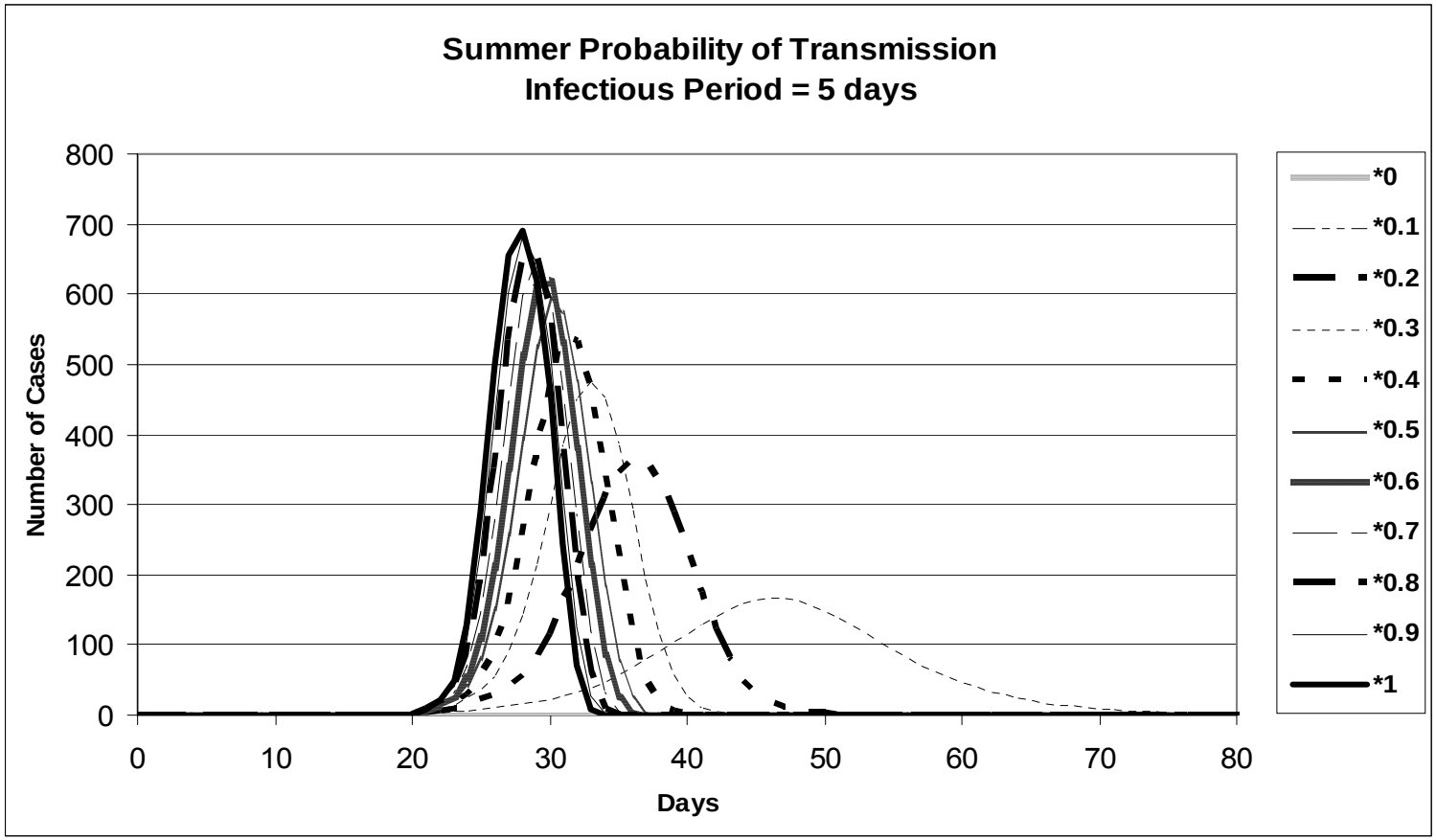

Figure 6.23: The number of cases infected when the probability of transmission is varied in the summer scenario.

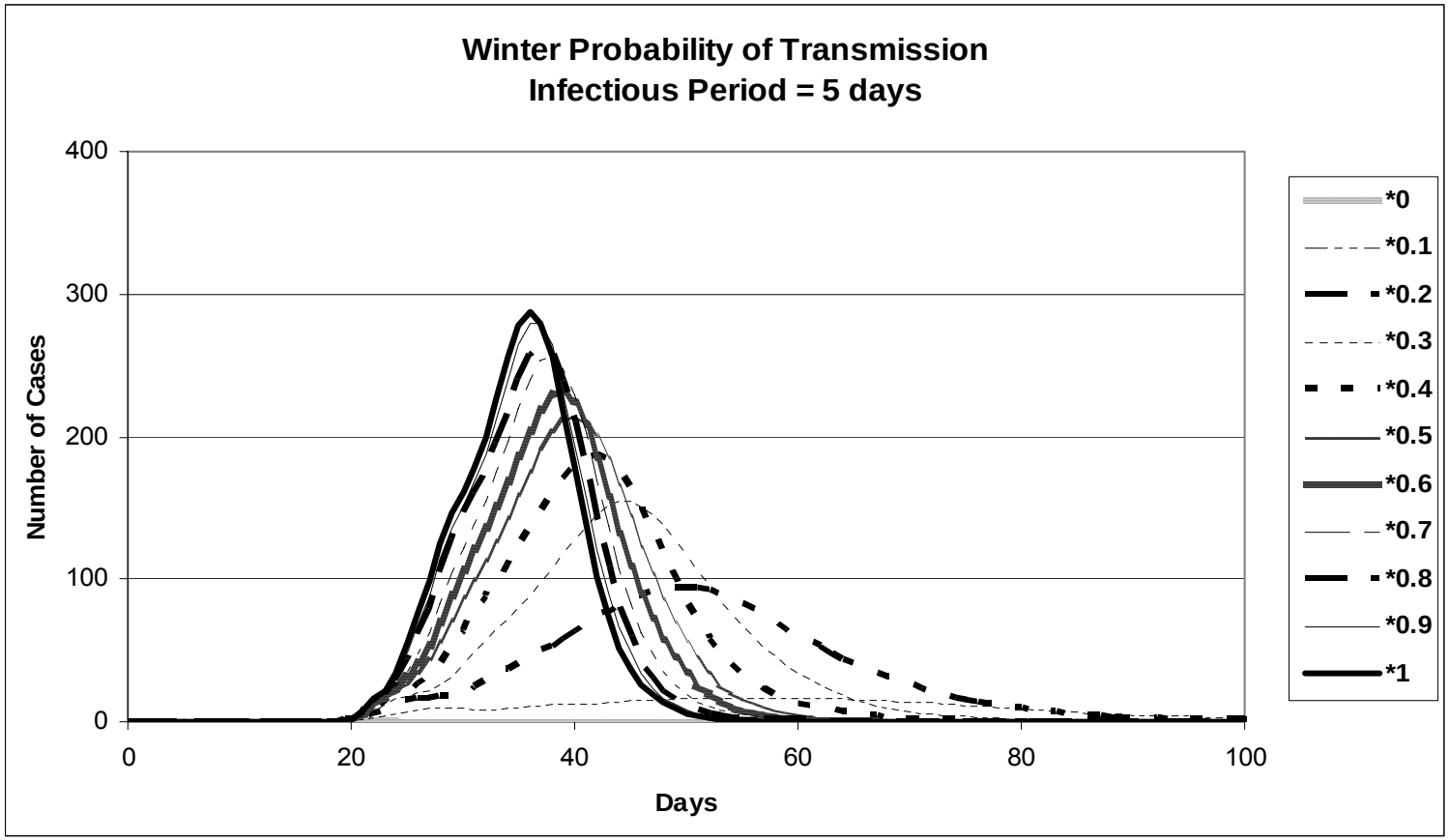

Figure 6.24: The number of cases infected when the probability of transmission is varied in the winter scenario.

