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The Devil in the Details: Evidence for the Affliction of Lyme Disease in Seventeenth Century Massachusetts

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THE DEVIL IN THE DETAILS: EVIDENCE FOR THE
AFFLICTION OF LYME DISEASE IN SEVENTEENTH
CENTURY MASSACHUSSETTS

A THESIS
SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF MASTER OF ARTS
UNIVERSITY OF SOUTHERN MAINE

AMERICAN AND NEW ENGLAND STUDIES

BY

MARY DRYMON DEROSE

2005

THE UNIVERSITY OF SOUTHERN MAINE
AMERICAN AND NEW ENGLAND STUDIES

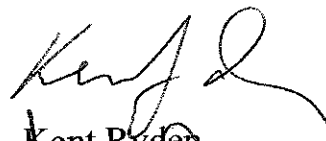
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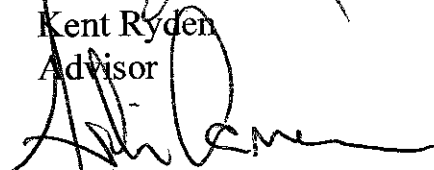
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THE DEVIL IN THE DETAILS: EVIDENCE FOR THE AFFLICTION OF
LYME DISEASE IN SEVENTEENTH CENTURY MASSACHUSSETTS

Be accepted as partial fulfillment of the requirements for the Degree of
Master of Arts.



Kent Ryden
Advisor



Ardis Cameron
Reader

Accepted



Dean, College of Arts and Sciences

ABSTRACT

This study looks for evidence that Lyme disease is an old affliction that predates its 'discovery' in Connecticut in the nineteen seventies. It analyzes the role that Lyme disease may have played in the history of English settlement in Massachusetts during the seventeenth century. Early settlers at Plymouth and in the Boston area described sicknesses that they suffered from at contact as being the result of starvation and scurvy. By 1692, the residents of the Salem Village area were describing physical and mental afflictions that they felt were caused by witchcraft. Some of the seventeenth-century symptoms are very similar to those that are suffered during Lyme disease. Various factors including deer and mouse population levels, the amount of mast (acorns) available, and the environmental landscape have been found to contribute to the modern risk of acquiring Lyme disease. Looking for similar factors in the past, it appears that many of the historic "afflicted" interacted with what can be described as a 'tick risky' environment. In Salem Village, the east/west division was more than a social configuration. It was a demarcation related to the very land itself. And it can be used to predict the risk of exposure to Lyme disease. This points to an environmental origin for the witchcraft afflictions.

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INTRODUCTION

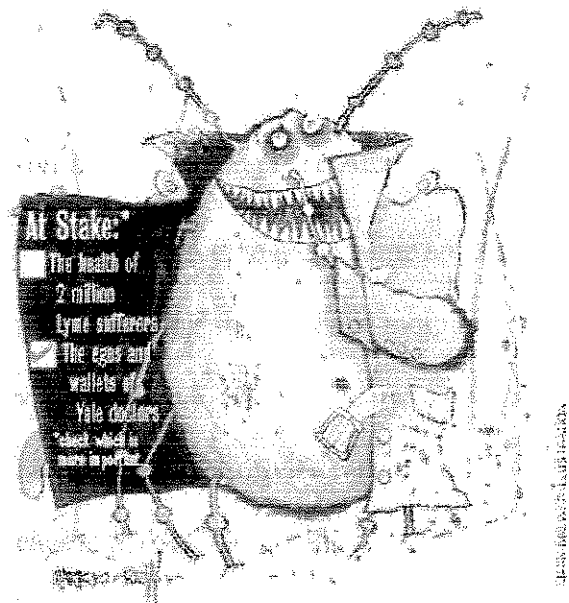
This work began when I read a written description of the symptoms of some of the 'afflicted' from the Salem Witch Trials of 1692 for the first time. I found the descriptions to be startlingly familiar as symptoms that I had experienced while suffering from Lyme disease. This led me to a search for evidence of Lyme disease in New England's past. This task has been complicated because the modern understanding of Lyme disease is very much a work in progress that has often raised many more questions than it has answered. I have found Lyme disease to be a surprisingly controversial topic to study. My interest in this subject is motivated by my background as a patient and as the moderator for a local Lyme Support Group. Over the past ten years I have come into contact with members of the medical profession and many of my contemporary Lyme sufferers. The story that can be told about the affliction of Lyme disease is a compelling one.

Modern Lyme disease research is a very complex tableau of contrasts: cutting edge science sharply framed by controversy and complaint set against a dark backdrop of conflicts of interest. Some of the most shining Lyme-epidemiological heroes of the late nineteen seventies have found their reputations tarnished in more recent times. Dr. Alan Steere, the Yale researcher who helped give what he considered a new disease its name, now works in a laboratory in Boston that has to be protected by security guards. He has received death threats. He is unable to publicly lecture on his pioneering work without being picketed by members of the radical Lyme Activist Movement. This group of Lyme patients objects to his decision to back away from his own initial definition of a persistent

third neurological stage for Lyme infection, identifying it instead as an autoimmune reaction. His redefinition helped limit the length of antibiotic therapy for many patients and may have given rise to a surge in Fibromyalgia as yet another rapidly emerging condition during the late nineteen eighties.¹

The scientific literature itself is fraught with contradictions. There is a stark dichotomy of opinion about the severity of Lyme disease. Clearly Lyme disease is not life threatening in the majority of cases, but numerous articles persist in repeating the

statement that “no one has ever died from Lyme disease.”² This statement is directly contradicted by a clear record of death certificates that list Lyme disease as the cause of death. Since many autopsies do not routinely include tissue and fluid testing for the *Borrelia burgdorferi* bacteria, the death rate is currently unknown.³ Lyme disease is described as both “an easily treatable condition” and “a complex, difficult to diagnose” problem that is “the leading cause of vector borne illness in the United States today.”⁴ The



The Dirty Truth About Lyme Disease Research

Figure 1. Lyme Cartoon from The Fairfield County Weekly in Connecticut.

key blood tests, the Western Blot and the Enzyme Linked Immunosorbent Assay (ELISA) that are used to diagnose the disease, are subject to controversy. They are notorious for false results. And even the very determination of what constitutes a positive

test result changed in 1994 when the United States Food and Drug Administration (hereafter FDA) removed two outer surface protein (Osp) marker bands as positive diagnostic markers. Lyme activists see a conspiracy in this action, saying that Lyme disease itself was redefined to help facilitate the development of the Lymerix vaccine. Lyme activists have protested that this new standard effectively eliminates eighty percent of those who are suffering from Lyme disease from ever testing positive. It has been suggested that this new standard was set to make an ineffective vaccine (that both the federal government and a drug company had heavily invested in) appear to be highly successful by seeming to protect at least eighty percent of the tested population. There would appear to be some money related conflicts of interest between those who stood to profit from the vaccine- university patent holders, drug companies, and the FDA- and the welfare and health of the American public.⁵

The vaccine made some apparently healthy people who participated in testing trials very sick and sickened more after it was approved for use on adults. These maimed victims seem to either have had a pre-existing infection that the vaccine reactivated⁶ or had a genetic sensitivity caused by HLA-DR4 and HLA-DR2 alleles that reacted and caused permanent arthritic conditions.⁷ When the SmithKlineGlaxo Company voluntarily recalled the vaccine due to "a lack of interest," it is estimated that they had already reaped a twenty million dollar net profit. Lyme disease vaccine research is still heavily funded by the National Institute of Health, the National Institute of Allergy and Infectious Diseases, and the Food and Drug Administration. A recently published study now suggests that we could control Lyme disease by vaccinating all the wild mice in the United States. The profit from such a plan would be a bonanza for the drug companies

involved. As of today, federally funded Lyme disease research has gleaned over a billion dollars in grants, and worldwide sales of testing supplies for this single disease could annually top the billion dollar mark within two years.⁸

This tidal wash of cash has not found a cure and seems to have done little to halt the continuing spread of Lyme disease, especially in New England (where Maine is currently the frontier for infection), or protect the population from the disease. The United States Center for Disease Control reported that the incidence rate for Lyme disease jumped by forty percent between 2001 and 2002 to an all time high.⁹ It continues to climb. The common sense list of personal precautions to protect against ticks has not changed in over fifteen years and has actually never been subjected to blind tests to verify efficacy.¹⁰ Current treatment protocols range from those described as “highly effective” to the standard treatment for third stage Lyme disease being described by one activist as “no treatment at all.” Lyme disease science is described as being both cutting edge and unresponsive to the afflictions of Lyme disease patients.¹¹

A similar mental dichotomy can be found in relation to the witchcraft afflictions in the seventeenth century. When there was public support for the diagnosis of witchcraft, the Massachusetts Bay Colony threw massive amounts of political attention and legal might into the problem. When public opinion had turned against this diagnosis, the symptoms of affliction became less important. Very little is known about the later life of the “afflicted girls” from 1692. Abigail Williams is supposed to have died young. Betty Parris grew up to marry and lived to an old age. Did their afflictions continue even after several people were executed for causing them? The answer seems to be a qualified yes. When Ann Putnam, Jr. stood up in church several years later to apologize for her actions

she stated that she had long “continued to be ill.” While 1692 stands out because of the written record of the Salem Trials, there are large silent gaps in the history of affliction in Massachusetts that may never be fully understood.¹²

In many ways Lyme disease assaults modern cultural biases. We no longer have the seventeenth century’s dread of the wilderness. This is a disease contracted as we contact nature. Nature is supposed to be, as Martha Stewart would say, “a very good thing.” As a culture we have gone back to nature. We eat natural foods. We commune with nature. We contribute to the Fresh Air Fund. We rally on Earth Day. We support environmental causes. It is almost unfathomable that such a good thing could make anyone sick. So we equivocate. In the nineteen eighties Lyme disease had a high profile. When modern science had not eradicated it by the nineteen nineties, it moved to the edges of the national radar screen. The disease’s very complexity may be more than the American public is willing to comprehend.

The modern debate about the affliction of Lyme disease is complicated by the ongoing controversy between whether this is a *new* disease, perhaps the result of an errant escape of germs from Plum Island, the United States Department of Agriculture research lab located off the coast of Long Island, New York¹³, or a very ancient form of affliction that peaks and ebbs in response to environmental and other conditions.¹⁴ This work will investigate the concept of Lyme disease as an *old* infection. It will focus specifically on seventeenth-century Massachusetts, where sporadic afflictions are recorded but given diagnoses that are idiosyncratic to that specific century.

In his book *Making Sense of Illness*, Robert Aronowitz discusses the processes by which we recognize, name, classify, and find meaning in illness. He specifically

discusses Lyme disease in a chapter entitled "*The Social Construction of a New Disease and Its Social Consequences*." The social construction of a disease is the way that non-biological factors like beliefs, economic relationships, and societal institutions greatly influence, if not define, a social group's understanding of any particular illness.

Aronowitz uses the modern "discovery" of Lyme disease in the early 1970's as a classic case study for the social construction of disease. Lyme disease was conceptualized in Connecticut as a *new* disease for a variety of reasons that influenced the prevailing interpretation of a set of biological and epidemiological facts. These included the nationality of investigators (American vs. European), the disciplinary background of the medical researchers at Yale University who named the disease (rheumatologists vs. dermatologists), intellectual or attitudinal features (American skepticism of the quality of past research and European intelligence) and professional concerns (potential self-interest in being the medical pioneer who discovered a new disease). European dermatological research, dating back to as early as 1910, had described an expanding ring shaped rash that developed at the site of a tick bite (the classic Erythema Chronicum Migrans [hereafter ECM] or bull's eye rash.) Studies done in Norway during the 1930's identified possible bacterial spirochetes in ECM lesions. A study done in 1955 found penicillin to be highly effective for treating ECM. In 1970, North American dermatology textbooks that mentioned ECM, described it as an infective process resulting from a tick bite that was possibly caused by spirochetes and was responsive to antibiotics. The first Center for Disease Control (CDC) reported case of ECM in the United States was from Wisconsin in 1969. The first modern case cluster of Lyme disease in the United States was identified by dermatologists working at the Naval Submarine Medical Center in Groton,

Connecticut, during the summer of 1975. It was diagnosed as an outbreak of ECM and effectively treated with antibiotics.

It was the rheumatologists from Yale University, however, who were called in later to study the group of sick children living in Lyme, Connecticut in the early nineteen seventies. They dubbed the sickness Lyme arthritis. Severed from any historical past and tainted by the European association of earlier research, the disease went on to be publicly accepted in the United States as a newly emerging disease that was 'discovered' by American medical researchers. A myriad of symptoms caused by the same bacteria soon began to be recognized---including among others neurological problems, the set of symptoms called Bannworth's syndrome, and skin lesions. At the behest of Dr. William Burgdorfer, the discoverer of the Lyme spirochete and a Lyme victim himself, the afflictions were given the uniform name of Lyme disease. ECM, once the name of the disease in and of itself, was relegated to become the name of the ailment's characteristic rash.¹⁵

By conceptualizing Lyme disease as a new disease, the need to look for its role in the past was essentially eliminated. When it *is* looked for, there is evidence that challenges this perception of newness. For example, DNA amplification work has found that the *Borrelia* bacterium dates back at least to the Paleolithic Age.¹⁶ The *Borrelia burgdorferi* bacterium that causes Lyme disease and its *Ixodes scapularis* tick host have probably been coexisting in North America since long before the creation of the United States. Part of the ecological process for this disease may have even been understood by pre-contact Native American groups who clearly linked fever and joint pain with human

interactions with deer. They constructed the sickness as being the direct result of disrespect for the spirit of the deer, cloaking it in religious overtones.

When the earliest English colonists and their domesticated animals began to occupy the same land, they suffered from many ailments compatible with a diagnosis of Lyme disease. These early settlers constructed their definition for their sickness by contrasting their existence with the life they had left behind in England. They were sick, they felt, because of *starving times*, exposure to the elements, and scurvy. They believed that their cows languished because they were forced to eat American grass that was either too rich or inferior to their accustomed diet of English grass. By 1692, a long list of afflicted persons (and their animals) in Massachusetts had sporadically experienced symptoms for over seventy years. The Salem Witch Trials gave some of them the chance to describe their experiences for the written historical record. Testimony records the preternatural appearance of pins sticking into the flesh of victims, various skin rashes that often looked like round bite marks, lameness and swellings, and a long list of neurological afflictions and hallucinations. The landscape that the victims and accused witches lived in and described, when plotted onto a map, contains large areas of terrain that could be classified in modern times as tick-risky. Young children and women who traversed this landscape in their long (tick collecting) skirts and petticoats seem to have suffered from a higher rate of infection than their male counterparts. Mysterious illnesses struck domesticated animals.

The list of Lyme-like neurological symptoms is a long one: it includes a child with Bell's palsy, seizures, the sensation of heat and pricking on the skin, transitory sensory loss, irritability, lethargy, and occasionally a fatality. The sicknesses and

afflictions of 1692 were medically, socially and culturally constructed as being the result of witchcraft. The cultural and social acceptance of this definition led to the legal prosecution of those accused of practicing witchcraft.

Over the course of one century Lyme disease may have gone from being defined as an illness associated with deer, to being scurvy, to being the result of witchcraft. By the twenty-first century it is once again defined as a disease associated with deer. Lyme disease may have also acquired some new labels during its passage through time. Some of the upper class rusticators of the early twentieth century, for example, after reestablishing contact with the *healthy* natural world suffered from neurasthenia¹⁷, an ill defined disease which had some very Lyme- like neurological and fatigue characteristics. Modern physicians have depicted patients who experience the symptoms of neurological Lyme disease as having yet another class of disease--the psychiatric disorder.

When the twentieth-century patient, Polly Murray (who later became one of New England's leading Lyme activists) was admitted to the mental ward of a Boston hospital,¹⁸ she may have been reluctantly following in the footsteps of several centuries' worth of Lyme disease sufferers. Lyme disease, called "the great imitator" because it carries with it such a wide array of symptoms ¹⁹[See Figures 2 and 3], may have been an omnipresent force throughout New England's history.

This paper will explore the environmental and cultural context for Lyme-like disease in seventeenth-century Massachusetts, assess risk, and attempt to contrast the personal perceptions from historic records with modern research. It will find evidence of Lyme disease lurking in historic records, hidden by the haze of time. For example, if it

Figure 2. Diagnostic Checklist for diagnosis of Lyme disease.

Figure 2.

DIAGNOSTIC CHECKLIST

To aid the clinician, a workable set of diagnostic criteria were developed with the input of dozens of front line physicians. The resultant document has proven to be extremely useful not only to the clinician, but it also can help clarify the diagnosis for third party payers and utilization review committees. It is important to note that the CDC's published reporting criteria are for surveillance only, not for diagnosis

LYME BORRELIOSIS DIAGNOSTIC CRITERIA	RELATIVE VALUE
Tick exposure in an endemic region.....	1
Historical facts and evolution of symptoms consistent with Lyme	2
Systemic signs & symptoms consistent with Bb infection (other potential diagnoses excluded):	
Single system, e.g., monoarthritis.....	1
Two or more systems, e.g., monoarthritis and facial palsy.....	2
Erythema migrans, physician confirmed.....	7
Acrodermatitis Chronica Atrophicans, biopsy confirmed	7
Seropositivity.....	3
Seroconversion on paired sera.....	4
Tissue microscopy, silver stain.....	3
Tissue microscopy, monoclonal immunofluorescence.....	4
Culture positivity.....	4
B. burgdorferi antigen recovery	4
B. burgdorferi DNA/RNA recovery	4

DIAGNOSIS

Lyme Borreliosis Highly Likely.....	7 or above
Lyme Borreliosis Possible.....	5-6
Lyme Borreliosis Unlikely.....	4 or below

I suggest that when using these criteria, you state Lyme Borreliosis is "unlikely", "possible", or "highly likely" based upon the following criteria"- then list the criteria.

[This is a Diagnostic Checklist used by Dr. Joseph Burrascano to help identify patients with Lyme disease. The symptoms Checklist on the next page is filled out by new patients and is used to aid diagnosis. It shows the wide variety of symptoms that can be experienced as well as other problems (like heart murmur) that Lyme can mimic.]

Figure 3.

SYMPTOM CHECK LIST

This is not meant to be used as a diagnostic scheme, but is provided to streamline the office interview.
Note the format- complaints referable to specific organ systems are clustered to better display multisystem involvement.

NAME _____ DATE _____

RISK PROFILE (PLEASE CHECK)

Tick infested area _____ Frequent outdoor activities _____ Hiking _____ Fishing _____ Camping _____
Gardening _____ Hunting _____ Ticks noted on pets _____ Other household members with Lyme _____

Do you remember being bitten by a tick? _____ No _____ Yes _____ when _____

Do you remember having the "bull's eye rash"? _____ No _____ Yes _____

Any other rash? _____ No _____ Yes _____

Have you had any of the following? CIRCLE ALL YES ANSWERS

1. Unexplained fevers, sweats, chills, or flushing
2. Unexplained weight change- (loss or gain- circle one)
3. Fatigue, tiredness, poor stamina
4. Unexplained hair loss
5. Swollen glands: list areas _____
6. Sore throat
7. Testicular pain/pelvic pain
8. Unexplained menstrual irregularity
9. Unexplained milk production; breast pain
10. Irritable bladder or bladder dysfunction
11. Sexual dysfunction or loss of libido
12. Upset stomach or abdominal pain
13. Change in bowel function- (constipation, diarrhea)
14. Chest pain or rib soreness
15. Shortness of breath, cough
16. Heart palpitations, pulse skips, heart block
17. Any history of a heart murmur or valve prolapse?
18. Joint pain or swelling: list joints _____
19. Stiffness of the joints or back
20. Muscle pain or cramps
21. Twitching of the face or other muscles
22. Headache
23. Neck creaks and cracks, neck stiffness, neck pain
24. Tingling, numbness, burning or stabbing sensations, shooting pains, skin hypersensitivity
25. Facial paralysis (Bell's Palsy)
26. Eyes/Vision: double, blurry, increased floaters, light sensitivity
27. Ears/Hearing: buzzing, ringing, ear pain, sound sensitivity
28. Increased motion sickness, vertigo, poor balance
29. Lightheadedness, wooziness, unavoidable need to sit or lie down
30. Tremor
31. Confusion, difficulty in thinking
32. Difficulty with concentration, reading
33. Forgetfulness, poor short term memory, poor attention, problem absorbing new information
34. Disorientation: getting lost, going to wrong places
35. Difficulty with speech or writing; word or name block
36. Mood swings, irritability, depression
37. Disturbed sleep- too much, too little, fractionated, early awakening
38. Exaggerated symptoms or worse hangover from alcohol

was June of 1630, and you had just taken a long sea voyage and then you came down with a fever, your joints began to ache, and you were too tired to move-- even though you were eating strawberries-- was it unreasonable for you to "know" that you suffered from scurvy? We now know that strawberries are packed with vitamin C, making scurvy an unlikely diagnosis. We also know that June is a month where there is a high risk for contact with nymphal ticks and the bacteria that they may carry. It is therefore possible to suggest the alternative diagnosis of Lyme disease.

Even something as minor as a pin (which plays a prominent role in some descriptions of witchcraft) had a different appearance in the seventeenth century. Most pins and needles today are made of shiny steel, although they sometimes have the traditional brass colored designation for the eye end. This is an eons-old persistent remnant from the distant past when poor vision was not correctable. Although they are a somewhat rare archaeological find, in the seventeenth century pins were usually made of brass or they were iron with a brass head.²⁰ It is unlikely that a shiny modern pin would ever be mistaken for a tick, but what would happen if the visual image of an embedded pin closely mimicked both the appearance and colors of an attached deer tick? In 1692 it did.

A nymphal *Ixodes scapularis* tick has a decidedly brassy coloring. A female adult is bi-colored with a brass or rust colored body and a dark brown or black mouth end area with a clear circular delineation between the two. When attached, a tick is not only stuck into the flesh, it contracts its legs and assumes a pin or thorn like appearance.²¹

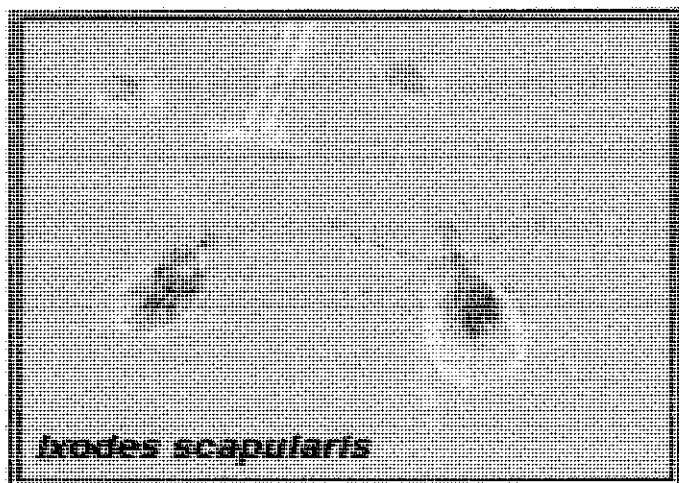


Figure 4. Ticks next to the head of a modern pin. Top: Nymphal stage. Bottom: male on the left, female on the right.

When Mercy Short lay down on a bed in Boston in 1692 and, to the horror of onlookers, pins were found sticking into her flesh,²² could they have been attached ticks? Did she come acquire *Ixodes scapularis* when she visited the jail that housed several accused witches from the Salem Village area? Were ticks inadvertently brought into Boston on the skirt hems of the accused? The floor of the jail was covered with a layer of sawdust or wood shavings. This would make an ideal microhabitat for *Ixodes scapularis* ticks. We know that Mercy reached down and picked up a handful of these wood shavings to throw at Sarah Good. Did that action bring her into closer contact with ticks? Or, did the bed she was lying on, like others in the seventeenth century, have a mattress that was stuffed with wood shavings, straw, cornhusks, eelgrass or even leaves? Was the mattress infected with bedbugs that included ticks? While the preternatural pins fit the

seventeenth century mental mindset, there is also a plausible modern explanation that the pins were attached ticks.

By examining descriptions of personal experiences written during the seventeenth century an understanding of how the first settlers in Massachusetts viewed and interacted with their environment can be formulated. It may provide valuable insights into the past that might be applicable to the present. This is especially true for Lyme disease.

I. THE AFFLICTION OF LYME DISEASE:

"When psychosis is present in an area endemic for Lyme disease it [should] be factored into any diagnosis or treatment plan."¹

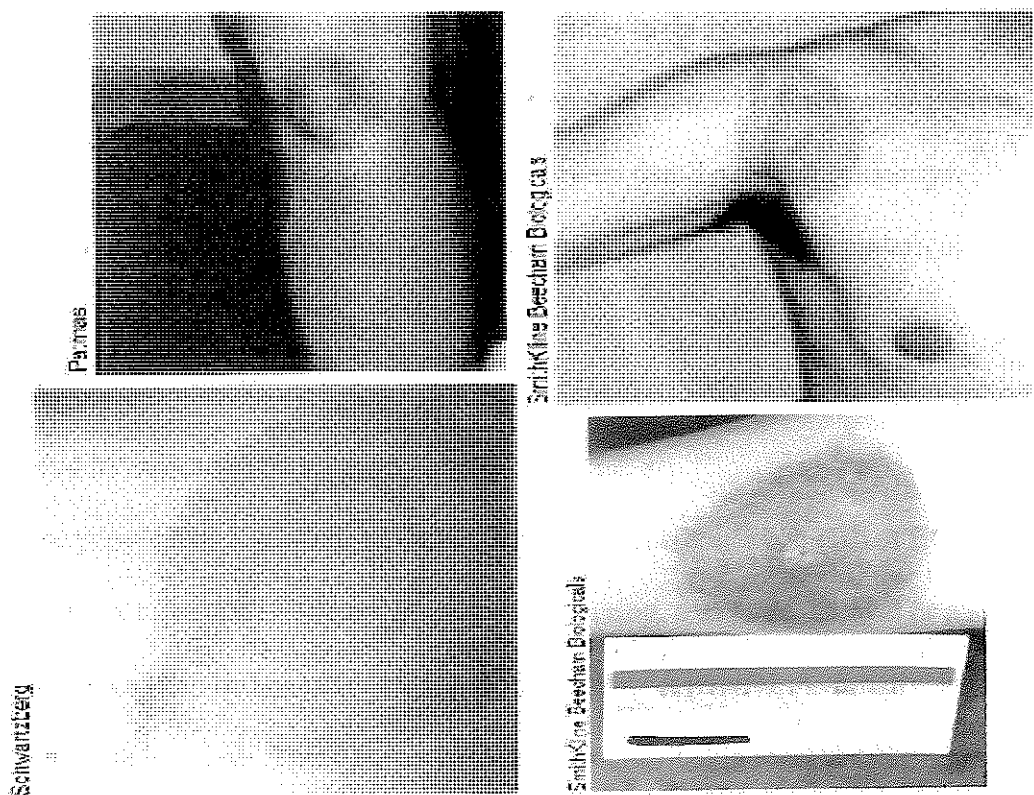
**Brian Fallon, M.D.
Columbia University**

Lyme disease is recognized by the United States Center for Disease Control as "the leading cause of vector-borne infectious illness in the United States."² Lyme disease creates a variety of symptoms and its virulence can vary from year to year and from person to person depending on environmental conditions. The experience of Lyme disease can be understood by looking at the medical and scientific literature. But it is also a very idiosyncratic condition that can be described either by the person experiencing the symptom or by an observer of the afflicted, based on their own senses. A growing body of modern source material can be found that includes descriptions of the experiences that Lyme disease victims feel. Many of these descriptions mirror information recorded during the Salem Witch Trials of 1692. My own interest in this topic was piqued when I read, for the first time, descriptions of the pinching and the sensation that needles were being pushed into the flesh of some of the afflicted. These descriptions accurately describe some of my own symptoms after contracting Lyme disease. I sometimes felt as if someone was poking a hot spike into my shoulder and my skin often crawled and pricked for no apparent reason. I contracted Lyme disease in 1994 in an endemic area, so my diagnosis came fairly quickly and my symptoms never reached an advanced stage. But I have also observed an acquaintance who suffered from Lyme-induced Bell's palsy--it is a terrifying sight even in the twentieth century when the cause is known, it must have been even more so in the 1690's.

Polly Murray contracted Lyme disease over thirty years ago. She lived in Lyme, Connecticut. She, her husband, and three of her children contracted the disease sometime before the early 1970's. Her work, in trying to find answers to what was happening within her family and her community, has been key to raising scientific and public awareness of this disease. Her book, *The Widening Circle* is an autobiographical account of her own experiences with Lyme disease. Some of the symptoms that occurred in her family also occurred in the seventeenth century.³

Another well documented modern experience with Lyme disease comes from the novelist Amy Tan, who was diagnosed with a case that had advanced to late stage symptoms. She suffered from hallucinations (at night when she was in bed like so many of the seventeenth century afflicted) which she has described. She saw a naked man approaching her bed and thought it was her husband. "It was the middle of the night," and "he wasn't saying anything or doing anything else. He was just coming toward me (before stopping) next to the bed stand, as though he was turning on the light." She "thought someone was dead. I reached for him and the image started to warp as I realized he wasn't real." The experience was terrifying. Amy Tan's other symptoms have included hair loss, fatigue, tinnitus, memory loss, olfactory hallucinations, and misspelling words when writing. When speaking, even after antibiotic treatment, she sometimes replaces words with similar sounding gibberish.⁴

Lyme disease can be accompanied by seizures. The mother of a teenager from an endemic area of New Jersey described the seizures that he developed after contracting Lyme disease. He had "episodes of tremors, wherein his body would shake and twitch uncontrollably."⁵



(Photos courtesy of Lyme Disease Foundation, www.lyme.org)

Figures 5. Examples of Lyme related *Erythema migrans* rash.

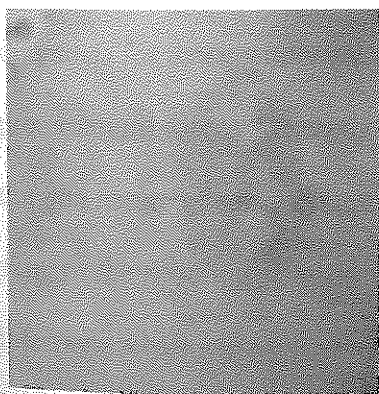


Figure 6. Example of human bite mark-it creates a jagged red spot that is clear in the middle.

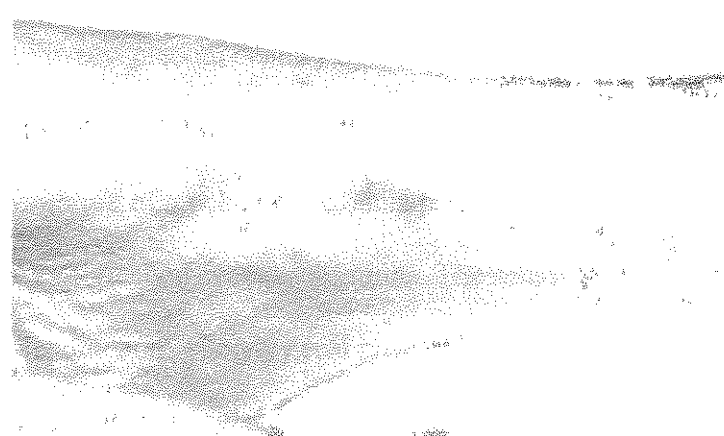


Figure 7. Photograph of scurvy hemorrhage on ankle-note red spot. *Mediscan.com*

Modern research has found that because so many body systems are involved, Lyme disease can take on a wide array of forms. People with mild infections may show no signs at all. In other people Lyme disease is so severe that it completely disrupts normal life. Lyme disease can become chronic, or it can follow a cyclical pattern of active infection, remission, and relapse. Infected women may have more risk for experiencing periods of difficult Lyme symptoms because their immune system functions are influenced by menstrual hormone changes.⁶

Some people who are bitten by ticks develop a single bull's eye rash or ECM, at the site of the tick bite. An ECM may be shaped like a ring, a triangle, an oval or a long thin ragged line and can be hot to the touch, itchy, burning or painful. Alternatively the skin at the site can be numb. There are several descriptions of skin manifestations that are described in the witchcraft testimony-ranging from a thin red line across a palm to a mark appearing that is round and resembles a bite mark made by the upper and lower teeth.⁷

It is the neurological component of Lyme disease that makes it a compelling diagnosis for those who were afflicted in the seventeenth century by what they felt were preternatural forces. When the mind itself is affected the Lyme sufferer must deal with the odd nature of some symptoms. The sensation of a poke on the back, pricking on the skin, or the shooting sensation of heat are experienced as real sensations. The mind interprets a hallucination as a very real phenomenon. The very real physical response of seizure, sweat, tension, and hormonal release can be triggered by this bacterial infection. Lyme disease can infect the brain. The relatively recent development of brain scans, especially the MRI has made the brain damage done by *Borrelia burgdorferi*

spirochetes visible to researchers for the first time. In neurological Lyme disease MRI scans have found white matter hyperintensities in the brains of approximately 15-45 % of patients. These seem to be similar to the damage seen in multiple sclerosis patients.⁶

The most common Lyme associated neurological conditions are cranial nerve palsies. Understanding these palsies is important because they may have caused many of the symptoms described or experienced in the seventeenth century as noted in the witchcraft literature. There are twelve pairs of cranial nerves that can be affected by Lyme disease and create specific symptoms. They are:

Cranial Nerve Pair I- Olfactory: afflicted may experience loss of smell or smells may be overly intense or noxious.

Cranial Nerve Pair II- Optic: Partial or total loss of vision may occur.

Cranial Nerve Pair III- Oculomotor: The eyelids may droop, the eyeball may deviate outwards, or the pupils may become dilated. Some patients may squint involuntarily or see double images.

Cranial Nerve Pair IV- Trochlear: The eyeball may rotate upwards and outwards or double vision may occur when looking down.

Cranial Nerve Pair V- Trigeminal: Pain or numbness in parts of the face, scalp, forehead, temple, jaw, eye, or teeth have been reported. The muscles used for chewing may become paralyzed or dysfunctional, making it difficult to chew, and the jaw may deviate towards the paralyzed side.

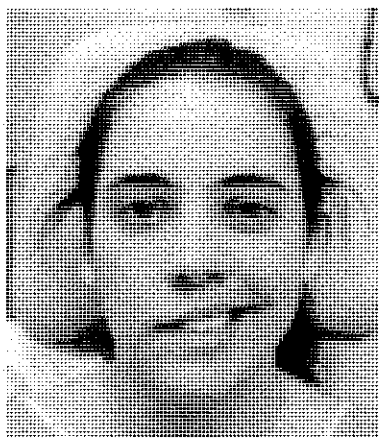
Cranial Nerve Pair VI-Abducens: The eye may deviate outwards, and excessive squinting or double vision may occur.

Cranial Nerve Pair VII-Facial: The improper functioning of these nerves can result in Bell's palsy on one or both sides of the face. Hearing loss can occur on the affected side. Problems chewing and speaking may result.

Figures 8. BELL'S PALSY, WHICH AFFECTS CRANIAL NERVE PAIR VII, IS ONE OF THE MOST COMMON NEUROLOGICAL MANIFESTATIONS OF LYME DISEASE.



Figures 8. BELL'S PALSY cont'd.



This boy developed Bell's Palsy and later died from Lyme disease. His parents feel that he contracted the disease in utero through the congenital transfer of bacteria.

All images from The Lyme Disease Foundation

Figures 9. APPEARANCE OF CRANIAL NERVE PAIR III IMPAIRMENT-ONE EYE MAY CLOSE, SKEW TO THE SIDE OR ONE PUPIL MAY BE DILATED.



All images from The Lyme Disease Foundation

Figure 10. CRANIAL NERVE PAIR XI PROBLEMS CAUSED PARALYSIS ON ONE SIDE OF THIS PATIENTS THROAT.

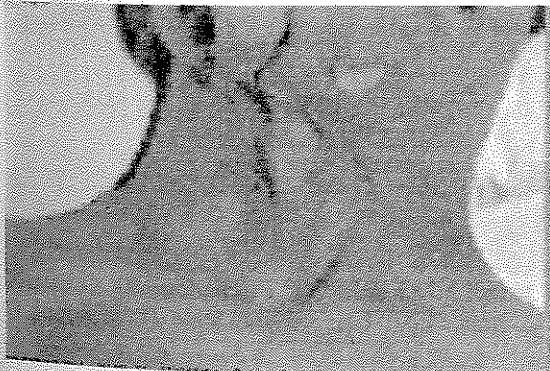
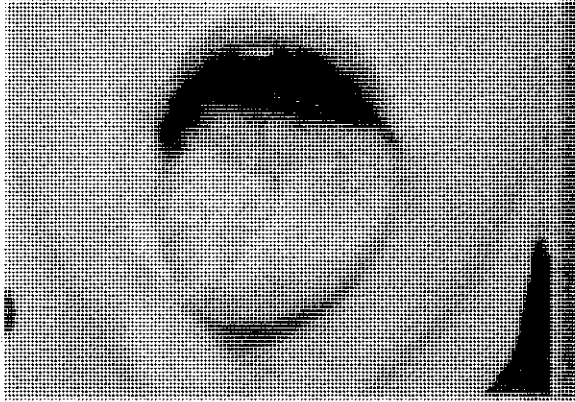


Figure 11 CRANIAL NERVE PAIR XII IMPAIRMENT CAUSES THE TONGUE TO DEViate TO ONE SIDE.