- **The Probability of Transmission at an Infectious Period of 5 and 7 days**

A final study was performed to test the impact of changes in both the transmission and infectious period. The infectious period can last 3-5 days in adults and up to 7 days in children (Chin and Ascher, 2000). In this study a range of probabilities for transmission was tested at an infectious period of 5 days and 7 days. The results of simulations varying this parameter are shown in Figure 6.25, 6.26, 6.27 and 6.28. As the probability of transmission increased, the duration and peak time increased to a maximum at the probability of transmission of 0.1 in both the winter and summer epidemics. The infectious period had little impact in the summer epidemic in relationship to the transmission probability. In the winter epidemic, a longer infectious period had a longer duration and peak time only at the maximum at the probability of transmission of 0.1. Otherwise, there was little impact from infectious period. A longer infectious period increased the peak number of cases in the winter and summer epidemics as the transmission probability increased. The total number of cases reached a maximum too quickly to draw many conclusions but the pattern would probably be the same as the peak number. The basic differences between summer and winter scenarios were unaffected. A summer epidemic was short and intense and a winter epidemic was longer and less intense.

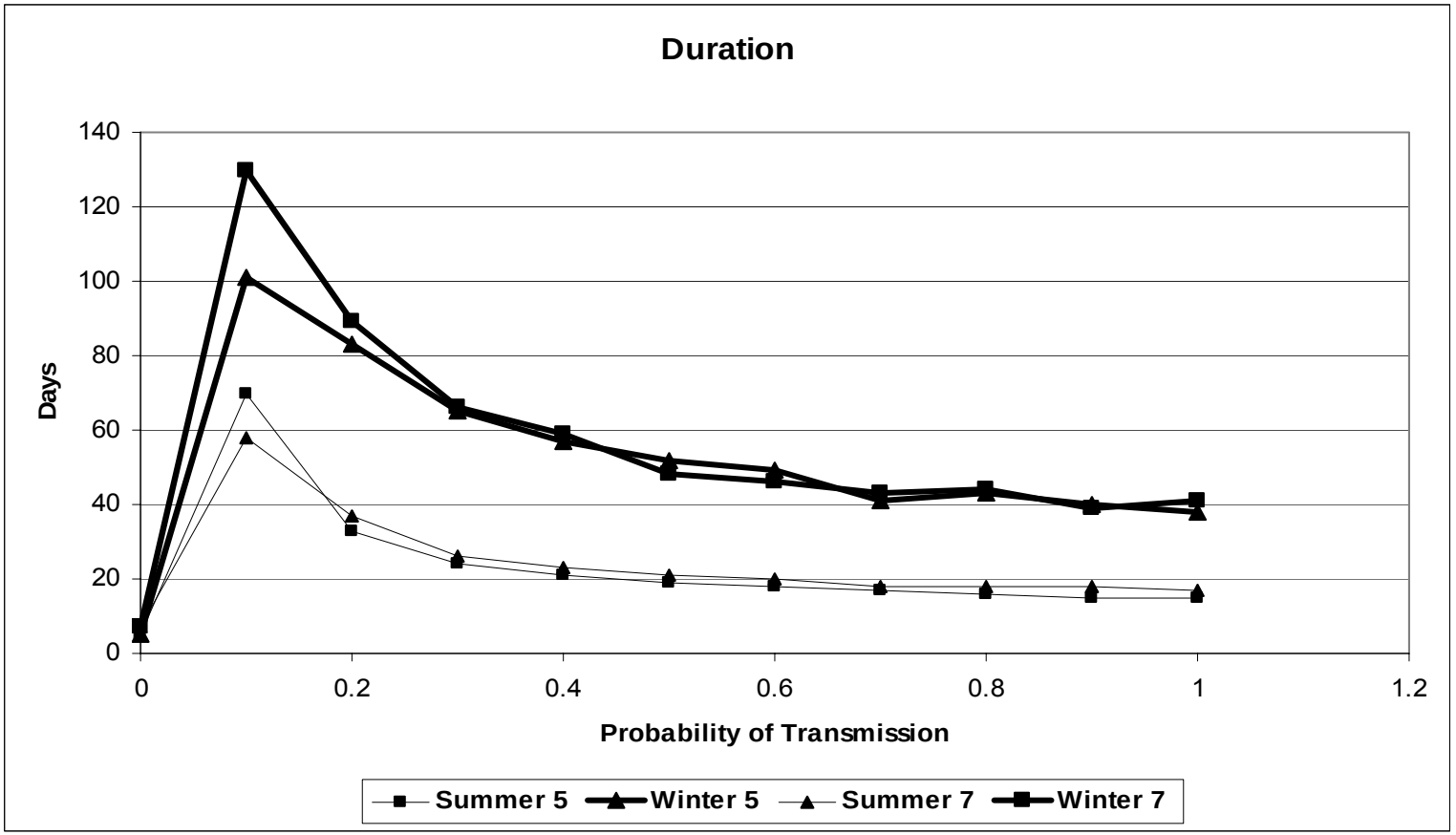

Figure 6.25: The duration of the epidemic in days when the probability of transmission is varied at an infectious period of 5 and 7 days in the summer and winter scenario. Graph legend: Summer 5 refers to an infectious period of 5 days in the summer scenario.