All images from The Lyme Disease Foundation

FITS AND SEIZURES.



Figure 12. Illustration of a "fit" from J.M. Charcot, Lectures on the Diseases of the Nervous System (London, 1877). Note tongue.



Figure 13. A patient having a seizure during the encephalitis outbreak in New England of the 1920's.

Cranial Nerve Pair VIII-Vestibulocochlear: hearing disturbances such as tinnitus, full or partial hearing loss can occur. Equilibrium problems can occur causing dizziness and falling down.

Cranial Nerve Pair IX-Glossopharyngeal: Problems occur with the mucus membranes in the back third of the tongue, may cause difficulty in swallowing and paralysis of the glottis.

Cranial Nerve Pair X-Glossopharyngeal: Dysfunction of the muscles in the throat, shoulders and back may cause difficulty in swallowing or talking. Drooping shoulders and an inability to rotate the head away from the dysfunctional side may also occur. Malfunctions within these branches of this nerve may cause heart problems, breathing difficulties (including slow respiration and a sense of suffocation), cough, glottis paralysis vocal cord spasms or paralysis and gastrointestinal problems.

Cranial Nerve Pair XI- Spinal Accessory: Disrupted function or paralysis of the upper back and neck. Back spasms and the inability to tilt the head to the shoulder or rotate the head in either direction can occur.

Cranial Nerve Pair XII-Hypoglossal: Affects the tongue, speech and swallowing. Can cause a deepening in the tone of the voice.

Meningitis, an inflammation of the membrane that covers the brain and, encephalomyelitis, an inflammation of the brain, can occur in late stage Lyme disease. Encephalopathy, a dysfunction of the brain itself, can also occur. Cognitive problems and mood disturbances such as irritability, bursts of crying, and temper flares occur. Profound fatigue, confusion and difficulty sleeping are reported. Depression, mood changes, hallucinations, panic attacks, Tourette syndrome, verbal aggressiveness, extreme agitation, inappropriate and manic behavior and psychosis have been reported. Some patients have attempted suicide.⁹ All of these symptoms can be found to be described in the witch trial transcripts.

During the Salem witch trials oral testimony was written down, often by Rev. Parris.¹⁰ These records provide a rare opportunity to study the afflictions suffered by members of a community from the past. Those who are usually- because of time, space, gender, the accident of record preservation, and insignificant social standing- rendered inarticulate are instead given loud voices. Although records are fragmentary at best, Salem Village of 1692 remains one of the most articulate communities from America's collective past. Each person called to testify at the trials is given a forum to describe the world from his or her own point of view.

Everything from the mundane acts of yoking a pig or conversing with a neighbor, to the extraordinary sensation of flying over the tree tops in the dead of night, or the amazement at the combustibility of a mare's flatulence are included. A set of beliefs emerges that firmly include the existence of witchcraft as a cultural construction-- a way to understand aberrant behavior, disease, and death.

Modern scholars have also, to a large extent, relegated the "afflictions" of 1692 to an imaginary world somewhere between the psychiatric ward of history and a special hell reserved for liars, belittling the "girls'" afflictions, judging their veracity, and preferring to concentrate on more important parts of the story. There are several excellent books written about the Salem community. Some pre-date the modern discovery of Lyme disease. John Demos wrote a description of Salem's place in Puritan social history.¹¹ Stephen Nissenbaum and Paul Boyer's *Salem Possessed* outlined the social and political factions of Salem.¹² Mary Beth Norton's *In the Devil's Snare* added an Indian Wars post traumatic stress psychological evaluation to the body of work.¹³ But in only a few instances has any attempt been made to analyze the "afflictions" of the accusers from Salem Village as anything other than imaginary.

In 1976, Linda Caporeal, in an article titled "Ergotism: The Satan Loosed in Salem," was the first to offer an analysis of the Salem Witch Trials where the "afflicted" were treated as patients who suffered from real symptoms. Concentrating on the hallucinatory nature of some of the symptoms, her analysis included a flawed environmental study of the Salem Village in 1692. She fails to inventory the full list of afflicted from the records and connect them to potential sources of ergot- a fungus that infects rye plants. She also does not explain why some members of a household eating

from the same supply of flour remain symptom free while others become hallucinogenic. She presents what she calls a circumstantial case for the "afflicted" girls suffering from ergot ingestion. While dubious, this theory has worked its way into Salem's modern oral history of its own past. It is included in the tour script at several of the historic sites in town and was the basis for a Halloween special on television.¹⁴

In 1999, Laurie Winn Carlson published *A Fever in Salem: A New Interpretation of the New England Witch Trials*. She proposed Encephalitis Lethargica, caused by bites of mosquitoes as a possible source of the "afflictions."¹⁵ Another scientific paper has suggested that the girls suffered from some form of group schizophrenia. Group hysteria has been suggested.¹⁶

The afflictions described in the witchcraft testimony were the central catalyst for this historic episode, and as such, need to be taken seriously. The idea that the afflicted were actually sick has been given short shrift by modern scholarship. Mary Beth Norton, for example, states in her book that her research into this area was limited to a conversation with a "renowned" physician who said it couldn't possibly be encephalitis!¹⁷

A very long list of people who were living very real lives in 1692, however, lay out an all too real tale of actual human suffering. Wives lose husbands. Husbands watch their wives descend into madness or die. Children convulse and animals behave strangely. Life itself is turned upside down in some households. The root cause for some of this suffering may have been lurking in the ticks that lived in the Salem landscape. Many English cultural practices in the seventeenth century would have been conducive to contact with ticks and exposure to Lyme disease.

English Cultural Practice or Concepts-----> RESULT

High Carbohydrate/ Low Protein Diet

Switching to high protein/low carbohydrate diet creates fluctuation in blood insulin levels- called "starving" in Plymouth and Jamestown

Use of Matchlock, Not Deer Hunters, Preference for Beef

LR High deer population=High tick population

Scurvy is a disease caused by a lack of fresh foods and carbohydrates in the diet that causes joint pain, fatigue and spots on the skin-it was brought to England by the Normans

Lyme-like symptoms diagnosed as scurvy

Use of pins made of brass or iron with brass heads

A pin is the same color as an attached *Ixodes scapularis* tick

Burning leaves and undergrowth in the winter and early spring

Excellent tick control

Using straw, eel grass, corn husks and leaves to stuff mattress ticks, problems with bedbugs, use of same word for both the bed case and the bug- possibly derivative

LR Potential for bringing ticks directly into home

Women and children wear long skirts that brush into vegetation

LR Drag collecting of ticks

Pulling heavy outer skirt over head and shoulders for warmth or protection from rain (This practice is described in witchcraft trial testimony against Susanna Martin.)

LR Transfers ticks to the neck/head area which creates higher risk for neurological Lyme symptoms. More likely to occur on chilly fall and Spring days when questing ticks are active

PANNAGE-pigs eat acorns in Fall

Controls ticks by removing food for deer, mice and bird blood meal hosts.

English Cultural Practice or Concepts-----> RESULT

Pasturing Cattle in 'the woods'

LR Requires trek through ecotone on regular basis

Fragmenting Forest, retention of woodlots and windrows, planting orchards

LR Conducive to high tick population, increases risk for Lyme disease, deer attracted to fruit trees in Fall.

Wood collection for cooking and heating

LR Humans in tick habitat

Dogs and Cats as pets

LR Ticks brought into closer contact with humans, dogs kill wolves.

Military Service causes disability of Rheumatism

LR Specifically links intensive interaction with forest and ecotones to disease

Transportation by water

LR Riparian corridors may be tick habitat in cleared areas

Use wool for clothing, Dye cloth various colors

LR Brings humans into close contact with tick blood meal hosts and tick inhabited fleece. Natural materials used as dyestuffs may come from forest or ecotone vegetation

Autumn Slaughter

LR Brings humans into close contact with animals during time of high level of questing Adult *Ixodes scapularis* ticks

Pigs confined to sty or fenced in

LR Deer not driven away, humans in forest collecting acorns at high Adult *Ixodes scapularis* time

English Cultural Practice or Concepts-----> RESULT

Observing skin for preternatural marks

LR Makes skin manifestations of round rash and sucked on area (witches teat) part of witchcraft etiology. Interestingly, creates a long list of marks observed on the skin of the afflicted that could be ECM's

Sensations of heat or pricking of skin are caused by preternatural forces

LR Logically links neurological symptoms to heat of hell and pins

Under the evil hand, Behagged, and Bewitchment as medical diagnoses

LR Gives medical validation to afflictions while freeing practitioner from responsibility for cure, makes neurological symptoms a component of bewitchment

Hallucinations are real but 'otherworldly' events

LR Allows neurological symptom to admitted as spectral evidence in court testimony

Witchcraft is a capital crime punishable by death

Facilitates and validates prosecution and execution of suspected witches for causing physical afflictions

Because New England is an area that is now endemic for this illness, Lyme disease should be considered as an additional diagnosis for the suffering of the humans and animals that were afflicted in the seventeenth century. This should in no way be taken to denigrate or deny work done by previous researchers and authors. While historians sometimes find solace in simplification, the real world is a complex place. Someone in 1692 could have been depressed and traumatized, have swilled down tainted rye products, and suffered from encephalitis at the same time. But there are too many similarities between the symptoms that are described in the seventeenth century court records and those found in the very real experiences of modern people with Lyme disease to ignore.

Lyme disease is a challenge for both doctors and patients because it can be difficult to diagnose and hard to treat and can have frightening symptoms. Consider James Carr, the rejected suitor who visited a physician in 1672 with a set of aggravating physical symptoms. The diagnosis that he received was that he was behagged.¹⁶ This was a scary diagnosis that included no known medical cure and the very real threat of death. Consider Amy Tan, the modern novelist, whose early diagnosis for the cause of her Lyme disease symptoms included amyotrophic lateral sclerosis, better known as Lou Gehrig's disease. This terrifying diagnosis included little medical help and was almost certainly a death sentence. Fifty thousand dollars and numerous tests later she received a proper diagnosis of late stage Lyme disease.¹⁷

Polly Murray of Lyme, Connecticut, was part of an "afflicted" group during the last three decades of the twentieth century. She worked diligently to attract the "eye" of modern science for those who were suffering in her community. One of modern science's

first answers for her problems was a stint in the Psychiatric Ward at Boston General Hospital. Seventeenth-century diagnoses like "behagged" had somehow been transformed over time into *all in your head* with the same accompanying measure of loss of control over one's life. James Carr probably died convinced of the fact that he had been bewitched. Polly Murray has lived to see her "afflictions" defined as Lyme disease and is an articulate and effective advocate for its modern victims.²⁰ Ticks, bacteria and blood have been a potent combination in New England for centuries.

Lyme disease is usually spread by the bite of ticks of the genus *Ixodes* that are infected with the *Borrelia burgdorferi* bacteria. For Lyme disease to exist in an area, at least three closely interrelated elements must be present: the bacteria, the ticks that can transmit the bacteria, and mammals (such as mice, deer, and humans) or birds that provide blood meals for the ticks through their various life stages.

Ixodes ticks are usually found in temperate regions with high relative humidity at ground level. In New England, *Ixodes scapularis* ticks are associated with deciduous forest and habitat containing leaf litter. Leaf litter provides a moist cover that protects from wind, snow, and other elements. *Ixodes scapularis* ticks are generally found in heavily wooded areas that edge onto tracts of cleared land--the transitional ecotone that is also favored by white tailed deer. In areas where most of the land is cleared, ticks may be limited to riparian areas that are regularly visited by thirsty host mammals.

Knowing the complex life cycle of *Ixodes* ticks is important in understanding the risk for humans acquiring Lyme disease. *Ixodes scapularis* has a two year life cycle. Adult female ticks lay eggs on the ground in early spring. By summer the eggs hatch into larvae. Larvae feed on mice, other small mammals, deer, and birds in the late summer

and early fall, molt into nymphs and are then inactive until the next spring. Nymphs feed on rodents, small mammals, birds, and humans in the late spring and summer and then molt into adults in the fall. In the fall and spring, adults feed and mate on large mammals (preferably deer) and bite humans. The key role that deer play in this cycle is that they can nourish a large number of ticks, assuring population stability. Deer have an antibody to *Borrelia burgdorferi* bacteria in their blood (their health is not affected by the spirochete) which may act somewhat prophylactically to eliminate spirochetes in adult ticks during their last blood meal. The adult female ticks then drop off and lay eggs in the spring which completes the two year life cycle. The *Borrelia burgdorferi* bacteria does not appear to be passed on by a mother tick to her eggs: all larvae hatch bacteria free and are infected only after contact with an infected blood meal host.

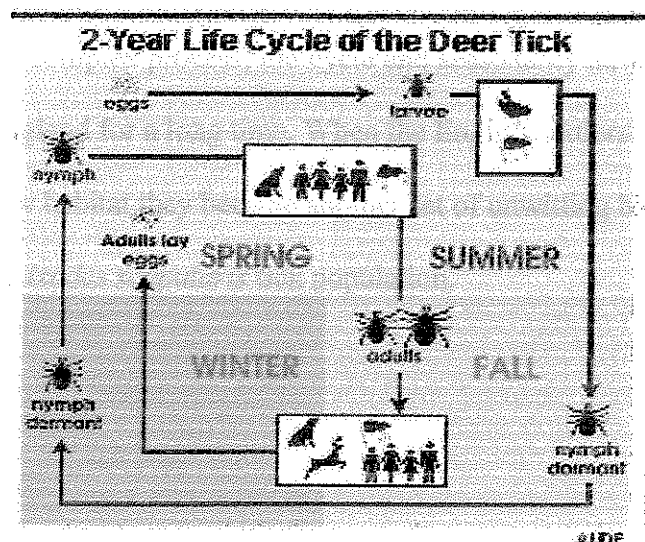


Figure 15. Ticks have three chances in their life cycle to acquire *Borrelia burgdorferi* bacteria. Chart courtesy of The American Lyme Disease Foundation.

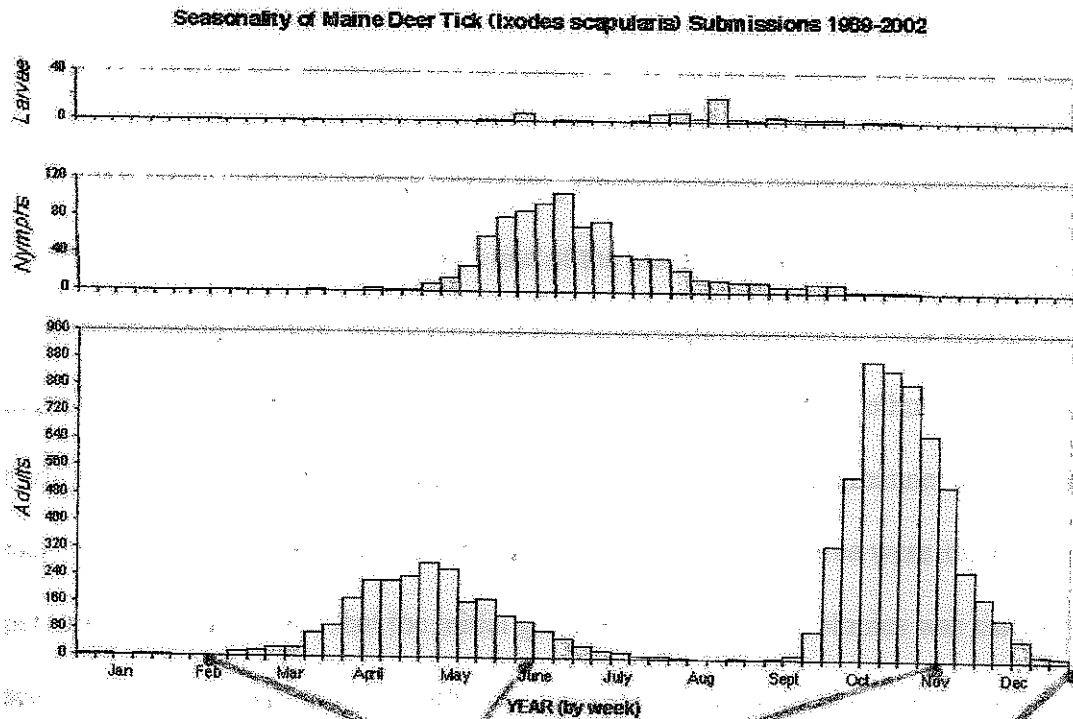
Ticks, birds, rodents, and some other animals all serve as natural reservoirs for the *Borrelia burgdorferi* bacteria. The bacteria can usually live and grow within these

hosts without causing them to die. Tick larvae and nymphs generally become infected with the Lyme spirochete when they feed on small animals and birds that carry the bacteria. Once infected, the bacteria remains in the tick for the rest of its life. Infected ticks bite and transmit *Borrelia burgdorferi* to rodents, birds and other mammals in the course of their normal feeding behavior.

Ticks search (or quest) for host animals from the tips of grasses, shrubs, and leaf litter and transfer to animals or people that brush against this vegetation. Ticks can only crawl. They do not fly. Ticks can attach to any part of the body but will often crawl to a moist hidden area like the groin, armpit, or where clothing is tight and where they are hidden. They feed on blood by first applying a sort of secreted anesthetic to the host's skin and then attaching their mouths into the skin itself. Their bodies slowly enlarge with blood as they become engorged over several days.²¹

This complex process, life cycle and seasonality have probably been occurring in New England for a long time. When the English settlers landed and interacted with their new habitat they became another set of unwitting blood meal hosts for the pre-existing *Ixodes scapularis* tick population.

Figure 16.



WHEN APPLIED TO THE SEVENTEENTH CENTURY:

Arrival of Mayflower

The Winthrop Fleet Arrives

The *Lyon* arrives with supplies and lemon juice-were people cured by the lemons or was it because ticks had become dormant?

Groups move from Massachusetts to Connecticut-June, September and November.

'Afflicted girls' in Salem begin to show symptoms

II. ENGLISH SETTLEMENT IN MASSACHUSETTS:

Pease porridge hot, pease porridge cold,
Pease porridge in a pot nine days old.
Some like it hot, some like it cold,
Some like it in a pot nine days old.¹

Old English Nursery Rhyme

The story of the founding of the English colonies in the New World during the first half of the seventeenth century is one of the many 'histories' that make up a larger mythic American identity. Many parts of this foundation myth have been analyzed, questioned, embellished, interpreted and re-interpreted for over three hundred years. Two parts of the story that have been passed down in a relatively intact manner are the 'facts' that the first settlers at Plymouth and in the Massachusetts Bay area were subject to periods of starvation and that the initial great sickness in both colonies was caused by scurvy. This study will analyze both the deeply rooted cultural subjectiveness of seventeenth-century English food preferences and the actual diet that was being eaten in this early time period, as described by the settlers themselves. Since scurvy is now known to be caused by a deficiency of Vitamin C in the diet, any possible sources of this nutrient will be examined. It will also propose Lyme disease, contracted after contact with a 'geography of risk' for this ailment, as an alternate diagnosis for the root cause of the initial period of illness in both Plymouth and Massachusetts Bay and as the cause of sporadic outbreaks of illness throughout the seventeenth century, and as a possible cause of the afflictions of the deponents in the Salem Witchcraft Trials of 1692. It follows a trail of evidence that begins with food.

Exactly what is considered to be food can be a highly subjective, culturally determined concept. The first settlers to come to Massachusetts brought with them a set of cultural attitudes related to both food and foodways. The traditional diet of the seventeenth-century middle class yeoman in England was based largely on legumes and grains. Studies of records from medieval archaeological sites show a high level of cereal in the diet.² A typical meal was porridge or pottage, a thick soupy stew made up of a grain (usually oats) or legume base with some onion or other vegetables mixed in along with the occasional chunk of expensive beef or pork, with a hunk of coarse bread on the side. Beer was brewed from barley and hops. Add in some dairy products made from cow, goat, or sheep's milk and a meal was complete. Bread was endorsed in biblical terms as the "staff of life." Christ himself beseeched God to "give us this day our daily bread." This diet created a grain-centric cultural mindset that was carried within each English settler who stepped foot ashore in North America.³

Shipboard food mimicked the land diet as closely as it could but was definitely high in salty preserved meat and lacking in the fresh fruits and vegetables that would have been available on land. The lack of Vitamin C in this diet made for a prevalence of shipboard scurvy, especially during long journeys. It takes three months of vitamin C deprivation for scurvy to set in. Scurvy is a disease that is characterized in later stages by mouth sores and hemorrhages (mucosa hemorrhage), loose teeth, extreme fatigue and prostration, and ecchymoses--a form of macula (spot, blemish or stain) appearing in large irregular shaped hemorrhagic areas of the skin. It is preceded by a period of ill health characterized by sallow complexion, loss of energy, and pains in the extremities and joints.⁴ Scurvy haunted ships crossing between England and early Massachusetts and was

a definite concern for travelers. Even in the seventeenth century, scurvy was considered to be somehow connected to a poor diet. Several remedies for this side effect to cross-Atlantic travel were included in the prevailing English body of common knowledge. These included lemon juice, and teas made from lemon grass, sassafras, and rose hips. All are sources of Vitamin C except the watered lemon grass.⁵

Both Edward Winslow and William Bradford state that the occupants of the *Mayflower* began to become ill soon after they landed at Cape Cod. Winslow states that they suffered from "coughs and colds, the weather proving suddenly cold and stormy, which afterwards turned to the scurvy."⁶ Bradford wrote that half of his company died "being infected with the scurvy and other diseases which this long voyage and their inaccommodate condition had brought upon them." Bradford connects the illness with both poor living conditions and a lack of proper food and drink (beer). More than one third of the early chapters of Bradford's *Of Plymouth Plantation* revolve around attempts to acquire or grow food, especially grain.⁷

In Europe at any time during the Middle Ages the primary determining factor for diet was class. The laws instituted in England after the Norman Conquest in 1066 reserved the meat of wild mammals like boar, rabbits and deer for the aristocracy. Because of this higher classes ate higher percentages of meat than lower classes. One study of account records from 1338 shows meat and fish making up fifty eight percent and bread twenty six percent of the diet of an upper class household, while meat made up twelve percent and bread and grains sixty four percent of the diet for the lower working class. If these percentages held through time, the middle class seventeenth century

English settlers would have been accustomed to eating a diet with a similar high percentage of grain based foods.⁸

Once on shore in New England, the Pilgrims immediately identified and accepted maize as an edible grain, renaming it Indian corn after the common corn (wheat) of Europe and the Native Americans whose caches they had looted. The rest of their initial diet revolved around this grain, allowing the settlers to retain an acceptable English grain-centric food model with little alteration. Over time they substituted a new world bean, calling it a pea bean, for the 'pease' in their porridge, to create the now classic New England Baked Beans. Again an indigenous food substitute was renamed to occupy an already accepted role in the colonists' diet.⁹

Any understanding of the concept of having a poor diet and hard or starving times, as described by the first English settlers, needs to be examined with this cultural bias and mindset included in the interpretation. Starving times can more accurately be described as 'lacking grain times.' The English would go to great lengths to obtain grain based food. Thirteen years earlier at the settlement in Jamestown, Virginia, a lack of grain made those desperate Englishmen vulnerable to "sailors who would pilfer biscuits to sell, give, or exchange (with the settlers) for money, furs, sassafras, or love."¹⁰ In Plymouth, William Bradford, greeting a ship with seven new mouths to feed, bemoaned the lack of anything to make bread. However, at the same time (Spring 1622), the same ship, the *Sparrow*, laden with a cargo of fish but "no victuals nor any hope of any," was allowed to leave Plymouth to sell the fish in Virginia. Apparently fish were considered to be barely fit for an English stomach.¹¹ (Chemical analysis of the composition of ancient human bones from 183 early Britons show that this preference for grains [and land

animals] over seafood began, even in the coastal areas of England, over 6,000 years ago¹² and was probably well engrained in the English psyche by the seventeenth century.)

At another point in time Bradford sent Edward Winslow to Monhegan Island, off the coast of Maine, to procure what provisions he could from the English ships that were fishing in the area. He returned with a store of meal that they could spare (but not fish).¹³ The Pilgrims traded with the Native Americans for corn. When a Native woman offered two traveling Pilgrims a platter of corn and dried oysters, they accepted the corn but gave away the oysters to their Native traveling companions.¹⁴

The English considered fish a "difficult" food because it was hard to cook, since it tended to fall apart and was full of bones. And then it had to be eaten. This was difficult when the only utensils available were knives, spoons and fingers. Fine as a trading commodity, its proper place was on someone else's plate. The food of penance in pre-reformation England, fish continued to carry this stigma into the seventeenth century. It was reluctantly eaten, replaced by other foods when they became more available, and would not reappear with any frequency until the early nineteenth century.¹⁵ This is clearly delineated to have happened in the Massachusetts Bay settlement. Archaeological research done at a seventeenth-century Boston privy (Cross Street 4) shows that fish accounted for 6.9 percent of the total consumed bio-mass (food from animal sources) in the earliest levels (+/- 1650) but dwindled erratically down to 0.6 percent by the dawn of the eighteenth century.¹⁶

The almost homogenous middle class status of those first settlers in Massachusetts (in contrast to Jamestown's fifty percent population of upper class "gentlemen" settlers)¹⁷ made their initial eating of fish and 'wild' meats a marked

departure from the normal diet they had eaten in England. A normal diet would be once again assumed when more grain and domesticated animals became available in the New World.

The hunting of deer, for example, was not only illegal for the lower classes but was seen as the exclusive sport of a decadent upper leisure class in England. Because of their class status the settlers had not participated in this pastime in England. And because the Pilgrims and Puritans had not been deer hunters back in England, these first settlers were probably without much cultural interest in hunting them in the New World.¹⁸ Although John Winthrop's first meal in the New World was "a good venyson pastye,"¹⁹ he probably considered any consumption of wild flesh to be a temporary stop-gap measure needed to fill the void caused by a shortage of domesticated cattle. This shortage was expressed in 1630 when Winthrop wrote to his wife "we are here in paradise, though we have not beef or mutton."²⁰

In the archaeological work done in association with Boston's Big Dig the food remains in several seventeenth-century privies were examined. They show that venison was an extremely rare item in the prevailing foodways of Boston between 1650 and 1700. Out of hundreds of faunal remains found, deer bones turn up only three times, including once in the Katherine Nanny privy and again in a privy belonging to a wealthy pewterer.²¹

This lack of interest in shooting deer was clearly shown by the actions of the set of armed Englishmen who made one of the first exploratory trips on Cape Cod in 1620. They were properly armed (with fowling pieces and a spaniel bird-hunting dog) to hunt (and did eat) fowl during their trek. When they saw three deer, Edward Winslow wrote

that he'd like to have taken one of them, but no one actually fired a shot. Perhaps their muskets were too heavy and awkward to quickly aim and fire. Maybe their fowling pieces had shot that was too light to kill a deer. Another explanation might be that they had little inclination to shoot because they were simply not culturally molded to be deer hunters. They were not technically on a food procurement foray. However, when they found a stored cache of Indian corn nothing kept them from taking it. To a grain-centric mind the deer with its wild meat could easily be ignored, but the corn was much too tempting to leave behind.²²

Hard times in Plymouth Plantation came whenever grain was in short supply or gone. There was, however, a seemingly adequate supply of other, less desirable, foodstuffs that actually created a nutritionally adequate diet. The one small boat that Plymouth had, while not "over-well fitted," was sent out net fishing for bass and other fish. If the boat was late coming back or had a poor catch, "all went seeking shellfish," which they "dugged out of the sands" at low tide. In the winter they would gather ground nuts (Jerusalem artichokes) and, of necessity, shoot fowl and deer, although they appear to have obtained most of their venison from their Native neighbors.²³

On the arrival of the ship *Anne* in 1623, Bradford stated, "for food they [the settlers at Plymouth] were all alike, save some that had got a few pease of the ship that was last here. The best dish we could present our friends with was a lobster or a piece of fish without bread or anything else but a cup of spring water. And the long continuance of this diet, and their labors abroad, had abated the freshness of their former complexion; but God gave them health and strength in good measure, and showed them the truth of that word that man liveth not by bread only." He continued, "God fed them out of the sea

for the most part.”²⁴ In other words, in their “sad state” they were tanned, healthy and strong, but had by necessity, and not choice, a diet that depended upon seafood of all types and lacked bread. Even worse, there was no beer! To a grain-centric mind this was as close as it could get to starving. Considering the “sad state” Bradford felt their diet was in, it is easy to understand why he felt that they were suffering from scurvy! (Although skin manifestations are never mentioned anywhere in the historic records for this contact period, a comparison between photographs of the skin hemorrhages that are a symptom of scurvy and the ECM of Lyme disease shows great similarities in appearance).²⁵

Looking at this diet with twenty first century eyes, it appears to be an early high protein- low carbohydrate diet that might be admired by the likes of the late Dr. Atkins, washed down with lots of pure free spring water (we now buy it by the bottle). Transition to this diet would have initially been accompanied by the very real physical phenomenon know as “carbohydrate craving,” caused by a drop in blood insulin levels (the induction phase in the Atkins’ diet), but even those pangs would diminish over time as the body became accustomed to a lowered insulin response.²⁶ After the great sickness of the first winter, and except for normal fevers and such, the residents of Plymouth seemed to remain remarkably healthy and many lived long lives. This may have been one of the most direct results of their healthy diet.

Bradford himself questioned the depth of Jacob’s Biblical famine. “They had such great herds and store of cattle of sundry kinds, which, besides flesh, must needs produce other food as milk, butter and cheese, etc. and yet it was counted a sore affliction.” His poor colony of Plymouth “not only wanted the staff of bread but all these things [meat and dairy products]” and were stuck eating seafood. Like any other English settler,

Bradford must have longed for the homey comfort of a bit of beef and a slab of bread slathered with a thick slice of English cheese washed down with a nice warm beer.²⁷

In turn, twenty-first century eyes can question the depth of Bradford's sore afflictions. Surely if his people were starving, he, as Governor, would not have allowed even the pesky adventurer Thomas Weston to send a ship full of edible fish to Virginia when it was needed to feed people at home. In actuality, the food consumed during the first years of settlement, as described by William Bradford, Edward Winslow and others, shows a healthy nutrient level, including more than adequate amounts of vitamin C. It is unlikely that they had a vitamin C deficiency.

The early settlement period in what is now the state of Massachusetts was marked by an initial phase of dramatically high mortality rates followed by a more normal and predictable cycle of disease and mortality. At Plymouth, after a healthy eight week voyage across the Atlantic that included only two deaths, a period called the "great sickness" and "starving times" began- the mortality rate for the first eight months of settlement was at almost fifty percent. This figure includes one human spontaneous abortion that occurred about six weeks after landing was made on Cape Cod.²⁸ Eight years later, history seemed to repeat itself when the Puritan settlers landed nearby in what is now Salem, Massachusetts. The landscape they encountered would have been very similar to one that the Pilgrims had encountered earlier-- a well oaked forest, abounding with deer, interspersed with some stretches of cleared land that had been depopulated by the waves of sickness that had decimated the local Native American tribes. Once again cleared land at the edge of the forest had accumulated several years' worth of shrubby growth. Once again there would have been a decrease in the seasonal burning of

undergrowth. Once again the settlers became very, very sick. Although a handful of stray settlers predated them, the Massachusetts Bay colony was to a large degree initially settled by two fleets. It is estimated that eighty colonists out of the two hundred from the first 1628-29 Higginson fleet died and many became sick. More than two hundred out of nine hundred colonists from the second 1630 Winthrop fleet died, and one hundred went back to England where most of them died anyway, making for a +/- thirty three percent initial death rate.²⁹

At Plymouth the mortality was blamed on cold weather, catching cold, poor living conditions, poor diet and scurvy.³⁰ In Salem, Boston, and Charlestown it was blamed on hot weather, fever, the mental defect of "pining for home in England," poor living conditions, poor diet and scurvy.³¹ When population groups went to Connecticut they also seem to have once again encountered an initial phase of sickness. It also infected their cattle, causing malaise and spontaneous abortion.³²

The diagnosis put forward by William Bradford, Edward Winslow, Plymouth physician Samuel Fuller, and John Winthrop in their writings is that the initial sickness in Plymouth and Massachusetts Bay was scurvy caused by a diet that lacked grains and other proper food. Some of the symptoms pointing to this were: lameness, joint pain, and extreme fatigue to the point of not being able to get out of bed. This story was repeated by William Wood who wrote "whereas many died at the beginning of the plantations it was not because the county was unhealthful but because their bodies were corrupted by sea diet." Because of the great sickness, John Winthrop sent a ship back to England to bring over an emergency supply of proper English food and lemon juice (a known cure for scurvy). The level of scurvy does seem to have abated by the time the ship returned in

February of 1631. Credit was given to the lemon juice, not the time of year (ticks are not active in the coldest part of the winter), for causing a cessation of new cases of 'scurvy'.³⁵ At face value, on arrival after a long period at sea, some passengers may have had a deficiency of vitamin C and developed scurvy. This self-diagnosis becomes suspect, however, because once they arrived on land the settlers immediately began to supplement their shipboard diet with indigenous foods and mention doing so. Clams, mussels, oysters, wild onions and leeks, sassafras, and rose (hips) are among the foods mentioned.³⁴ A diet that included even some of these food items would actually have been extremely rich in vitamin C.

Clams, which were described as "being at their doorstep,"³⁵ would be an excellent source of Vitamin C. A modern size clam contains 8.84 mg of vitamin C.³⁶ To mentally picture this; three modern soft-shell clams (*mya arenaria*) contain vitamin C equal to the amount found in one lime. But shellfish were described as being much larger then than they are today. Clams were "as big as a penny white loaf" of bread. Oysters were "great ones in the form of a shoe horn," some "a foot long."³⁷ Frances Higginson described oysters at Salem that were ten inches wide.³⁸ This larger size at contact is supported by archaeological evidence.³⁹ If a small modern oyster contains 1.78 mg⁴⁰ a ten inch oyster might outstrip even a lime for vitamin C content.

Onions may still be a common ingredient in many recipes today because, in addition to adding taste, they add vitamin C. Wild onions and leeks were described as being available even in the winter (in 2003, for example, they were still found in edible condition growing on Cape Cod on November 12).⁴¹ When this ingredient was added to

pottage for taste, it made it into an excellent source of vitamin C. Wild onions contain 38 mg in every ounce. Wild leeks weigh in at 80 mg for every 3.5 ounces.⁴²

The modern recommended daily adult allowance of vitamin C is 60 mg.⁴³ This is a fairly general number meant to cover the nutritional needs of a wide spectrum of body weights. The number for seventeenth-century adults can only be estimated but even a small amount of some of the foods they mention eating would have provided an adequate daily allowance for vitamin C.

While they certainly believed that they had scurvy themselves, the scurvy diagnosis for the Winthrop fleet in Salem and Boston is even more suspect, because of the time of year and one of the first activities they mention doing after landing. They arrived on June twelfth and immediately picked (and presumably ate) strawberries. These strawberries were described as "in abundance, very large ones, some being two inches about," so prolific that "one may gather half a bushel in a forenoon."⁴⁴ A strawberry is a vitamin C powerhouse with a whopping 86.18 mg per cup.⁴⁵ Again the scurvy diagnosis does not match with the facts. It is also difficult to believe that in a countryside full of sassafras (a known anti-scorbutic in the seventeenth century) that was so plentiful that they were exporting it back to England, they would not have used it to help cure their own illnesses. The general population was certainly receptive to ingesting medicinal fluids to help their disorder. Nicholas Knopp, for example, was found guilty after he was brought before the Massachusetts General Court in 1631 for taking it upon himself to cure the scurvy with a water of no worth nor value, which he sold at a very dear rate. It is very likely that the sick settlers tried many "cures" but that they didn't work.⁴⁶

III. WAS IT LYME DISEASE?

"The pathogens were always here, but we had no vectors that fit both the reservoir and the people. That was accomplished when the deer herds came back."¹

Andrew Spielman M.D.

Harvard University School of Public Health

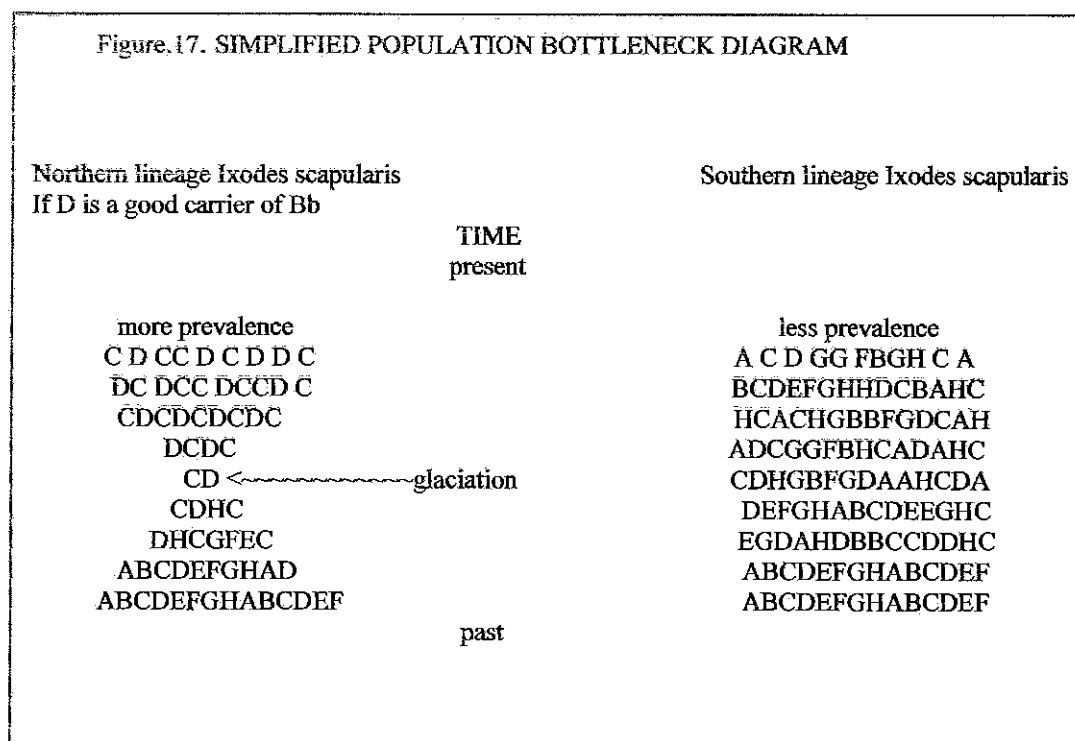
Nicholas Knopp may have been an innocent man. He may have been offering an effective elixir full of vitamin C that would have cured even the scurviest of victims. But it didn't work because the settlers did not suffer from a lack of vitamin C. The colonists suffered from an ailment that had nothing to do with their diet. What they really may have needed was an antibiotic that would cure the bacterial infection of Lyme disease. Just as many descendants of those first English settlers still inhabit the New England landscape, there are other descendants of other organisms from the seventeenth century that still live here today, including white tailed deer, ticks and even bacteria.