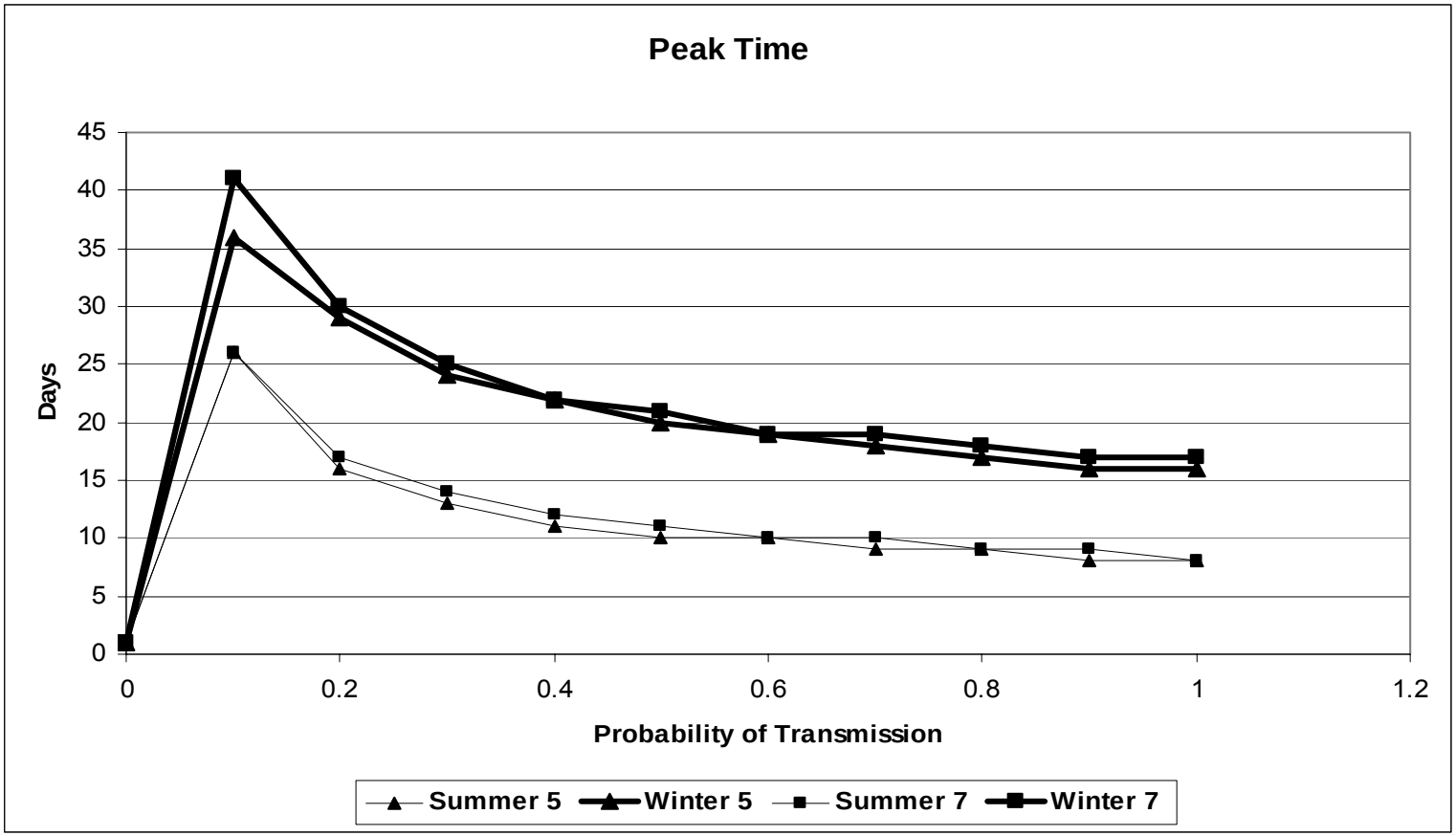

Figure 6.26: The peak time of the epidemic in days when the probability of transmission is varied at an infectious period of 5 and 7 days in the summer and winter scenario. Graph legend: Summer 5 refers to an infectious period of 5 days in the summer scenario.

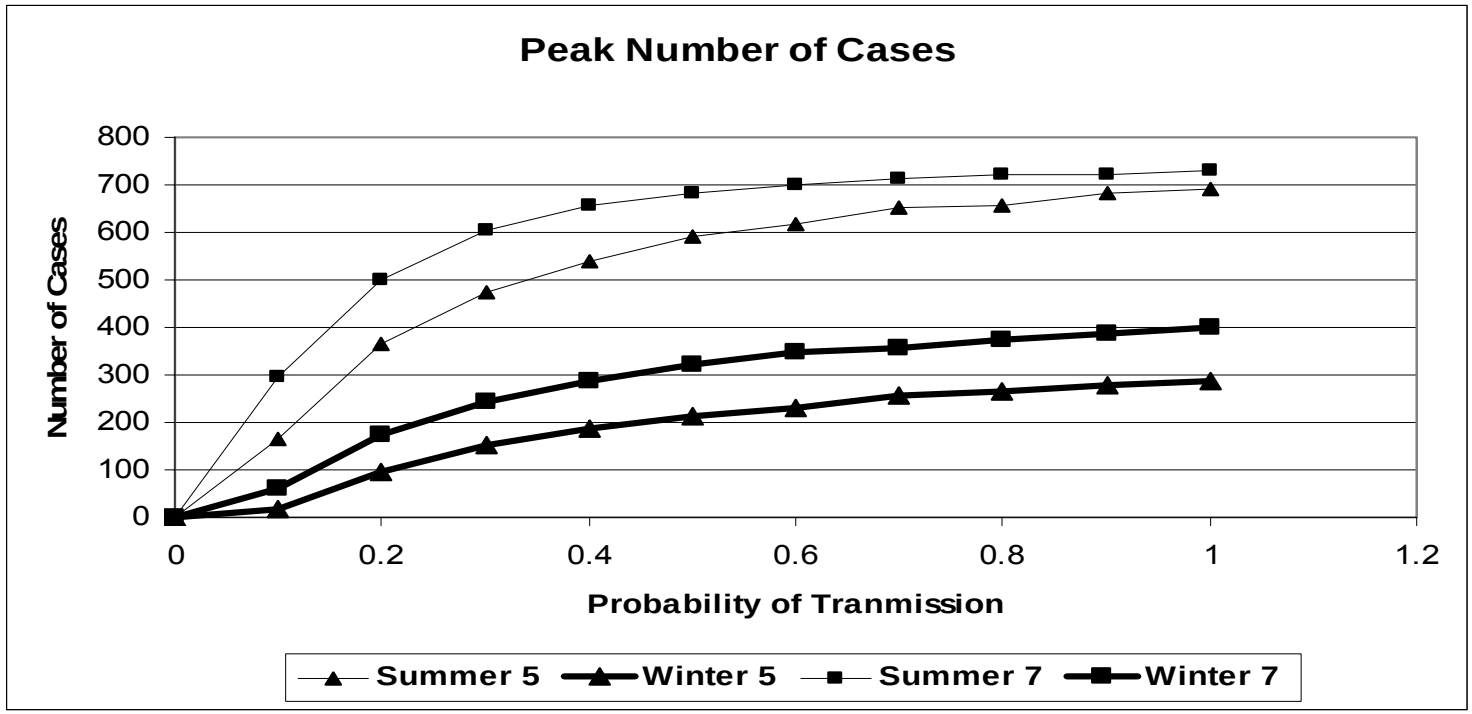

Figure 6.27: The peak number of infected cases during the epidemic in days when the probability of transmission is varied at an infectious period of 5 and 7 days in the summer and winter scenario.
Graph legend: Summer 5 refers to an infectious period of 5 days in the summer scenario.