Like so many other features of New England's landscape, the prevalence of Lyme disease is directly influenced by both glaciation and the 'hand of man.' Glaciation related population fluctuations affected both the *Borrelia burgdorferi* (hereafter Bb) bacteria and the *Ixodes scapularis* tick vector on a genetic basis. High prevalence rates for Lyme disease skirt northern latitudes on a world wide basis in areas with deciduous mixed forests that usually have a high percentage of oak trees. These are areas that were influenced by the last glaciers from between 18,000 and 15,000 years ago. It has long been observed that species diversity of many animals and plants decreases with higher latitude and glaciation may have played an important role in this phenomenon.²

Because of its importance as a pathogen of humans and animals and the value of complete genome sequence information for understanding its life cycle, the *Bb* bacteria were subjected to a gene sequencing study in the late 1990's. It contains a linear chromosome of 910,725 base pairs.³ Studies have found a relative genetic homogeneity among *Bb* bacteria species in northern sections of North America which is indicative of an evolutionary history of recent (15,000 years or so) population growth and rapid geographic range expansion for this species.⁴

In an important genetic study of the *Ixodes scapularis* tick in North America substantial differences were found in the genetic structure and evolutionary history between ticks found in northern areas and those found in the inland southern areas. This underscores the ongoing debate about whether both sets of ticks belong to the same species. Although they are taxonomically now described as a single species, two distinct mitochondrial lineages have been identified within the species. One lineage, the "Southern clade," is common in the south, and another, the "American clade," is common in the north (and also occasionally shows up in the mixed genetics of coastal southern area ticks, probably brought down by migrating birds). These patterns of genetic composition suggest that the northern and southern populations have separate and different evolutionary demographic histories. The northern tick population showed evidence of exponential increases in effective population size. This population explosion after a period of extreme contraction is sometimes described as an 'evolutionary bottleneck'. The contraction was caused by temperature stress and die off during glaciation. Corresponding genetic patterns of southern population samples showed a long evolutionary history of constant and stable population size.⁵

The localization of Lyme disease in the range of the northern lineage of *Ixodes scapularis* suggests that this genetic factor may play an important role in the concentration of this disease in the north. The low rate of Bb infection in southern *Ixodes scapularis* could be the result of the diverse genetic heterogeneity of southern ticks.



A study of a forest/tundra ecotone in modern Colorado found that tree establishment is correlated with climate change. Warm, wet conditions caused the conifer/tundra ecotone to advance into the tundra⁶. The glaciations of North America created a slowly moving set of temperature related transitional zones, or ecotones, over time. In 18,000 BP, Massachusetts was tundra at the edge of a glacier. This, in turn, edged a larger swath of conifer dominated zones that covered most of the eastern seaboard, the Midwest, and the upper southern area. A temperate/conifer mixed woodland was restricted to two zones on what is now the southern Gulf Coast.⁷

The genetic consequence of tick population contraction during the glacial maxima and population expansion during the intervening warming periods for northern ticks was morphological change with a loss of variation, especially when compared to the genetic diversity of southern populations that were not affected. One recent study has proposed the idea that ecotones are a source of speciation, which appears to have almost happened to Northern tick populations. For many years the northern *Ixodes* clade was called *dammini* and the debate over whether it is a truly separate species has been a lively one.^o Pleistocene glaciation events seem to have caused parallel biogeographic patterns of reduced genetic variability in the north for a wide range of animal and plant species. Various genetic studies of northern animal populations have often found short-spanning star phylogenies among haplotypes (differing from each other with one or two nucleotides) that is observed in northern *Ixodes scapularis* ticks.^u

Biodiversity also plays a key role in the spread of Lyme disease. When ticks have a variety of blood meal hosts they maintain a lower rate of Bb infection, due to the variability of host transmission capacities for the bacteria. Lizards, opossums and squirrels, for example, are considered poor transmitters. This is called a Dilution Effect Model. In reverse, when ticks feed on a limited number of species of animals that are good transmitters (mice and birds) there is a dramatic rise in the level of infected ticks.^{iv}

The 'hand of man' has helped to decrease the number of species living in New England today. Certain species, like the Passenger pigeon, have been hunted to extinction. Other species, like the white tailed deer and the beaver, have tottered close to decimation and then rebounded. The domesticated pig played a role in the ecology of the seventeenth-century forests by competing with indigenous animals for food and territorial

space. When forested landscapes were carved up and fragmented, biodiversity decreased.¹¹ New England's forest was extensively altered by Native Americans, deforested by European settlers, and has become reforested during recent decades because of farm abandonment.

Two modern studies have found a relationship between forest fragmentation and disease. Research done at the University of Florida found that fragmented forests had higher concentrations of parasites and that animals living within the fragments suffered from a higher level of infection than those living in undisturbed areas.¹² This may be related to an almost unavoidable interaction with an edge environment. A study of birds in western Minnesota, for example, found that brood parasitism was higher in nests located near a wooded edge than those located far from an edge.¹³ Another study found that the density and infection rates for *Ixodes scapularis* ticks with Lyme disease was dramatically higher in small forest fragments. Fragments that totaled less than five acres carried a Lyme disease risk that was seven times greater than that found in larger areas. This was also found to correlate with a high population of white-footed mice in these same forest fragments.¹⁴

Like everything else involved with Lyme disease, explanations for the episodic and epidemic level of virulence in New England among humans, ticks and mammals is complex and still subject to much study. But there seems to be a direct link with both biotic and genetic deprivation caused by glaciation and human activities like hunting, deforestation, and reforestation. In response, New England's Bb bacteria have evolved to specialize on the populations of genetically similar hosts which increases virulence and creates an elevated disease incidence rate.

Against this background, if those first English settlers visited modern Cape Cod, the wooded areas outside Boston, or suburban Connecticut today, and did exactly the same things they did back then, and wore exactly the same type of clothing that was worn more than three hundred years ago, there is a high likelihood that they would come into contact with *Ixodes scapularis* ticks and the *Bb* bacteria that they carry. Although the landscape has undergone major transformations over time, there are areas that can be identified as being most likely to have been inhabited by ticks in the 1600's.

On Cape Cod, these areas occurred in the marginal zones (ecotones) described by William Bradford and Edward Winslow as being between woods and sandy shore, in the "mosaic" of field and wood, under oak trees, and along paths used by deer. Later, when the settlers chose a site that was "fit for situation"¹⁵ at Plymouth or chose the most defensible sites around Boston Harbor, they chose sites that were undergoing the natural process of re-forestation. These areas had been previously cleared by Native Americans who had died in waves of earlier epidemic sickness. It is estimated that the Native population of the Massachusetts, for example, had gone from a pre-contact high of +/- 9,000 to about 900 by 1620.¹⁶ The entire village on the land that would become Plymouth Plantation died of disease in the 1616-17 plague.¹⁷

This meant that at least three years worth of brushy vegetation had accumulated on that site. The immediate adjacent area would also not have been subjected to at least three years of "burning," the Native practice for clearing brush and fallen leaves in the vicinity of their villages. Modern studies show that burning is an extremely effective tick control measure.¹⁸ But it also creates an area of luscious new growth in the following spring that is highly attractive to deer. The deer population in the area was described by

early observers as being near “abound,” probably because of the lack of Native hunters and the continued presence of both mast (acorns), the deer’s preferred food, and good habitat conditions.¹⁹ Re-forestation creates a marginal brushy edge zone that, when coupled with a supply of birds, mice, deer and ticks, is now part of the modern “geography of risk” for Lyme disease.²⁰

Modern scientists use several factors to predict the amount of risky ‘tick friendly’ habitat a geographic area contains. Tick densities are highest in moist but well drained areas with sandy soils over underlying sedimentary bedrock. Because the immature stages of *Ixodes scapularis* overwinter in topsoil and leaf litter, they prefer a forest like that in early Massachusetts that includes many deciduous trees. The brushy transition zone under trees and between forest and cleared land is most likely to harbor questing ticks. In areas where land is cleared, tick populations may be limited to river corridors.²¹

The following recommendations for the prevention of Lyme disease made by the Massachusetts Department of Public Health in 2001 would have been difficult or almost impossible to follow in *the seventeenth century*:

1. Educate your family about tick-bite prevention. *In the seventeenth century even if the colonists saw these minute ticks or found one attached to their skin they did not yet connect insects with disease transmission,* 2. Dress properly--wear light colored clothing, long sleeves, tuck everything in. Wear a hat. Wear tightly woven clothing. Wear shoes with covered toes. Clean your clothes immediately and properly using hot water and a hot dryer after potential contact. *Seventeenth-century men conformed somewhat with tucked in layers; women and children wore long untuckable skirts. Clothing was neither immediately nor properly cleaned,* 3. Avoid contact with tick infested vegetation. *During*

the exploratory initial phase of settlement the opposite was done. 4. Conduct tick checks anytime you have been in a tick habitat. *Non-engorged deer ticks are the size of the period at the end of this sentence and are hard to see even if anyone was actually looking for them,* and, 5. Use tick repellants and pesticides. *Some herbals may have been used as insect repellants but DEET was three centuries away from being available.*²²

Lyme disease is caused by the transfer of the *Borrelia burgdorferi* spirochete from an infected tick to the blood supply of a host bird or animal. In an endemic area it is possible that a percentage of the population is infected by the spirochetes at all times but only a portion of those infected ever become symptomatic. One early study by Dr. Alan Steere found that for every person in an endemic area who exhibits symptoms, there may be another person who has been infected but displays no symptoms whatsoever.²³

One of the earliest signs of infection can be an Erythema Chronimum Migrans (ECM), the classic jagged edged round bull's-eye rash at the site of the tick bite, although not everyone develops this rash. Other signs of early infection are nonspecific: sore throat, fever, muscle pain, joint pain, swellings and fatigue. Within days to weeks of the initial infection, the spirochetes may disseminate to tissues throughout the body through vascular or lymphatic channels.

Lyme disease is an extremely frustrating ailment to describe because it presents such a diverse variety of symptoms. Because so many body systems can become involved, Lyme disease can take on a wide array of forms. In some people it is an extremely severe pathology and can be fatal, especially if the heart muscle is infected or pneumonia develops. It is believed that prior to the establishment of an antibiotic protocol for Lyme disease, it was generally a miserable but, in normal circumstances, survivable

condition.²⁴ In one sad study also done by Dr. Steere in 1988, ninety patients in the former Soviet Union with ECM and flu-like symptoms that developed after documented tick bites were studied, *but not treated with antibiotics*. Within two weeks to four months of infection, sixty four percent of the patients had developed neurological abnormalities, including cranial neuritis, Bell's palsy, meningitis, and radicular pain. Another four percent developed arthritis. Untreated Lyme disease may have been responsible for some of the rheumatic conditions, aches, pains, dementia and mental illness that are thought to be a normal part of the aging process in Massachusetts.²⁵

The *Bb* bacteria itself has a complex relapsing or shape shifting life cycle depending on its environmental circumstances. It changes form. When its spirochete form



Figure 18. *Borrelia burgdorferi* in its spirochete form. Courtesy Dr. Burgdorfer.

encounters adverse conditions like an immune system attack, an antibiotic, or even a dunk into distilled water, it can transform itself into either a spheroblast L-form that can invade the host's own cells, spewing outer surface proteins (Osp) in the process, or into a cyst as a survival mechanism to escape unfavorable conditions. The cysts play a role in

protecting the bacteria's DNA with its genetic markers. Dr. Wily Burgdorfer states that this cystic material is found in every animal and human tissue that is infected with *Borrelia burgdorferi*.²⁶ These cysts are highly resistant to destruction.

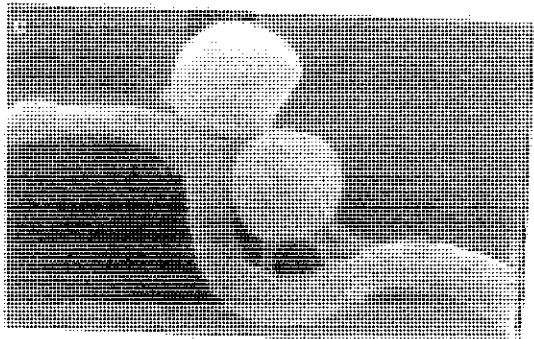


Figure 19. *Borrelia burgdorferi* converting to cyst form. Courtesy Dr. Burgdorfer

Some hormonal responses, however, especially those associated with menstruation, stress, and a weakened host immune system, seem to trigger an "all clear to revert to spirochete again" signal as can the arrival of a new set of bacteria.²⁷ One recent study found that *Bb* spirochetes also participate in a form of mating that transfers genetic material in the process.²⁸ Previously cysted *Bb* within a host's body may sense the outer surface proteins of newly arrived bacteria and transform into spirochetes to meet them. This may be one of the problems discovered during the Lymerix vaccine test trials--people who had a pre-existing inactive cysted form of the bacteria within their bodies had it transformed into an active spirochete infection by a the outer surface protein factor in the vaccine, making them sick.²⁹

Signs that a Lyme infection has disseminated (spread through the body) can include severe pulmonary, cardiac, neurological and musculoskeletal symptoms,

headache, malaise, fatigue, profuse sweating, swollen glands and hoarseness. Patients who are bitten on the head or neck have a forty percent greater chance of having neurological problems. The most common Lyme-associated neurological conditions are cranial palsies. And the most common (occurring in five to ten percent of patients) of these is Bell's palsy (which affects the face usually causing paralysis on one side). Meningitis and encephalitis, inflammations of the membrane that covers the spinal cord and brain, and encephalomyelitis, an inflammation of the brain itself, can occur in late stage Lyme disease.³⁰ Because of this brain involvement, Lyme disease is also linked to psychiatric manifestations. Some patients have attempted suicide.³¹

The *Borrelia* bacteria has been found to be secreted in milk, urine, and semen. It therefore may have additional pathways of infection that include sexual and congenital transfer although this is still subject to further research and controversy.³² Modern studies of pregnant women from endemic areas in Italy who unfortunately suffered from undetected Lyme disease during their pregnancies show that spontaneous abortion, stillbirth and extremely rare cases of fetal deformity can occur.³³ One of the side effects found in the FDA trials for the Lymerix vaccine was a high rate of spontaneous abortion among pregnant women after inoculation.³⁴ There are two well described births from the Boston area in the seventeenth century where severe fetal deformities occurred which may or may not have been related to this disease.³⁵

Modern veterinary studies have shown that domesticated animals are subject to Lyme disease infection. *Bb* spirochetes have been cultured from dogs, cats, goats, cows and horses. Cows that are infected can become lame and lethargic. They are also subject to a higher than normal rate of bovine abortion, may stop producing milk,

or may pass the spirochetes along in their milk (it is killed by the heat in the pasteurization process).³⁶ In the twenty-first century, Lyme disease is endemic in New England, epidemic in some areas, and Cape Cod has one of the highest incidence rates in Massachusetts. Was Lyme disease present in seventeenth-century Massachusetts? There are many tantalizing clues that push the disease's existence back to the seventeenth century and even earlier.

The tick itself has an evolutionary history that spans at least 120 million years. The earliest fossilized tick from North America (*carlos jerseyi*), dated at 90 to 95 million years old, was found in a piece of amber excavated from central New Jersey. It was found in association with a small bird feather which may denote the early precedent for a relationship between ticks and migratory birds.³⁷ The subgenus *Ixodes* may have formed +/- 80 million years ago, prior to the break up of Laurasia into North America and Eurasia.³⁸

Borrelia burgdorferi, the Lyme disease bacteria, has been traced by DNA amplification back to at least the Paleolithic Era (anywhere from 400,000 to 15,000 years ago). It was first acknowledged as a human pathogen in medical literature in the United States between 1982 and 1983. The most studied transfer mechanism for Lyme disease is the zoonosis model which involves the bacterium being present in local vertebrate reservoir hosts (white tailed deer, white footed mice, migratory birds) and then being transferred incidentally to humans via an arthropod vector. Lyme disease has been found to be endemic on a world wide basis, caused by several different genetically distinct strains of *Borrelia* carried by various members of the *Ixodes* tick species.³⁹

This pan-endemic occurrence and its complex shape shifting physiology are probably the strongest evidence to support a lengthy and protracted evolutionary

existence for the bacteria.⁴⁰ Over time the *Bb* bacteria developed effective mechanisms to evade the immune systems of its blood meal hosts to survive and sometimes thrive. One DNA sequencing study has led to a hypothesis that the *Borrelia* pathogen is distantly related to African Swine Fever, which has a similar relapsing life cycle, mammal host vector (pigs), and arthropod (louse) mechanism for spreading.⁴¹

European ticks were probably cross infected from several points of contact over a long period of time including after the Viking interactions with North America (Vinland), and again after 1492. The sixteenth century prevalence of a form of land scurvy and the English claim that this affliction was brought over by the Normans in 1066 may simply point to a dietary downturn for the conquered English people. But it could just as easily be the result of a tick and *Borrelia* bacteria accompaniment to the invading army, perhaps on or within humans, horses, dogs, cattle and swine.⁴² However they got there, a wide variety of subspecies of *Borrelia* are found in various parts of Europe today.