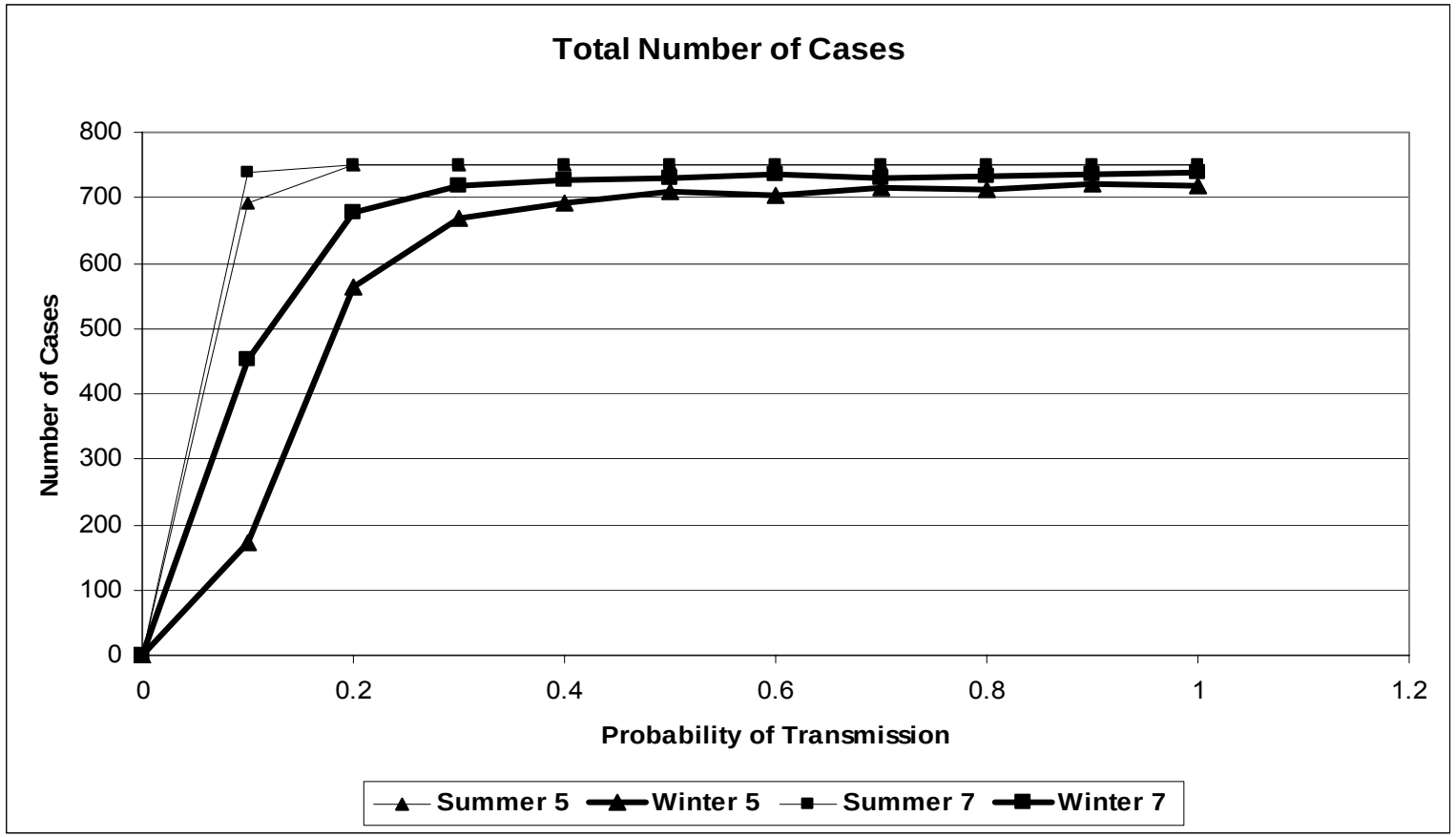

Figure 6.28: The total number of infected cases during the epidemic in days when the probability of transmission is varied at an infectious period of 5 and 7 days in the summer and winter scenario. Graph legend: Summer 5 refers to an infectious period of 5 days in the summer scenario.

- **Summary**

The replication study provided a simple qualitative analysis technique for curves generated by a stochastic model using the range of values and standard deviation for the epidemic characteristics of the winter and summer epidemics at control values. The repetition study determined that averaging 1000 simulations for each parameter change provided a smaller range of variation than averaging 100 simulations, but the average of 100 simulations may be perfectly adequate for many research questions. A summary of the parameter tests is provided in Table 6.16. The winter scenario was impacted by all the parameters tested except the time of the first infection. The summer scenario was impacted by the number of people per family, the contact times, the infectious period, and the transmission. The population size was not tested in the summer scenario. The increase in infectious period in conjunction with an increased transmission probability only had an impact on the peak number of cases, although the total number of cases might have been impacted if the maximum had not been reached so quickly. Evidence of epidemic burn out was seen in four parameters: the probability of contact within families, the probability of contact between families, the infectious period, and the probability of disease transmission. The winter and summer epidemics continued to differ throughout the project even at extreme values. The summer epidemic was short and intense and the winter epidemic was long and less intense. The next chapter will further discuss these results.

Table 6.16: A summary of the impact on epidemic characteristics for each of the parameters used in the model

Parameter	Winter Epidemic	Summer Epidemic
Camp Stay	impacted	no impact
Time of First Infection	no impact	no impact
Path	impacted	no impact
Camp Distance	impacted if > Infectious Period	no impact
Population Size	impacted	not tested
Population proportion	impacted	no impact
Number per Family	impacted	impacted
Contact within Families	impacted	impacted
Contact between Families	impacted	impacted
Infectious Period	impacted	impacted
Transmission	impacted	impacted

Chapter 7: Discussion and Conclusions

Archival information and computer simulation allowed us to glimpse the past. In the summer, we could see the Norway House people gather at the fort in close proximity to friends and families. We could imagine feasts and festivals, trade and negotiation, or simply the sharing and cooperation that occurred between the hunting groups. In the winter, we could see some people separate into small hunting groups and scatter to their traditional family hunting grounds while others remained at the fort. Occasionally, the men would return to the fort from the hunting camps to trade their furs for supplies and quickly return to their families in the camps. We envisioned a harsh, cold environment where living on the land was difficult but possible. Cooperation within and between the hunting groups would have been essential for these people to live in this land. Tragically, the worst flu epidemic in history swept through this community in the winter of 1918-19 (Herring, 1994). We can visualize the epidemic entering the fort with the mail packet and spreading to the winter hunting grounds via the men traveling back and forth. These men would have unwittingly brought disease and frequently death to their families. What cannot truly be imagined is the suffering these people went through that winter. From this tragedy, some knowledge can be gained about the interactions between people and disease. Understanding components of the human-pathogen interaction may help avert tragedies like the Norway House epidemic.

In this project, a computer simulation was constructed using biological information about the influenza virus, historical information about the 1918-1919 influenza epidemic, and ethnographic and demographic information about the Norway House community and their experience with the 1918-1919 influenza epidemic. The modeling technique for this project was new and untested so each parameter in the model required thorough testing and analysis. The resulting model functioned in a manner that is consistent with basic epidemiological principles. Verification studies for each of the parameters in the model demonstrated that the model was working as originally intended. Although no model can simulate the exact events that occurred in the Norway House community during the 1918-1919 influenza epidemic, the basic components of the community structure and the disease that might have influenced the epidemic were portrayed by this model. Any model is only as good as the information used to create it and it is never possible to know whether the parameters chosen were the factors that truly influenced the influenza epidemic. Keeping these caveats in mind, the model was used to explore two basic research questions about the influence of culture on the course of an epidemic.