The well traveled Vikings may have been active players in Lyme disease dissemination. In the Icelandic Sagas, the areas around Vinland (thought to be a section of Newfoundland and the St. Lawrence River Valley in modern Canada) were described to be heavily populated by deer. The Vikings used puffins and other seabirds as a significant dietary resource.⁴³ Modern puffins that are found in the area between the islands off the coast of Maine in North America and Scandinavia in Europe have been found to be infected by *Ixodes uriae* ticks and to carry a form of the Lyme spirochete. Modern Scandinavia is an area where Lyme disease is prevalent.⁴⁴

Possibly the earliest written description of Lyme disease in Europe comes from the Island of Jura (Norse for Deer Island) located off the coast of Scotland. This island

was once within the Viking sphere of influence. After visits in 1764 and 1771 by the Reverend Dr. John Walker, he wrote "in the island of Jura, the cripples are remarkably numerous; owing to a very singular disease with which this island is infected." He goes on to describe a worm that is "of a reddish colour and of a compressed shape with a row of feet on each side" that "penetrates the skin with several small ichorous orifices [tick bites]...the worm disappears soon after this stage of the disease [a tick falls off after a blood meal] but when it is suffered to come this length, it never fails to cripple the patient for life. And the intense pain with which it is accompanied sometimes destroys the appetite and spirits and occasions death."⁴⁵

There are several different interpretations for the history of the North American spread of Lyme disease that can be pieced together. It may predate human contact. Endemic in Siberia, it may also have been carried over from Asia with the earliest human occupants and their dogs when they walked across the Bering Strait. The bacterial infection of Lyme disease can sometimes be detected by the appearance of scar damage to the bones of infected animals, especially in the joint areas. Lyme-like peripheral polyarthritis lesions (scars from infectious damage to the ends of multiple bones) have been found on one set of human skeletal remains in Alabama that date to 5,000 B.C.⁴⁶ Another possible diagnosis of Lyme disease as the cause of similar pathological bone conditions comes from a prehistoric Tchefuncte Indian adolescent skeleton that dates from between 500 and 300 A.D.⁴⁷ This theme can be continued by theorizing that Columbus, landing on islands that lie along the route of birds migrating from the north (including an astounding estimated population of five million of the now extinct Passenger pigeons), then brought Lyme disease back to co-infect Europe again from the

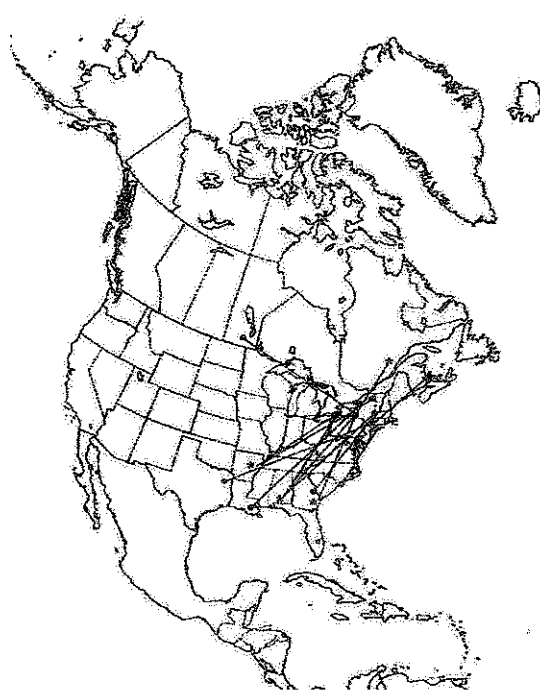
New World after 1492. It is of some interest to note that Columbus himself suffered from severe arthritis, periods of temporary blindness, and hallucinations (all symptoms of late stage Lyme disease) only after he returned from the New World. He was so terrified during his fourth voyage to the New World by "a sea turned to blood boiling as a cauldron on a mighty fire" that he abandoned his ship for an extended period.⁴⁸

Birds have been found to play a crucial role in both the spread of the disease in the Midwest and along the Atlantic coast of North America. They are competent maintenance hosts with good transmission capacities for the *Bb* spirochetes. Birds can be sensitive to temperature related ecotone variations in North America. Their ranges have been found to expand and contract as ecotones are displaced. However, population density and competition also play an important role in determining bird distribution. Infected birds play a prominent role in transferring *Bb* to larval and nymphal ticks as well as by transporting ticks into wide ranging areas.⁴⁹

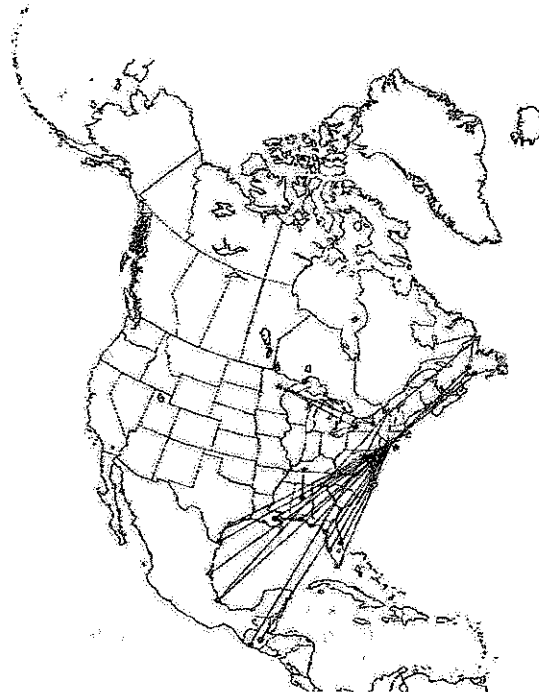
The first English settlers in Virginia arrived in 1607. Almost immediately they began to suffer from what they called "the summer sickness" which included the symptoms of "swellings," fevers, extreme lethargy, and irritability.⁵⁰ All these symptoms can be attributed to Lyme disease. The Jamestown area is located along the major Atlantic coastal bird migratory route and is, in modern times, endemic for Lyme disease.

The islands that Columbus first interacted with in 1492 are also located along these migratory routes. Birds may have spread ticks and the infection along a swath of the North American coastline.

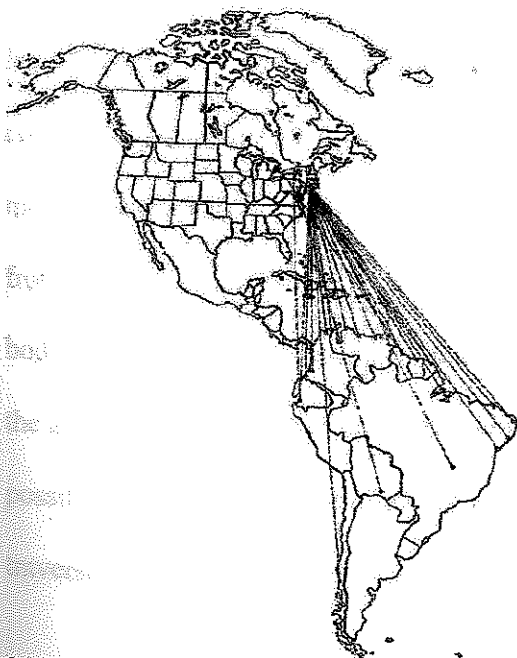
Figure 20. MAJOR BIRD MIGRATORY ROUTES IN NORTH AMERICA



1. Southeastern U.S. migration pattern.



2. Circum-Gulf migration pattern.



3. Caribbean/Western North Atlantic migration pattern.

From: *Emerging Infectious Diseases*. Vol. 6 No. 4. July-August 2000, 325-326.

Two sets of pig (*sus scrofa*) skeletal remains from the Boston Cross Street privy 4 archaeological site may show possible evidence of a *Borrelia burgdorferi* infection in seventeenth-century Massachusetts. One bone, a scapula, contains signs of a massive bacterial infection. Another set of bones, from an immature pig skeleton (aborted fetus), show deformity on the distal half of a tibia that would have been the result of an infection of the bone via the blood stream.⁵¹

Studies of journals that were written during historic periods of intense interactions (military camp-outs) in what are now Lyme endemic landscapes also show numerous references to deer, ticks and lingering ailments. In a 1685 petition for a land grant in return for their service during the Narragasset Expedition, the veterans wrote:

We think we have reason to fear our days may be much shortened by our hard service in the war- from the pains and aches of our bodies, that we feel in our sinews, and lameness thereby taking hold of us much, especially in the spring and fall⁵²

Veterans from the American Revolutionary War of 1776-1783 and the American Civil War of 1861-1865 suffered from high levels of "rheumatism." Records for Revolutionary War veterans are somewhat rare, but even those often list rheumatism contracted while in service as a disabling condition.⁵³ Records from the Civil War are more extensive. A Civil War veteran census that was taken in 1890 showed that disability from rheumatism afflicted about twenty five percent of that population, second only to bodily damage from gunshot wounds. In numerous instances it states that men went into the army in full health but had suffered from rheumatic joint pain ever afterwards. It is possible that some of these ailments were actually Lyme disease contracted while traversing, camping in, and otherwise intensely interacting with a mosaiced and "tick

risky" landscape, especially the coastal area of Virginia and Maryland during the Peninsular Campaign.⁵⁴

The examination of museum specimens' DNA for the *Borrelia burgdorferi* bacteria has yielded an earliest found actual infection date in North America of 1894 for a white footed mouse (*Peromyscus leucopus*) from Dennis, Massachusetts (located on Cape Cod).⁵⁵ The earliest preserved *Borrelia* bacteria found in Europe sets an 1884 infection date for an *Ixodes ricinus* tick from a museum collection in Germany.⁵⁶ A lack of earlier datable specimens precludes earlier dates.

Native American oral history can also be examined for evidence of a pre-contact North American existence of Lyme disease. At least two Native American groups have traditional stories that clearly link deer with arthritis. These may be reflective of a very real health problem in pre-contact Native society. In a traditional Creek story, hunters are warned to be cautious with deer because they have mysterious powers. If a hunter doesn't show proper respect when he has killed a deer, they would cause him rheumatism and the hunter would be forced to walk the rest of his life with aches and pains. Cherokee folklore contains both the strong admonition against touching the skin of a diseased deer and the following story about respecting the spirit of slain deer:

Little Deer, the chief of the deer and the animal spirit who took vengeance on unthinking hunters, ran as swiftly as the wind to a deer just killed. Bending over the blood spots on the ground, Little Deer asked the spirit of the deer if it had heard the hunter make amends, the proper prayer. If the answer was yes, then Little Deer left. If no, Little Deer followed the trail of blood left by the hunter who then carried the deer to the hunter's door, where he "[put] into the hunters body the spirit of rheumatism that shall rack him with aches and pains from that time henceforth."⁵⁷

There are litanies of Native American herbal treatments for arthritis and rheumatism showing that these were diseases that indigenous people confronted. Some of

the Native cures, like bearberry and goldenseal, have mildly antibiotic properties that would possibly be effective for treating Lyme disease.⁵⁸

IV. WHAT HAPPENED IN 1620?

Arriving on November 11, 1620, at what would have been a peak time period for questing adult *Ixodes scapularis* ticks. The Pilgrims violated each and every anti-tick recommendation put forth by modern epidemiologists. The historic records state that initially the weather was quite warm and pleasant which means that any local ticks could have been active. The Pilgrim's methods of exploration brought them into direct contact with a mosaiced landscape full of marginal areas-'the geography of risk' for Lyme disease. The men describe exploratory journeys in which they marched through boughs, bushes, and edge ecological zones full of "brush, wood-gaille, and long grass." They followed paths made by deer.¹ Deer have tarsal glands halfway up their legs that produce pheromones to mark their territory as they walk. Ticks find these gland secretions highly attractive, which leads to a greater abundance of ticks questing along deer traveled paths. By using deer paths, these explorers not only exposed themselves to a greater concentration of questing ticks, they probably acquired an odor that would attract Ticks.²

When the first group of men left the *Mayflower* to explore the landscape of Cape Cod, they would have been wearing many layers of clothing. For the upper body, a man in the seventeenth century wore a long, short sleeved collared linen shirt. On top of that he wore a doublet which was relatively close fitting with long sleeves and padded shoulders. It buttoned down the front. A collar and cuffs might have also been attached. A felt hat or cap was usually worn and in cold weather a cloak was added for warmth. For the lower body, men wore front buttoning breeches which extended to the knee and were worn with knee length socks and low heeled leather shoes or boots.³

Preparing to face the unknown dangers of 1620, the exploring men were all armed and wore protective chain mail under a form of upper body armor called brigandine corselets. These were made of quilted cloth with small metal plates sewn inside. William Bradford noted that the boughs and bushes in some areas were so thick that they tore their "armour in pieces."⁴ Miles Standish, as a professional soldier and veteran of the wars in the Netherlands, probably owned his own set of full metal gear with a metal helmet. He was also equipped with the most modern of seventeenth-century weapons: the revolutionary snaphance. This was the first firearm that used a flint to spark a firing mechanism. Others carried cumbersome, difficult to aim, matchlock muskets which required a lit wick in order to be fired.⁵

While this male apparel, with its layering and pants tucked into tall stockings, may have been mildly protective (although once covered with deer pheromones this advantage may have been lost) if a tick found its way to human flesh it was probably there to stay unless scratched off. Bathing was not part of any prevailing practices at the time.⁶ A lack of bathing would tend to be protective of any attached ticks. A tick needs to bite, attach, and be in contact with a host's blood for at least 24 (some experts say 36 some say more) hours to successfully transfer the *Borrelia* bacteria and subsequent Lyme infection. If the attachment lasts at least that long the infection rate can be extremely high.⁷

The apparel of the women who had left the *Mayflower* to refresh themselves would have put them at high risk for Lyme disease. A woman's one undergarment consisted of a long short-sleeved linen shirt that was fastened in front. Over this shirt, one or more ankle length petticoats were worn. Over these she wore a wool gown that

consisted of two parts: a bodice (which may have had removable long sleeves) and a skirt. The skirt was ankle length. Women wore knee high socks held up by garters and low shoes and a cloak in cold weather. If the children were allowed on land many of them would also have been wearing long skirts. Children of both sexes wore long gowns made of linen or wool with long sleeved bodices until they were about 8 years old.⁸

In November of 1620, some women came ashore on Cape Cod to wash, and in the process did what could be best described as an unintentional tick collecting procedure. In modern epidemiology this is called a flag or drag test. It is done by dragging a light colored cloth over the ground and then counting the number of ticks that have attached themselves to the cloth.⁹ The Pilgrim women's clothing left them vulnerable to collecting questing ticks when their long cloth skirts were dragged over the ground. Any ticks attached to their skirts would have been carried unnoticed aboard ship. Once on a skirt hem a tick could easily find exposed flesh for a blood meal.

Even worse, piles of wood and shrubby possibly tick filled juniper branches were collected and transported from shore to ship to be burned on a daily basis.¹⁰ A 1999 study of the relative potential for acquiring *Ixodes scapularis* tick nymphs while crawling, walking, or sitting in a deciduous woodland found that some risk for acquiring ticks existed for all three activities, but that crawling on the ground raised the risk. Also, tick nymphs were found on +/-85 % of the logs large enough to sit on in the study area which indicates that sitting on one was tick-risky behavior. While it is impossible to know, it is highly likely that both men and women sat on convenient logs in 1620.¹¹ The Pilgrim men walked along deer traveled paths and then slept overnight in the grass at the edge of the woods. Because of these activities, if there were ticks infected with Lyme disease in

the area in 1620, they would have had numerous opportunities to infect both the settlers and the *Mayflower* itself.

Within a few weeks of contact with the landscape of Cape Cod people began to get sick. Some of the symptoms of Lyme disease that William Bradford and Edward Winslow described were flu like symptoms including coughing (scarce any of us were free from vehement coughs),¹² headaches (he complained greatly of his head),¹³ joint pain (William Bradford feels pains in his ankles and hip),¹⁴ fatigue (the weakness of our people),¹⁵ death, and possibly a congenital transfer of the bacteria to a fetus. Goodwife Allerton delivered a stillborn child on December 22 and then also died herself within a few weeks.¹⁶ Out of 102 settlers and an unknown number of ship's crewmates only 52 settlers and a handful of crew mates survived through that first winter. The oldest man, James Chilton, was one of the first to die. Many other men, most of the women, and many of the children died. Others got very sick. William Bradford himself was "vehemently taken with a grief and pain, and so shot to the hackle-bone (hip)." He felt some pain in his ankles "by times" and came close to dying but did survive.¹⁷ All of these symptoms would be highly similar to those of scurvy which would account for the original diagnosis. Although no skin rashes are noted anywhere in the historic record, it is interesting to note that the ECM of Lyme disease might have been mistaken for the skin spots that accompany scurvy.¹⁸

But there is also an odd distribution of survivors that argues in favor of Lyme disease and against the disease that mostly closely mimics its symptoms- rheumatic fever and rheumatoid diseases. Prior to the discovery of the *Borrelia burgdorferi* spirochete, many people who suffered from Lyme disease were often given some sort of rheumatoid

diagnosis. One of the factors that led to the discovery of the Lyme disease spirochete was the fact that clusters of rheumatoid disease began to occur in specific geographic areas-like Lyme, Connecticut. Since rheumatic disease is considered to be an individualized immune response, it is not considered to be contagious, which made the scientists at Yale University begin to look for another causative factor for the clusters of infection. Eventually *Borrelia burgdorferi* was discovered.¹⁹

The precursor of rheumatic fever and some rheumatic disease is usually strep throat, a common infection that is caused by the *streptococcus* bacterium. This bacterium has been one of the most extensively studied and analyzed pathogens known to mankind. It can spread rapidly in crowded conditions. It is spread by person-to-person contact with infected saliva and mucus. In some cases a severe streptococcal throat infection leads to a secondary disease- rheumatic fever-which may or may not have associated symptoms of rheumatoid arthritis. These diseases are thought to be an individualized immune response to the infection itself. The mechanism for rheumatic disease development within an individual patient's body is still unknown.²⁰ In the aftermath of Rheumatic fever, the majority of victims suffer life shortening heart and kidney problems.²¹ There is no record of the population at Plymouth suffering from these subsequent problems that are the lingering hallmark of Rheumatic fever. To the contrary, many survivors lived long productive lives, outstripping the life spans of their contemporaries back in England by decades.²²

Some members of the *Mayflower* population who would normally be at the highest risk for any type of contagious infection survived. They included some of the weakest, the oldest, and the youngest-newborns. These survivors have a common

attribute: they were among the least likely to go ashore. Survivors included the two women who had just given birth prior to contact with the land, as well as their newborn children. William Brewster and his wife, almost the most elderly in the group, survived—he didn't even get sick although he spent his time nursing others. But Miles Standish, who probably tromped his way through the countryside in front of the other exploring men clad in protective full military apparel (it would be difficult for a tick to attach to a metal surface) was likewise untouched by illness. He also spent his time nursing the ill back to health without catching the disease. This argues against a contagious infection spread by casual person-to-person contact.²³

Hormonal secretions related to stress have a triggering affect on the *Borrelia burgdorferi* bacteria.²⁴ When exposure to Lyme disease was added to the additional stressors of extremely crowded living conditions, exposure to the elements, poor shelter, and fear; it became a fatal encounter. Contact with the marginal, brushy, edge terrain, the 'geography of risk' for Lyme disease, at peak time periods for questing ticks could and did make people very sick. This was the result of a series of events that had occurred prior to 1620. The environment of Cape Cod, Plymouth and Boston area had been undergoing changes in response to the dramatic decline of the Native American. The deer population may have increased because of the decline in Native hunters.

New England's seventeenth-century tick populations would have thrived in response to any increase in the white tailed deer population. A modern study done at Great Island in West Yarmouth, Massachusetts points out this close relationship between deer and tick populations. A tick survey, using the 'drag test' method, was done between 1982 and 1985 on this small island off the coast of Cape Cod that was populated by white

tailed deer. The results showed both a high level of deer ticks and a high Lyme disease infection rate among the collected ticks. The Island was privately owned and the owner, whose grandchild had contracted Lyme disease, was in no mood for unequivocal results. After what must have been a nasty slaughter it was estimated that ninety percent of the island's deer population had been eliminated. Several subsequent tick tests on the Island show a corresponding drastic reduction in the *Ixodes scapula* population.²⁵ The same thing happened when deer were eliminated from Monhegan Island, off the coast of Maine. Inversely, a high population of deer would probably have been translated into a high population of ticks.²⁶

V. THE MASSACHUSETTS BAY COLONY: SCURVY DEJA VU?

Eight years after the Pilgrims experienced what they thought was 'scurvy,' the next group of settlers to come to Massachusetts experienced similar symptoms. Once again their self diagnosis was scurvy. Beginning with their first settlers in Salem in 1628, the Puritan settlements around Boston, less than forty miles north of Plymouth, experienced their own initial period of sickness. On March 15, 1630, John Pond wrote: "People here are subject to disease for here have died of the scurvy and of the burning fever two hundred and odd, besides many layeth lame, and all Sudbury men are dead but three and the women and some children."¹ Sudbury was even closer to the deer and tick infested forest edges than Boston.²

The Winthrop Fleet arrived in 1630 and some of its passengers spent time exploring the Massachusetts Bay region in June. This is a high level month for questing tick nymphs. John Winthrop and others went on exploratory journeys while trying to decide where to settle. The Puritans changed their minds about their chosen settlement site in response to a rumored threat from the French. This required some degree of trekking back and forth through an overgrown landscape.

They were then subjected to a period of disease. Once again interaction with the landscape immediately proceeded the period of sickness. Even the Puritans themselves made comparisons with the experience of Plymouth as a model to help get them through a similarly difficult time. John Winthrop wrote "the first at Plymouth suffered a similar tempest of pestilence but have now grown healthy and thriving." He sent for Plymouth's

Doctor Samuel Fuller "who did such good work with the scurvy"³ to help his sick colony. Dr. Fuller's cure, in modern times known to aggravate many conditions, consisted of "bleeding" the afflicted. His actions may have actually elevated the death rates in both colonies.⁴

The move by a large segment of the population from the outlying settlements in Massachusetts Bay Colony to Connecticut during the mid 1630's was the direct result of problems that they had with the health of their cows. Once again they looked at the quality of food as a causative factor, in this case the grass eaten by their cattle. In their ethno-centric minds even English grass was better than American grass for their cattle. Fields "that were grubbed down to bare soil by pigs and goats" could then be made superior by being sown with English grass seed.⁵ Inhabitants of outlying areas like Newtown, Dorchester, Salem Village and Concord felt that wild American grass was making their cows sick by being "too rich" and chose to move to the greener pastures in Connecticut.⁶

The symptoms described (weakness, failure to produce milk, spontaneous abortion) have all the strong earmarks of a bovine Lyme disease infestation. In Concord the problem with cows was compounded by a frustrating problem with the high population of wolves. In addition to killing a stray calf or two, every time the settlers planted a field of corn with fish as fertilizer, the wolves would come at night, dig everything up, and eat the fish. Things, however, did not initially go too well once the settlers moved to Connecticut either. In 1636 John Winthrop noted, "Their cattle died, many of them, and cast their young, as they had the year before."⁷

Once the settlers and their domesticated animals, especially pigs, began to focus their time and attention on the cleared ground in and around their homes, and reinstated the practice of burning (firing) leaves and vegetative litter, sickness abated. New arrivals to Plymouth and Massachusetts Bay did not suffer from any subsequent initial periods of great sickness and 'scurvy.' After the initial phase of exploration and until the population began to press against the 'wilderness,' contact with marginal areas in the environment became a sporadic occurrence with sporadic sickness occurring. Even after suffering from Lyme disease's effects, both the men and those women who survived complications from childbirth (the leading cause of death for women in the seventeenth century) were usually able to live long, relatively healthy lives. Life spans of sixty, seventy, and even eighty years were reported.

Once a site for settlement was decided on, the early settlers proceeded to burn off undergrowth and leaf litter, set loose their pigs on the forest edges to gobble up acorns, and place a physical and mental overlay of English social and civil order onto the landscape. Salem, Plymouth and Boston were all set up initially with a central nucleus of house lots with a set of common fields laid out around it. Traditional husbandry practices were altered to fit the New England climate. Animals needed to be protected from wolves and were more likely to be sheltered during the winter than they had been in England.⁸ New American crops like pumpkins, beans and corn were grown. Corn fields were fertilized with fish, an adoption of a Native American practice. English grass seed was imported. Like the human settlers, English grass thrived in New England and it begins to show up in bog core samples almost immediately after 1620.⁹

New England's almost instantaneous creation of a thriving local market for cows and swine was the foundation for Massachusetts's fledgling economy and its first West Indian links. The domestic animal and meat market kept New England afloat during the 1630's and offered a sure commodity for the West Indian trade when it began to develop in the 1640's. By 1633 John Winthrop stated that the domestic cattle trade had saved the Massachusetts economy from ruin. The colony "by reason of so many foreign commodities expended, could not have subsisted to this time [except] that it was supplied by cattle and corn, which were sold at a very dear rate." Cows sold for between twenty and twenty six pounds. These high prices led to the importation of animals from both England and Virginia for profit.¹⁰

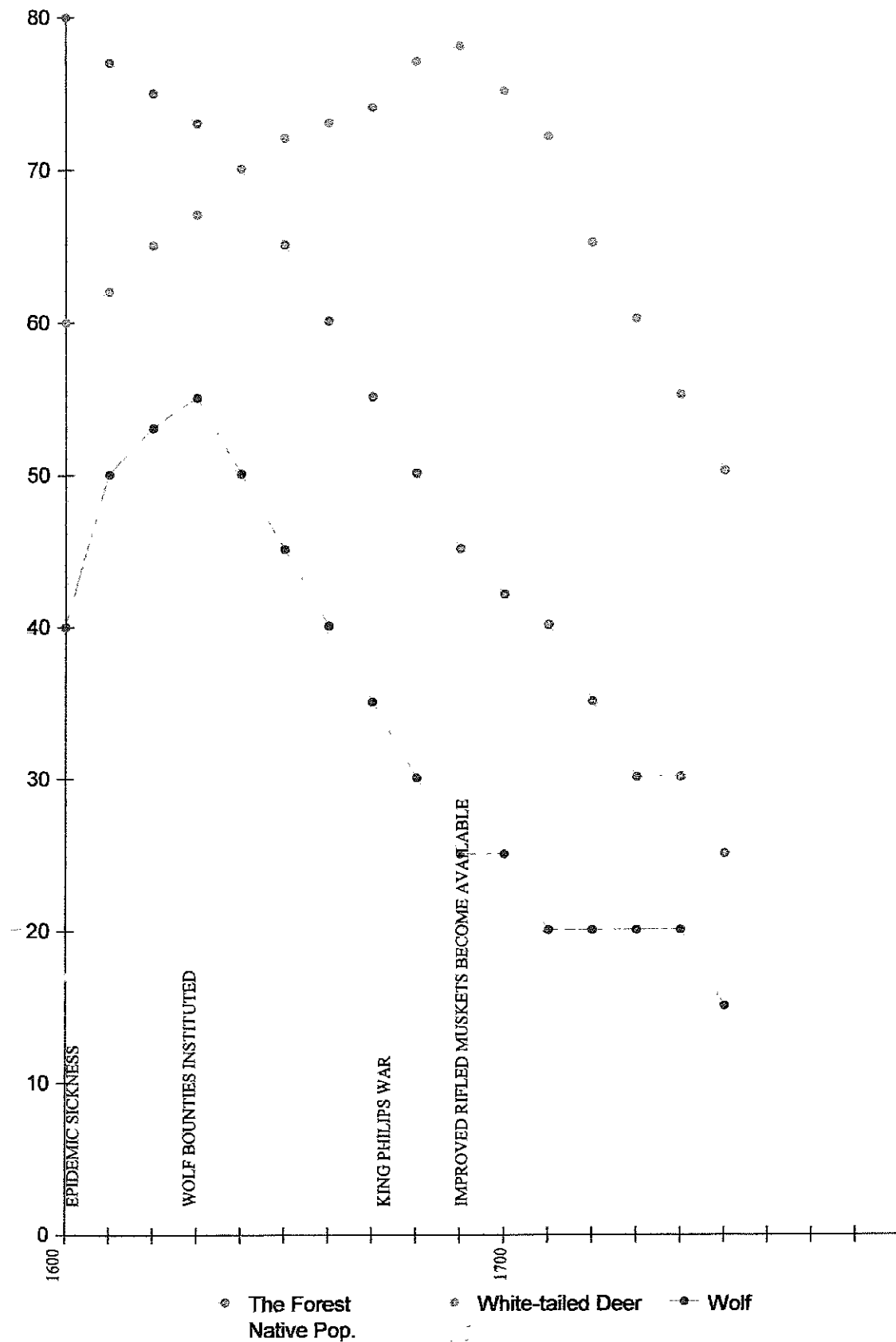
The introduction of an English style economy had some immediate effect on the forest landscape, especially on the home ranges of deer, mouse and wolf habitat occupants. A landscape model for seventeenth-century Massachusetts shows a high pre-contact deer population that grows even higher as their prime predators are diminished over time. It shows a wolf population that is affected and, by the nineteenth century, eliminated by a bounty eradication system. [See model]¹¹

There are several important factors that influence the development of this model. The first factor is the seventeenth century elimination of one of the deer's primary predators- the Native American. During the early seventeenth century it is estimated that epidemics of smallpox and the plague killed nearly ninety percent of the Native population in what is now the state of Massachusetts. This should be reflected on the population chart by a decrease in the number of hunters, and a reactive increase in the

A POPULATION DIAGRAM FOR 17TH CENTURY MASSACHUSETTS FOREST

THOUSANDS

FOREST IN THOUSANDS OF ACRES



total deer population. With more deer available there would have been another reflective increase- in the number of predators.

“Lions” were seen and described from 1620 on. In Plymouth the first settlers heard the growls of a mountain lion and had to hold their dogs back from attacking it.¹² Wolves were considered flat out pests. They became the pariah of the wilderness--dark, insidious predators biting at the heels of civilization. They had a price on their heads from almost the moment of contact with the English colonists. Well nourished on deer meat, this thriving wolf population was unfortunately not discerning enough to know a domesticated animal from their wild prey. When they began to add pork, beef, and mutton to their diet it was not tolerated.¹³ In 1678 Salem Village was rimmed by a set of wolf traps.¹⁴ The last wolf bounty in Massachusetts was paid in the nineteenth century at the end of a successful eradication program that took over 200 years to complete.¹⁵

The second factor, perhaps unique to the English colonists in Massachusetts, was the almost complete lack of interest in hunting deer that was displayed by the almost homogeneous middle class group of settlers who went there. In English common law, game (especially deer) was by custom owned by the crown or upper class nobility who had the legal right to hunt their own animals. Most of the first settlers in Massachusetts did not come from that social class. These settlers had not been deer hunters in England and seem to have shown very little interest in hunting them when they immigrated to the New World. There may also have been a moral imperative involved, as stag hunting had been a pastime for the “decadent “ upper classes at home in England. Both the Pilgrims and the Puritans would have had no desire to emulate this type of behavior.¹⁶

While the seventeenth century colonists were inclined to ignore them, a deer population that was "abound" led to other problems that required governmental intervention. A high deer population led to their unwanted incursions into crop and garden fields. *New England's Annoyances*, written in 1647, mentions this problem:

Even when it is grown to full corn in the ear,
Its apt to be spoil'd by hog, racoon and deer.¹⁷

The death records from King Philip's War include one farmer who grabbed his musket in anger to go out and shoot deer that had gotten into his corn. He was instead killed in the corn field himself by marauding Natives.¹⁸

The Massachusetts Court records note the appointment of deer reeves.¹⁹ A reeve in the seventeenth century was appointed to control animal pests, often in response to citizen complaints. These seventeenth-century reeves were not eco-friendly Park Rangers paid to conserve a precious resource. They were hired with the responsibility for keeping destructive wild deer out of lucrative crop fields.

A thriving deer population also created economic problems in what quickly became a cattle based economy. Prices for domestic cattle reached a seventeenth century high within months of the Puritans' 1630 settlements. The already established Plymouth Colony reaped a prolific profit by supplying cows and beef to the newly arrived Puritans. Numerous entries in John Winthrop's journals mention both the high price for cows and the practice of going to Plymouth to get them. Matching the number of animals found in the probate records for post-1630 Plymouth with the few actual archaeological bone assemblages found from the same time period invariably show in-home meat consumption at a much lower rate than the large total number of animals owned. This is

indicative of a thriving livestock market where a high percentage of the animals owned were being sold, slaughtered and eaten elsewhere.²⁰

Many early frontier towns in the Massachusetts Bay Colony, like Sudbury, were set up on inland meadows specifically as cow towns. The domestic beef market became a key part of the Massachusetts Bay Colony's economy that would later shift focus from the depressed domestic market to provide an important commodity for early trade with the West Indies.²¹ In 1692, for example, George Corwin, the sheriff of Salem, was kept busy barreling up meat from confiscated cattle to be shipped at a high profit to the Indies.²²

The records of the Massachusetts Bay Colony show that economic control was an ongoing governmental concern. When there was a shortage of wheat in 1641, fearing high prices, the directive was passed that "no baker, ordinary keeper or other person shall bake, to sell or set to sale any bread or cakes made of wheat meale."²³ That same year the building of ships was encouraged because it is "a business of great importance for the common good."²⁴ Citizens of the colony were encouraged to raise sheep because wool made cloth that was less flammable than cotton.²⁵ When the high price for livestock brought animals streaming to market from outside Massachusetts, a protective tariff was instituted to discourage competition and protect the farmers of the colony.²⁶ The supply of meat was protected by regulating the number of swine that could be kept at certain periods of time.²⁷ In 1643 a moratorium was placed on the killing of calves by butchers, but not those who did so for "their owne use, although they should part with some part thereof to some of their neighbors." As conditions changed this law was rescinded after "some months."²⁸ By 1685, the slaughter of animals in Boston was such a booming

business that there were complaints about "great inconveniences by reason of filth and dirt cast into their slaughter houses and yards of blood and other filth, although such houses and yards are scittuate neare streets and lanes much frequented."²⁹

The laws concerning deer that were entered into the records of the Massachusetts Bay Colony that are often cited as deer conservation measures were more likely to be related to the economic concerns about this crucial domestic beef market. One, for example, stating that deer could not be sold in Sudbury without governmental approval, was aimed at making sure that venison would not become a marketable commodity that was in any way competitive with domestic meat (especially beef) production. This ban was later expanded to include the entire colony.³⁰

Hunting bans on deer also seem to be related to, and protective of, fluctuations in the domestic beef market. By eliminating the competition of free venison in the winter months when tracking deer in the snow makes hunting easier, and shipping was limited or affected by the weather, meat prices could be kept fairly stable in the domestic market even if meat supplies were high. In the good shipping months having some of the domestic meat consumption needs supplied by venison would actually have been advantageous: it allowed producers to reap more profit from the barreled beef that could be sent to the West Indies.

Another factor related to the deer population was the fact that at first, even if the settlers wanted to hunt for deer, it would have been difficult. The settlers' earliest weapons, especially the matchlock musket, were not conducive to either the pursuit or shooting of deer. They were awkward and difficult to aim. They were so heavy that they needed a stick stand to hold them up while firing. The firing mechanism was dependent

upon having a lit wick (match) at the time of use. The awkwardness of this is evident in the events that occurred during the first two expeditionary forays of the Pilgrims on Cape Cod.

Sensing that they might be near a Native American village, these early explorers had to stop and make a fire to light their matches (actually a sort of smoldering wick) so they could be prepared to fire their muskets. Any deer (or Native Americans) that hadn't already run away would have been frightened away by the smell of smoke and fire.³¹ Later, during their "first encounter" with attacking Natives, part of the battle action consisted of having to pass around a burning log to light matches before the muskets could be shot. Once shot, the heavy muskets were dropped and replaced by cutlasses and sabers when the Pilgrims charged after their, by then, fleeing attackers.³² Since deer can run faster than humans, especially those who are weighted down with chain mail and armor, the Pilgrims' sabers and cutlasses were also probably not good deer hunting weapons.

In that 1620 encounter only one man had a snapchance, the earliest musket that used a spark from a flint to ignite a shot. Snapchances would become rapidly popular. By 1630, the Massachusetts Bay colonists had 300 of them on their inventory list to bring with them to the New World. But it took until late in the seventeen century for the flintlock musket to be perfected, and until between 1720 and 1740 for the barrel to be rifled (have grooves inside that put a spin on a shot as it is fired.) This created a light, accurate, aimable, easily fired weapon-- the American long-rifle.³³ It was only in the early eighteenth century, as New England's culture matured and began to deviate from the established patterns that had been initially brought from England, that this rifle was

aimed at deer. It was the eighteenth century that would see a dramatic decline in New England's deer population.

The focus of the English settlers in Massachusetts at first was on fishing, building homes and farms, and on their domesticated animals, not deer. It is difficult to analyze seventeenth-century deer usage in Plymouth, other than the occasional written mention of deer hunting, because the very early attempts at archaeology tended to throw out animal bones and remains as unimportant. The few modern archaeological studies done have turned up a very low number of deer bones in the assemblages.³⁴ In Boston, however, it can be stated that they were not eating much venison in the seventeenth century. The Boston Cross Street privy archaeological site 4 shows that deer were an extremely rare element in the colonists' diet at any time during the mid to late seventeenth century.³⁵

But the forest fringe environment around English settlement was in for a dramatic change. The abundant deer and lots of other species were going to be driven away from settled areas by the behaviors of one of the most prolific and environment altering of the new residents-- the pig.