The answer to the question whether the seasonal population movements of the Norway House community could influence the spread of the flu through the community is yes. A summer scenario, portraying the population aggregated around the fort, and a winter scenario, portraying the population dispersed on the landscape, were used throughout the project. Consistently, the summer and winter scenarios had different epidemic outcomes, as demonstrated by the

epidemic characteristics. In the summer scenario, the entire population is congregated in the fort resulting in a high population density. Individuals were frequently in contact with each other. When the virus was introduced into this population, the epidemic spread quickly through the population due to the high rate of contact and ended quickly because the majority of the susceptible people had been exposed to the virus within a very short period of time. This resulted in a short, intense epidemic in the summer scenario. In the winter scenario, the population is dispersed on the landscape resulting in a lower population density and less frequent contact with other individuals. When the virus was introduced into the fort, the epidemic spread was more limited because people were in less frequent contact with each other. The majority of the individuals were in isolated hunting camps causing the virus to rely on men traveling between the fort and the camps to reach this population. This mechanism of epidemic spread resulted in fewer people being exposed less frequently to the virus. The result was a long and less intense epidemic for the winter scenario. The model demonstrates that seasonal population movement could have influenced the spread of the flu through the community.

The second question addressed whether a single cultural vector, such as seasonal population movement, could be responsible for the observed seasonal differences. The answer again is yes. In the majority of the studies, the summer and winter scenarios were tested under identical parameters where the only variation was a cultural vector, seasonal population movement. The epidemic

outcomes were consistently different in each case. A single cultural behavior or vector can influence the outcome of an epidemic.

An interesting phenomenon occurred during the testing of contact probability, infectious period, and the probability of transmission. The duration and, usually, the peak time would reach a maximum and then decline as the probability or period increased. The peak number, total number, and percent infected would continue to climb until the simulation ended or 100% infection rate occurred. This phenomenon was likely due to the epidemic burning itself out. A high number of people were being infected very rapidly causing the epidemic to end in a shorter period of time. Further tests are needed to substantiate and further explore this phenomenon in this scenario.

Another interesting result was the lack of influence that infectious period had on duration and peak time when the probability of transmission was varied. On the other hand, the peak number was increased when the infectious period was increased in relationship to the probability of transmission. This occurred in both the winter and summer scenario. This phenomenon points to the need for more studies involving the co-variation of parameters. Focusing on a single parameter at a time only presents part of the picture.

Although the model resulted in useful insights into the Norway House flu epidemic, it had several limitations. The parameters, on which the model was based, could be incorrect. Biased, incomplete, or misleading historical, ethnographic, and demographic information could introduce error into the model. When information was not available, assumptions were made which may or may

not have been correct. The biological information about the influenza virus and, specifically, the virus that caused the influenza epidemic in 1918-1919 is constantly being updated and re-evaluated. Other factors that were not in the model could have influenced the epidemic. Possible factors include the psychological impact of being sick with the flu or losing family and friends to the flu, immune function, nutritional status, and health status of the individuals in the community. The epidemic may have involved multiple strains or a different strain of the influenza virus unlike the single strain of influenza portrayed in the model.

Because a new technique for modeling was in development for this project, only very basic parameters were included. Parameters such as birth rate, death rate, health status, and immunity were excluded from the model. In addition, the computer program could have undetected logic errors, which did not affect the operation of the simulation but could have affected the results. The computer program is based on a stochastic system that depends on random number generators and probability functions. Although stochastic modeling can simulate random effects in small population, it can be very difficult to analyze. In other words, the results may be correct but the analysis could be misleading or incorrect. Finally, human error and bias are always a possible source of error. Fortunately, future modification of the model may resolve many of these potential errors in the future.

The computer simulation was constructed with future applications in mind. The first modification to the original model was the addition of the probability of mortality and the time of death from the influenza virus during the epidemic. The

testing of the modified model is in progress. The original research questions and new questions about the impact of the differential probability of transmission and mortality based on age and gender, factors that could cause epidemic burnout, and the effect of co-variation of parameters will be addressed using this model. In addition, modifications are being made to the original program to make the model more user-friendly. A new parameter text file has been added which will change the parameters in the modeling program and document that change. This file decreases the probability of accidental alteration of the modeling program. The development of a more efficient data collecting system is in progress. This process will decrease the number of steps in the data collecting process. The result should be a decrease in human intervention and a decrease in the potential for human error during the data collection process. In addition, the computer simulation is being re-evaluated for modifications, which would decrease the run time per simulation.

Potential model modifications are almost limitless. Possible model modifications include different epidemics, different diseases, different communities, and different human cultural patterns. Human behavioral patterns such as decision-making behaviors could be constructed in the model. Agent-based computer simulations can also be integrated with a geographic information system (GIS) (Kohler et al. 2000) which would lend a more realistic environment to the scenario. Each of these modifications would require a great deal of time and work but the results may be well worth the effort.

In conclusion, agent-based computer simulation can be a valuable tool for anthropological research into historical epidemics. This project allowed me to experiment with individual components of an influenza epidemic in a small population. Specific characteristics of the influenza virus, population structure, seasonal population movement, and the interaction of the individuals within the population were analyzed. Although the model is only a rough facsimile of the real Norway House community during the 1918-1919 influenza epidemic, it did provide insight into the potential factors influencing an epidemic. The model very clearly demonstrated that human behavior is an integral component of an epidemic and that a single human behavior such as seasonal population movement can significantly influence the outcome of an epidemic.

Appendix A

The computer simulation used in this project is written in Java™, an object oriented computer language, and utilizes RePast, an agent-based modeling toolkit, to run the model. The actual programming includes three class files: Paths.java, Agents.java, and Model.java, and a text file, param.txt, which are provided below. The comments and false starts in the program which are differentiated by (///) before the statement are included in the program. These statements are a useful reference when the program is modified for a new project.

Paths Class

```
package norwayhouse;

public class  Paths {
  double moveProbs[];
  int exposed;
  int infected;
  int recovered;
  int susceptible;
  int pop;
  int xcoor;
  int ycoor;
  int number;
  int color;

  public Paths(int x, int y){
      moveProbs = new double[9];
      exposed=0;
      infected=0;
      recovered=0;
      susceptible=0;
      number=0;
      color=0;
      pop=infected+recovered+susceptible;
      xcoor=x;
      ycoor=y;
      for(int i=0;i<9;i++){
        moveProbs[i]=-1;
      }
  }
}
```

Agents Class

```
package norwayhouse;

import java.util.*;

public class Agents {
  String sex;
  String diseaseState;
  int xcoor;
  int ycoor;
  int campNumber;
  int towardsCamp;
  int campX;
  int campY;
  int infectionLength;
  int clique;
  String direction;
  static Random select = new Random();

  public Agents(int X, int Y, double M, double F, double C, int number, String
disease, int cliqueNo, int campDir){
    double T = (double) select.nextInt(1000)/1000;
    direction="none";
    if(T<M)
      sex ="M";
    else if(T<M+F)
      sex = "F";
    else
      sex = "C";

    diseaseState=disease;
    xcoor=X;
    ycoor=Y;
    campX=X;
    campY=Y;
    campNumber=number;
    towardsCamp=campDir;
    infectionLength=0;
    clique =  cliqueNo;

    if(number!=1)
      direction= "fort";
  }
}
```

Model Class

```
package norwayhouse;

import uchicago.src.sim.space.*;
import java.util.*;
import uchicago.src.sim.util.SimUtilities;
import uchicago.src.sim.gui.*;
import uchicago.src.sim.engine.*;
import uchicago.src.sim.analysis.*;
import java.awt.Color;
import uchicago.src.sim.space.*;

public class Model extends SimpleModel{
  //the world for the display colors
  Object2DGrid displayWorld;
  //the world to deal with the paths and the camps
  Object2DGrid pathsWorld;
  //some stuff for the display
  DisplaySurface dsurf;

  DataRecorder recorder;
  //dynamic graph chart
  public OpenSequenceGraph graph;
  //other variables
  int size;
  double p;
  double maleProp;
  double childProp;
  double femaleProp;
  String initDisease;
  double MDiseaseProb;
  double FDiseaseProb;
  double CDiseaseProb;
  double campStay;
  int fortX;
  int fortY;
  int recoveryTime;
  int campPop;
  int fortPop;
  int time;
  int timeOfFirstInfection;
  int pop;
  int infected;
  int exposed;
```

```
  int susceptible;
  int recovered;
  int clique;
  int noCliques;
  double pMeetingWI,pMeetingWO;

  //allows random variables easily
  static Random select = new Random();
  //some stuff for the model
  public Model() {
    Controller.ALPHA_ORDER = false;
  }

// The sequence of the number of susceptible
    class SeqSus implements Sequence {
      // This is the method to be defined for Sequence
      public double getSValue() {
        // Returns the appropriate value
        return (double) susceptible;
      }
    }

    // The sequence of the number of infected
    class SeqInf implements Sequence {
      // This is the method to be defined for Sequence
      public double getSValue() {
        // Returns the appropriate value
        return (double) infected;
      }
    }

    // The sequence of the number of recovered
    class SeqRec implements Sequence {
      // This is the method to be defined for Sequence
      public double getSValue() {
        // Returns the appropriate value
        return (double) recovered;
      }
    }

  public void setup() {
    super.setup();
    // Specify the parameters to be displayed for setting by the user

    params = new String[] { "MDiseaseProb" , "fortPop" };
```

```
    int numOfTimeSteps = 200;
    setStoppingTime(numOfTimeSteps);
    //size of the world
    size=100;
    //probability of moving along a path
    p=1;
    //proportions of people types
    maleProp=0.25;
    femaleProp=0.25;
    childProp=1-maleProp-femaleProp;
    initDisease="S";
    campStay=0.99;
    MDiseaseProb=0.2;
    FDiseaseProb=0.2;
    CDiseaseProb=0.2;
    recoveryTime=5;
    clique = 0;
    noCliques =10;  // Number of groups within fort
    pMeetingWI = 0.5;  // probability of meeting if members are of same clique
    pMeetingWO = 0.001;   // probability of meeting if members are of different
clique
    campPop=150;
    fortPop=150;
    time=0;
    timeOfFirstInfection=20; // time when first infection introduced to fort
    //initializing the worlds
    displayWorld = new Object2DGrid(size,size);
    pathsWorld = new Object2DGrid(size,size);

    // If there is already a graphics object from a previous run, we need
    // to delete it to clean up the screen.
    if (graph != null)
      graph.dispose();
//stuff for the display
    if(dsurf != null)
      dsurf.dispose();
    dsurf= new DisplaySurface(this, "The World");
    registerDisplaySurface("The World", dsurf);

  }

  public void buildModel() {

    //recorder = new DataRecorder("./writeMe.txt", this);
```

```
    //recorder.createNumericDataSource("MDiseaseProb", this,
"getMDiseaseProb");
    //initialize the world spaces
    for(int i=0;i<size;i++){
      for(int j=0;j<size;j++){
        displayWorld.putValueAt(i,j,0);
        Paths P = new Paths(i,j);
        pathsWorld.putObjectAt(i,j,P);
      }
    }

    //create all the rivers and trails
    //first the fort
    Paths fort = (Paths) pathsWorld.getObjectAt(size/2,size/2);
    fort.pop=fortPop;
    fort.susceptable=fort.pop;
    fort.number=1;
    fort.color=3;
    fortX=fort.xcoor;
    fortY=fort.ycoor;
    displayWorld.putValueAt(fort.xcoor,fort.ycoor, fort.color);

    //create the camp1
    Paths camp1 = (Paths) pathsWorld.getObjectAt(size/2+2,size/2);
    camp1.pop=campPop;
    camp1.susceptable=camp1.pop;
    camp1.moveProbs[4]=campStay;
    camp1.moveProbs[3]=1-camp1.moveProbs[4];
    camp1.number=2;
    camp1.color=2;
    displayWorld.putValueAt(size/2+2, size/2,camp1.color);

    //camp2
    Paths camp2 = (Paths) pathsWorld.getObjectAt(size/2,size/2+3);
    camp2.pop=campPop;
    camp2.susceptable=camp2.pop;
    camp2.moveProbs[4]=campStay;
    camp2.moveProbs[7]=1-camp2.moveProbs[4];
    camp2.number=3;
    camp2.color=2;
    displayWorld.putValueAt(size/2,size/2+3,camp2.color);

    //camp3
    Paths camp3 = (Paths) pathsWorld.getObjectAt(size/2-4,size/2);
    camp3.pop=campPop;
```

```
camp3.susceptable=camp3.pop;
camp3.moveProbs[4]=campStay;
camp3.moveProbs[5]=1-camp3.moveProbs[4];
camp3.number=4;
camp3.color=2;
displayWorld.putValueAt(size/2-4, size/2,camp3.color);

 //camp4
 Paths camp4 = (Paths) pathsWorld.getObjectAt(size/2,size/2-5);
 camp4.pop=campPop;
 camp4.susceptable=camp4.pop;
 camp4.moveProbs[4]=campStay;
 camp4.moveProbs[1]=1-camp4.moveProbs[4];
 camp4.number=5;
 camp4.color=2;
 displayWorld.putValueAt(size/2,size/2-5,camp4.color);

//create the paths for camp1
for(int i=size/2+1;i<size/2+2;i++){
  Paths river = (Paths) pathsWorld.getObjectAt(i,size/2);
  river.moveProbs[3]=p;
  river.moveProbs[5]=1-p;
  river.color=1;
  displayWorld.putValueAt(i,size/2,river.color);
}

//create the paths for camp2
for(int i=size/2+1;i<size/2+3;i++){
  Paths river = (Paths) pathsWorld.getObjectAt(size/2,i);
  river.moveProbs[7]=p;
  river.moveProbs[1]=1-p;
  river.color=3;
  displayWorld.putValueAt(size/2,i,river.color);
}

//create the paths for camp3
   for(int i=size/2-1;i>size/2-4;i--){
     Paths river = (Paths) pathsWorld.getObjectAt(i,size/2);
     river.moveProbs[5]=p;
     river.moveProbs[3]=1-p;
     river.color=5;
     displayWorld.putValueAt(i,size/2,river.color);
   }

//create the paths for camp4
```

```
      for(int i=size/2-1;i>size/2-5;i--){
        Paths river = (Paths) pathsWorld.getObjectAt(size/2,i);
        river.moveProbs[1]=p;
        river.moveProbs[7]=1-p;
        river.color=6;
        displayWorld.putValueAt(size/2,i,river.color);
         }

//create the agents in the fort
   for(int i=0;i<fort.pop;i++){

       clique = select.nextInt(noCliques)+ 1;
       Agents A = new
Agents(fort.xcoor,fort.ycoor,maleProp,femaleProp,childProp,fort.number,initDisea
se,clique,4);
       //System.out.println("A.clique= " + A.clique );
     agentList.add(A);
   }

//creates the agents for camp1
   for(int i=0;i<camp1.pop;i++){
     clique = select.nextInt(noCliques)+ 1;
     Agents A = new
Agents(camp1.xcoor,camp1.ycoor,maleProp,femaleProp,childProp,camp1.numb
er,initDisease,clique,5);
     agentList.add(A);
   }

 //creates the agents for camp2
   for(int i=0;i<camp2.pop;i++){
     clique = select.nextInt(noCliques)+ 1;
     Agents A = new
Agents(camp2.xcoor,camp2.ycoor,maleProp,femaleProp,childProp,camp2.numb
er,initDisease,clique,1);
     agentList.add(A);
   }

//creates the agents for camp3
   for(int i=0;i<camp3.pop;i++){
     clique = select.nextInt(noCliques)+ 1;
     Agents A = new
Agents(camp3.xcoor,camp3.ycoor,maleProp,femaleProp,childProp,camp3.numb
er,initDisease,clique,3);
     agentList.add(A);
   }
```

```
 //creates the agents for camp4
    for(int i=0;i<camp4.pop;i++){
      clique = select.nextInt(noCliques)+ 1;
      Agents A = new
Agents(camp4.xcoor,camp4.ycoor,maleProp,femaleProp,childProp,camp4.numb
er,initDisease,clique,7);
      agentList.add(A);
      }

    int M=0;
    int F=0;
    int C=0;

    for(int i=0;i<agentList.size();i++){

      Agents A = (Agents) agentList.get(i);
      if(A.sex=="M")
        M++;
      if(A.sex=="F")
        F++;
      else if(A.sex=="C")
        C++;
    }

    System.out.println("Pop: "+agentList.size()+", M: "+M+", F: "+F+", C: "+C);

// Create a sequence chart graph and set it up
      // (For details see Repast's appropriate "How to" document.)
      graph = new OpenSequenceGraph("Frequencies", this);
      graph.setXRange(0, 200);
      graph.setYRange(0,100);
      graph.setAxisTitles("Generations", "Number");
      // Add the sequences we have defined
      graph.addSequence("INF", new SeqInf());
      graph.addSequence("SUS", new SeqSus());
      graph.addSequence("REC", new SeqRec());

      // Display the graph right away
      graph.display();
      // Do the first update to it
      graph.step();

   //and then the dimensions of the grid
      DisplayConstants.CELL_WIDTH = 6;
```

```
    DisplayConstants.CELL_HEIGHT = 6;

    //I then create the colors; a different color for
    //each type of agent
    ColorMap Color_world = new ColorMap();
    Color_world.mapColor(0,Color.darkGray);
    Color_world.mapColor(1,Color.red);
    Color_world.mapColor(2,Color.green);
    Color_world.mapColor(3,Color.yellow);
    Color_world.mapColor(4,Color.blue);
    Color_world.mapColor(5,Color.white);
    Color_world.mapColor(6,Color.orange);

//this tells the program that the displays are there
    Value2DDisplay display_world = new
Value2DDisplay(displayWorld,Color_world);
    dsurf.addDisplayable(display_world, "display1");
    addSimEventListener(dsurf);

//this displays them right away
    for(int i=0;i<size;i++){
      for(int j=0;j<size;j++){
        Paths check = (Paths) pathsWorld.getObjectAt(i,j);
        if(check.pop>0)
          displayWorld.putValueAt(i,j,4);
      }
    }

    dsurf.display();

    int pop=0;
    int infected=0;
    int susceptable=0;
    int recovered=0;
    //updating the display
    for(int i=0;i<size;i++){
      for(int j=0;j<size;j++){
        Paths check = (Paths) pathsWorld.getObjectAt(i,j);
        if(check.pop>0)
          displayWorld.putValueAt(i,j,4);
        else
          displayWorld.putValueAt(i,j,check.color);

        pop+=(int) check.pop;
        infected+=(int) check.infected;
```

```
        susceptable+=(int) check.susceptible;
        recovered+=(int) check.recovered;

       }
      }

     System.out.println(time+", "+pop+", "+susceptible+", "+infected+",
"+recovered);
     time++;
    }

  public void step() {

     //adds an infected agent
      if(time==timeOfFirstInfection){
       //initialize with one infected, change this so that the initial infected agent
happens
     //outside the transient state of the model
     Agents X = (Agents) agentList.get(0);
     Paths fort = (Paths) pathsWorld.getObjectAt(X.xcoor,X.ycoor);
     X.diseaseState="I";
     fort.susceptible--;
     fort.infected++;

      }

      for(int i=0;i<agentList.size();i++){

        Agents person = (Agents) agentList.get(i);

        //System.out.println("agent " + i + " = " + person.diseaseState);

        if(person.diseaseState == "E") {
           person.diseaseState = "I";
           Paths update = (Paths) pathsWorld.getObjectAt(person.xcoor,
person.ycoor);
           update.infected++;
           update.exposed--;
        }

        if (person.diseaseState != "S") {
           Disease(person);
           continue;
        }
```

```
        encounters(person, i);
        //System.out.println("I'm here");
        Disease(person);
       }

      for(int i=0;i<agentList.size();i++){
        Agents person = (Agents) agentList.get(i);
        if(person.sex=="M")
          Move(person);
      }
      pop=0;
      infected=0;
      susceptible=0;
      recovered=0;
      exposed=0;
      //updating the display
      for(int i=0;i<size;i++){
       for(int j=0;j<size;j++){
         Paths check = (Paths) pathsWorld.getObjectAt(i,j);
         if(check.pop>0)
           displayWorld.putValueAt(i,j,4);
           //System.out.println("check.infected= " + check.infected);
         else
           displayWorld.putValueAt(i,j,check.color);

         //pop+=(int) check.pop;
         infected+=(int) check.infected;
         susceptible+=(int) check.susceptable;
         recovered+=(int) check.recovered;
         exposed+=(int)check.exposed;
       }
      }

      pop=infected + susceptible + recovered + exposed;

      Paths camp1 = (Paths) pathsWorld.getObjectAt(size/2+2,size/2);
      Paths camp2 = (Paths) pathsWorld.getObjectAt(size/2,size/2+3);
      Paths camp3 = (Paths) pathsWorld.getObjectAt(size/2-4,size/2);
      Paths camp4 = (Paths) pathsWorld.getObjectAt(size/2,size/2-5);
      Paths fort = (Paths) pathsWorld.getObjectAt(fortX, fortY);

      //System.out.println("F= "+fort.infected+" C1= "+camp1.infected+" C2=
"+camp2.infected+" C3= "+camp3.infected+" C4= "+camp4.infected);
```

```
    System.out.println(time+", "+pop+", "+susceptible+", "+infected+",
"+recovered+",   "+exposed+",  "+fort.infected+",   "+camp1.infected+",
"+camp2.infected+",   "+camp3.infected+",   "+camp4.infected);

     //recorder.record();
     // ... and write it into the file right away.
    // recorder.writeToFile();

     graph.step();
     dsurf.updateDisplay();
     time++;
    }

  public void Disease(Agents person){

        Paths P = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
        double disProb = (double) (select.nextInt(1000)+1)/1000;
        double disease=0;
        int infected = 0;

        if(person.infectionLength==recoveryTime){
          person.diseaseState = "R";
          P.infected--;
          P.recovered++;
          person.infectionLength=0;
        }

        if(person.diseaseState=="I") {
           person.infectionLength++;
           //System.out.println("person.infectionLength= " + person.infectionLength);
        }
    }

  public void Move(Agents person){
      //chooses only the males

     Paths spot = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
     double pM = (double) (select.nextInt(1000)+1)/1000;
     double sumprobs=0;
       int nextAction=10;
       int i=0;

//defines the movement if the agents are going towards the fort
        if(person.direction=="fort"){
          while (i < 9 && pM > sumprobs) {
```

```
            //System.out.println(  "i= " + i + " moveprob= " +spot.moveProbs[i]);
            if(spot.moveProbs[i] != -1)
            sumprobs += spot.moveProbs[i];

           if (pM <= sumprobs) {
            //System.out.println(i + " " + pM + " " + person.campNumber + " ");
            nextAction = i;
           }

           i++;
         }
        }

        if((person.xcoor==person.campX&&person.ycoor==person.campY)&&
person.direction=="camp"){
        //if((true && true) && true) {
           person.direction="fort";
           nextAction = 4;
            //System.out.println("true");
        }

//reverses the probabilities for agents going towards the camp
        if(person.direction=="camp"){
          while (i < 9 && pM > sumprobs) {
           if(spot.moveProbs[i] != -1)
             sumprobs += 1-spot.moveProbs[i];
           if (pM <= sumprobs) {
             nextAction = i;
           }

           i++;

         }
        }

        //actions for agents at the forts
        if(person.xcoor==fortX && person.ycoor==fortY){
          person.direction="camp";
          nextAction=person.towardsCamp;
        }

        //if the agents are at the camp
        if(person.xcoor==person.campX && person.ycoor==person.campY) {
           person.direction="fort";
        }
```

```
//updates the population of the spot
if (person.diseaseState=="S") {
    spot.susceptible--;
}

if (person.diseaseState=="I") {
    spot.infected--;
}

if (person.diseaseState=="R") {
    spot.recovered--;
}

spot.pop=spot.infected+spot.recovered+spot.susceptible;

  //the movements of the agents
if(nextAction==0){
  person.xcoor--;
  person.ycoor++;
  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;

  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}

if(nextAction==1){

  person.ycoor++;
  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;
```

```
  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}

if(nextAction==2){
  person.xcoor++;
  person.ycoor++;
  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;

  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}

if(nextAction==3){
  person.xcoor--;

  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;

  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}

if(nextAction==4){

  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
```

```
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;

  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}

if(nextAction==5){
  person.xcoor++;

  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;

  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}

if(nextAction==6){
  person.xcoor--;
  person.ycoor--;
  Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
  //updates the population of the spot
  if(person.diseaseState=="S")
    up.susceptible++;

  if(person.diseaseState=="I")
    up.infected++;

  if(person.diseaseState=="R")
    up.recovered++;

  up.pop=up.infected+up.recovered+up.susceptible;
}
```

```
      if(nextAction==7){

        person.ycoor--;
        Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
        //updates the population of the spot
        if(person.diseaseState=="S")
          up.susceptible++;

        if(person.diseaseState=="I")
          up.infected++;

        if(person.diseaseState=="R")
          up.recovered++;

        up.pop=up.infected+up.recovered+up.susceptible;
      }

      if(nextAction==8){
        person.xcoor++;
        person.ycoor--;
        Paths up = (Paths) pathsWorld.getObjectAt(person.xcoor,person.ycoor);
        //updates the population of the spot
        if(person.diseaseState=="S")
          up.susceptible++;

        if(person.diseaseState=="I")
          up.infected++;

        if(person.diseaseState=="R")
          up.recovered++;

        up.pop=up.infected+up.recovered+up.susceptible;


    }
  }

public void  encounters(Agents person, int i) {
   double probMeeting = 0;

   for(int j=0; j < agentList.size(); j++) {
      //System.out.println("i = " + i + " j= " + j);
      if (i==j){
         //System.out.println( "i = j " + i + j);
         continue;
```

```
        }
        Agents personEncountered = (Agents)agentList.get(j);
        if (personEncountered.diseaseState != "I") {
           //System.out.println("diseaseState = " +
personEncountered.diseaseState);
           continue;
        }

          if (person.xcoor == personEncountered.xcoor && person.ycoor ==
personEncountered.ycoor)   {
             if (person.clique == personEncountered.clique) {
                //System.out.println("clique = clique");
                probMeeting = pMeetingWI;
              } else  {
                //System.out.println("clique != clique");
                probMeeting = pMeetingWO;

                }


       }
          double prob = (double) (select.nextInt(1000)+1)/1000;

          if (probMeeting >= prob) {
             prob = (double) (select.nextInt(1000)+1)/1000;

             if (person.sex=="M" && MDiseaseProb >= prob) {
                person.diseaseState = "E";

                //System.out.println("newdiseasestate= " + person.diseaseState);
                Paths P = (Paths)
pathsWorld.getObjectAt(person.xcoor,person.ycoor);
                P.exposed++;
                P.susceptible--;
                break;
             }

             if (person.sex=="F" && FDiseaseProb >= prob) {
                person.diseaseState = "E";

                //System.out.println("newdiseasestate= " + person.diseaseState);
                Paths P = (Paths)
pathsWorld.getObjectAt(person.xcoor,person.ycoor);
                P.exposed++;
                P.susceptible--;
```

```
                break;
            }

            if (person.sex=="C" && CDiseaseProb >= prob) {
                person.diseaseState = "E";

                //System.out.println("newdiseasestate= " + person.diseaseState);
                Paths P = (Paths)
pathsWorld.getObjectAt(person.xcoor,person.ycoor);
                P.exposed++;
                P.susceptible--;
                break;
            }

        } else {}

    }


 }

 public double getMDiseaseProb() {
    return MDiseaseProb;
  }

 public void setMDiseaseProb (double n) {
    MDiseaseProb = n;
 }

 public int getFortPop() {
    return fortPop;
 }

 public void setFortPop (int n) {
    fortPop = n;
 }

  public String[] getInitParam() {
     String[] params = {"MDiseaseProb"};
     return params;
  }

 public static void main(String[] args) {
     SimInit init = new SimInit();
  Model m = new Model();
```

```
    init.loadModel(m, "C:/Documents and Settings/Connie/My
Documents/param.txt"

,true);
  //init.loadModel(m, null, false);
 }
  }
```

Parameters (param.txt)

```
runs: 400
MDiseaseProb {
 start: 0.15
end: 0.15
incr: .000

}
```

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