Boston Cross Street Privy Site

PERCENTAGE OF TOTAL BIOMASS BY SUBPHASE

	Subphase							
	<u>I-2^a</u>	<u>I-5^a</u>	<u>I-8</u>	<u>I-10</u>	<u>II-1</u>	<u>II-2</u>	<u>II-3</u>	<u>III</u>
<i>Bos taurus</i> (Domestic Cow)	59.5	74.3	65.8	63.2	59.5	67.7	34.6	47.1
<i>Ovis aries</i> / <i>Capra hircus</i> (Domestic Sheep/Goat)	11.5	7.2	19.8	11.4	16.1	7.6	24.4	17.3
<i>Sus scrofa</i> (Domestic Pig)	2.0	4.3	4.5	5.4	7.8	4.3	12.1	10.5
Wild Mammals	—	—	3.4	—	—	—	—	—
Wild Birds	0.2	0.1	0.2	0.5	0.1	0.1	0.1	0.1
Domestic Birds	0.2	0.4	0.2	1.3	0.5	0.1	1.2	0.3
Fish	6.9	1.1	1.1	1.6	2.3	1.0	3.6	0.6
Commensals	—	0.1	0.1	1.2	0.1	0.1	—	—

^a Nearly complete immature pig not counted in biomass totals for Subphases I-2 and I-5.

Figure 22. Level I-2 a at +/-1650 to level III at +/- 1690 shows that this Boston household was not eating much deer except for a short time in the 1670's.

VI. THE PIG IN THE PROMISED LAND:

The pig species, *sus scrofa*, played an important role in the European settlement of the New World. The Vikings had swine with them for the initial phase of colonization in Greenland.¹ The English settlers seem to have followed the same practice. Pigs were an environment altering force in the forests of New England. It is not recorded when pigs were first set loose in Plymouth but a few of them may have been passengers on the Mayflower.

Edward Winslow specifically leaves swine and chickens out of the 1621 list of animals needed by Plymouth when he wrote "if we have once but kine, horses and sheep, I make no question, but men might live as contented here as any part of the world." William Bradford noted the arrival of cows "the first of that kind of cattle [perhaps implying that other kinds were already there- goats, pigs and even chickens]"² in Plymouth. By 1624 there were "many swine."³ The Massachusetts Bay Colony is also described as having swine aplenty. In 1629 Francis Higginson wrote from Salem that "it is scarce to be believed how kine and goats, horses and hoggs do prosper here."⁴

One early Puritan settler wrote in 1631 that "the best cattle for profit is swine."⁵ First person accounts of early settlement mention there being "many swine" and there are extensive accounts of pig behaviors that have become problems. There are records of votes at town meeting to hire at public expense one or two keepers for town herds.⁶ The English medieval pig husbandry practice of a keeper watching the town's hogs seems to have prevailed in some areas, but there still seem to be a lot of pigs running around, based on the number of complaints mentioned in the Massachusetts General Court records. Swine are noted to be allowed to range free in the woods from November to

March.⁷ Some towns kept records of pig earmarks. Each family would have a distinctive notch that was cut into the ears of the pigs that they owned so that they could be identified by their owners.⁸

This onslaught of prolific swine had to have made an impact on the ecology of the newly settled areas of Massachusetts. What did they do when they were turned loose in the new world? Like their human owners, seventeenth-century pigs seem familiar because of their modern descendants, but may have actually been as different from them as we are from our seventeenth century ancestors.

What happens when pigs run free? Swinely characteristics and behaviors that might have been both understood and exploited by seventeenth-century pig owners are difficult to find in the modern domesticated pig's caged world. However, by looking at those few places where pigs now live freer lives, some important behaviors become evident.

The twenty-first century world has not caged every pig. There are still areas where pigs roam free. It was possible to find a modern body of information about these pigs and study it for characteristics and behaviors that might be useful when applied to the past. One of the best sources of information is the experience of those who have had to deal with feral pig populations.

Modern California has a pig problem. Various inhabitants including park rangers, wildlife control officers, environmentalists, golf course supervisors and the occasional Buddhist monk are battling this "wily" pig foe. That state's ever enlarging feral pig population was pegged at 135,000 and rising in 1999. At Pinnacles National Monument in San Benito County, for example, a walk through any part of the 24,000 acre park was

described as similar to what one would experience when "an army of roto-tillers had run amok." This is the work of rooting pigs, the park has built a thirty mile long fence at a cost of \$40,000 per mile to get rid of them.⁹

California's wild pigs are the direct descendents of a set that was intentionally let loose in the region in 1925 for sport hunting. Once loose, they became enamored with a few escaped domestic pigs and founded a genetically mixed herd. Even before the interest in pig hunting waned, this population was well established and since then it has skyrocketed. When hunting was re-introduced as a control mechanism in the late twentieth century, it was found that these pigs were more than a match for even the fiercest of hunting dogs.¹⁰

In California, the pigs have adversely affected many native species, including deer, squirrels, quail and other birds, by out-competing with them for food, especially acorns. Joe DiDonato, a wildlife program manager for California's East Bay Regional Parks, notes "we have been looking at the stomach contents of some of the pigs that we have killed and we find them stuffed with acorns. When we go out looking for acorns in the wild we cannot find any at all on the ground." This sentiment is echoed by Cody Stember, a professional trapper with the U.S. Department of Agriculture, who wrote "they're out there right now sucking up acorns like a vacuum. They'll run the deer away."¹¹

A recent study of the home range movements and habitat of these wild swine found that pigs will root anywhere but tend to root the most under oak trees. They tend to have a "home" territory which is not overly large. In California this rooting behavior is called "pig plowing." These pigs prefer dense brush or marshy vegetation. Because they

don't have a sweat mechanism for body cooling, during periods of hot weather pigs spend a good deal of time wallowing in ponds, springs or streams, usually in or adjacent to cover (the Buddhist monks awoke one morning to find a tranquil reflective meditation pool with a carefully [and costly] contrived landscaping of imported flora had been transformed into a mud filled wallow.) Pigs will eat anything from grain to carrion. There are anecdotal reports that pigs will attack and eat unattended fawns.

From this information some behaviors that may have been important in seventeenth-century pigs can be delineated. The preferred food for the pig is the acorn which was usually abundant in early Massachusetts. Pigs are territorial and their territories tend to be concentric rings around oak trees or other food sources. In a given geographic area they will out-compete native species for food resources, most notably deer and birds. They can handle the attack of a fierce hunting dog (roughly analogous to a wolf). Their rooting behavior, called pig plowing in California, can rapidly denude an area of vegetation bringing the ground down to bare soil. When this behavior, useful at first to clear land, became undesirable, it was dealt with by the seventeenth-century practice of putting a ring through pigs' noses.

While pigs were not a favorite subject for artists, some depictions from early time periods exist both in Europe and in the United States. Based on those depictions, seventeenth-century pigs seem phenotypically somewhat similar to the feral mixed breed pigs in California. Described as "little brown striped watermelons" that squeal, California's new born piglets may be living reflections of the earliest *sus scrofa* inhabitants of Massachusetts.¹²

In the *Good Cheape Husbandries*, written in 1614 by Gervase Markham, he suggests that the best British swine should have a "thicke neck, a short and strong groyne, and a thick chine well set with strong bristles. The colour is best which is all of one peece, as all white or all sanded, the pyed are the worst and most apt to take the mesles, the black is tolerable." Pig disposition is described as being "greedy, given much to root up grounds, and teare down fences, he is very lecherous. he is subject to much anger." Markham states that in England swine were fed in the morning and then brought out by either families or a hogmaster to root in either old fields, marshes, or (in the fall and winter) to the forests for mast during the day and then returned to the safety of the sty at night.¹³

A study of both modern and historic farming practices in England reveals another important fact related to the pig's preference for acorns. In *500 Points of Husbandrie*, a poem written by Thomas Tusser in 1577, the following lines can be found:

To gather some mast, it shall stand mee upon
With servant and children, yer mast be all gone:
Some left amongst bushes, shall pleasure thy swine,
For fear of mischief, keep acorns from kine.¹⁴

A mature Oak tree can produce up to 700 pounds of acorns in a good year.¹⁵ Gathering mast required all available hands to work at that time including those of servant and child. The verse suggests leaving some acorns on the ground for swine. It adds the important fact to keep acorns from cows. The pig and the goat, another animal abundantly represented in very early seventeenth-century Massachusetts, can digest acorns. But when they are eaten by either cows or horses, acorn poisoning can cause sickness and death.¹⁶ Knowledge of this fact would have been important in 1692 because English settlement had reached to the edge of the wilderness in the "Frontier" town of

Andover and in Salem Village. Cramped for pasturage, both horses and cows were being kept "in the woods."¹⁷ Knowledge of this fact is at the root of the modern English practice of "pannage." Using an archaic sounding set of calendar dates straight out of the medieval mind, this period traditionally lasts from September 29 to fifteen days before Easter hocktide, but may be changed to allow for weather fluctuations. During this time English farmers may assert their "right to mast." Pigs are allowed onto open forest land to eat acorns and, by so doing, keep cows, ponies and horses that graze there in warmer weather free from acorn poisoning.¹⁸

The "pig plowing" that is happening in California today shows that swine are a good land clearing animal. Centuries of pig domestication in England made Massachusetts's English settlers well aware of this pig habit. Plowing pigs could clear land without requiring any intensive human effort. While one was occasionally

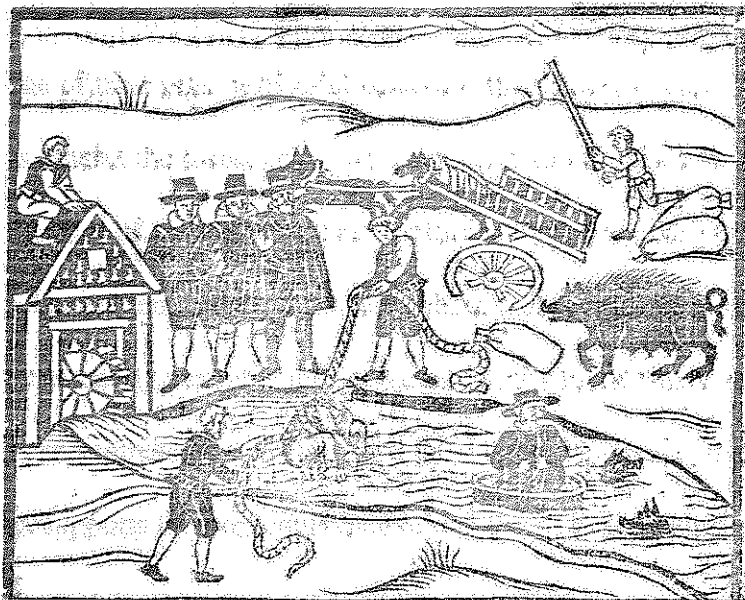


Figure 24. A seventeenth century depiction of a pig: English print of "swimming a witch" from 1613. Note that the sow depicted on the right still has "wild" phenotype. In collection of The Huntington Library, San Marino, California.



Figure 25. Fourteenth century depiction of pigs, with their keeper, eating acorns during pannage. Note wild characteristics. *November* from *Hours of Jeanne d'Evreux* by Jean Pucelle 1325-28. In the Cloisters collection of The Metropolitan Museum of Art, New York.

mentioned as being killed by a wolf,¹⁹ it is an infrequent notation. By using the knowledge of their pigs' territorial behavior, the settlers in early Massachusetts could let their pigs inhabit the forest edges with little concern that they would run away. The territorial sense of "home" could be reinforced by the regular presentation of food and garbage. The settlers had little to worry about maintaining their pig's food as acorns (mast) were readily available in the Massachusetts forest that was consistently described as being predominated by oak trees. Archaeological evidence, in the form of pollen core sampling, supports the preponderance of oak trees in Massachusetts seventeenth century forests.²⁰

Because of their territoriality, in years when acorn production was diminished, the local swine became garden pests, exploiting weak areas in their owners' fencing. John

Winthrop wrote in September of 1633, "There was great scarcity of corn by reason of the spoyle our hoggs had made at harvest and the great quantity they had eaten in the winter (there being no acorns.)"²¹ This seems to support both the need for strong fences to keep out pigs, laws requiring pigs to be "yoked," the territoriality of the local swine, and the possible collection, as recommended by Thomas Tusser, of acorns for winter feeding. The "gathering of akrons" is mentioned in a November 5, 1639 Massachusetts General Court order regulating outdoor 'fyers.'²² A lack of acorns led to the substitution of corn.

Following standard Middle Age practice, outside gardens were fenced. By 1624, Plymouth, had "gardens encased in clapboards" and "the towne" according to John Smith "is impaled."²³ Swine, wolves, French ne'er-do-wells and hostile natives were kept at bay in this controlled manner. This was a lingering medieval farming strategy. Early English archaeological sites often show a round fence, ditch, or dirt wall thrown around an entire site for both defense and animal control. The initial choice of terrain, especially in the case of both Plymouth and Boston, corresponds to the ancient English hill fort settlement pattern.²⁴

Seventeenth-century swine husbandry practices had the direct result of creating an expanding Pig Rooted Zone (hereafter PRZ) of ground around any site occupied by their human owners. By regularly offering food to their pigs in a certain area owners insured that a point became the center of the animal's home territory. If the food that was offered included collected acorns, a farm or town itself might have been synonymous to a well producing oak tree in the seventeenth-century pig's mental landscape.

John Winthrop, Jr., in a 1664 letter to his friend Lion Gardner of Long Island, recommended this pig plowing mechanism as having been successfully used in the

Massachusetts Bay Colony. He wrote that pigs and goats "closely grub the earth," which once cleared in this manner could then be planted with "proper English grass to create a meadow for cattle, horses or sheep."²⁵

Similar to the impacts observed in California, an immediate effect of this PRZ would have been the diminishment of a preferred food source for at least five species indigenous to seventeenth-century Massachusetts: the now extinct migratory passenger pigeons, wild turkeys, white footed mice, and white tailed deer.²⁶ *Ixodes scapularis* tick populations would have been affected by the constant stirring and rapid decomposition of leaf litter caused by rooting pigs. While mice, deer, and ticks seem to have had co-existed in the oak forests of New England for eons, co-existence with pigs would have been at best difficult. A mouse with any sense whatsoever would not take on a large sharp toothed pig to get an acorn. Even a deer would have stepped aside for these nasty new acorn consumers. Since wild pigs have been observed to kill and devour fawns in California, this behavior may also have had some effect on a local deer population. As is happening in California, mice, birds, and deer would have been driven away. To survive, indigenous species would have retreated outside the PRZ. This competition may have been partially responsible for William Bradford's statement that wild fowl "is not as plentiful hereabouts as it once was."²⁷

The now extinct but once prolific passenger pigeons, so numerous that that they blackened the skies as they flew over, ate acorns. On September 26, 1633, William Hammond of Massachusetts Bay Colony wrote "when it is an acorn year here our pigeons abound."²⁸ Pig related acorn depletion may have been the first nail in that specie's coffin of extinction, followed by an apparent seventeenth-century consumption

level that was equal to the domesticated chicken, at least in Boston.²⁹ In California, a distant relative, the Bandtail Pigeon, still gorges on acorns before migrating south to Mexico for the winter and is considered threatened by pig acorn depletion.³⁰

Acorns are low in protein content but very high in fat and carbohydrates. Birds, deer and pigs who ingest large quantities of acorns in the fall gain weight and bulk up for the winter ahead. Studies have shown that deer can gain weight rapidly in just two weeks of this acorn gorging. Birds gain strength, body mass and energy for their long migratory flights.³¹ Humans in early New England exploited this acorn driven pre-winter weight gain in their pigs by turning late Fall into slaughter time, usurping the added body mass for their own dinner tables. This custom follows the English November through Shrovetide (late February) slaughtering pattern.³²

New England's tick populations are sensitive to any movement, growth or degradation in the white tailed deer population. Pig driven movement of deer would have also moved ticks. When the deer around English settlements were crowded out by the resident pigs, any attached deer ticks would simply have moved along with them. Those left behind would not have flourished. A leaf litter layer in a PRZ was not a protective feature, especially in cold weather. It was constantly being churned up. Tick eggs would have been susceptible to being exposed to below freezing temperatures if they were churned to the surface during the winter.

After the initial phase of sickness at contact, records show that the English populations in early Plymouth and Massachusetts became relatively healthy. While human health became fairly robust, Massachusetts's pigs were not so lucky. Pigs were

constantly "at risk" for Lyme disease because they occupied almost exclusively the geography of risk for Lyme disease--between the forest and cleared land.

This underscores a critically important function for the PRZ as a biological sanitizer.

Pigs on the perimeter of human occupation zones were in a position to carry the brunt of possible Lyme infection. They would live a lifecycle shortened by annual slaughter, which would have the effect of limiting the ongoing spread of tick borne disease. If the pork from an infected pig was cooked to a temperature above 160 degrees F. the Lyme bacteria was killed, which ended any further transmission of the disease.³³

This PRZ function may be corroborated by the bones of some of the earliest pigs that have been found in Massachusetts. During archeological work done in association with Boston's "Big Dig," 371 bones representing the heads, bodies and feet of seventeenth century pigs were found, as well as a nearly complete fetal pig carcass. One of the most important findings in these pig remains was evidence of fairly widespread pathologies. One bone, a scapula, contains massive exostoses (pitting from infection) around the periphery of the glenoid (socket); there also appears to be an ankylosis (joint fusion) evidenced by a rough and pitted surface on the glenoid that has extended beyond the original surface.

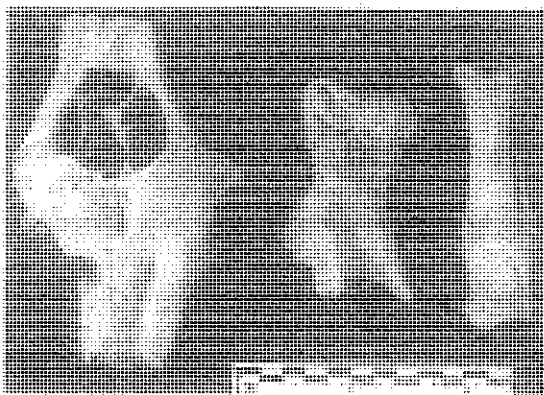


Figure 26. Evidence of disease in pig remains: a. trephined skull; b. exostoses on scapula; c. swelling on shaft of tibia, from The Cross Street Back Lot Privy. *Historical Archaeology* Vol.32, No. 3, 77.

The most interesting pathology is from an immature pig skeleton. This appears to be a fetus that would have been described as being “cast off” or aborted in the seventeenth century. The skeleton had a general swelling on the distal half of a tibia which may have been the result of an infection of the bone via an infection in the blood stream. Lodging in the Haversian system (bone marrow and other parts of the bone), especially in a young animal, such infections can cause death.

All of these pathologies are consistent with a Lyme disease diagnosis for these pigs. They seem to indicate that these animals (including the fetal pig’s mother) were active participants in the PRZ process. By occupying the edges of the English cultural zones, seventeenth-century pigs may have carried the brunt of Lyme disease infection and kept humans healthy.³⁴

In seventeenth-century Massachusetts, the English and their domesticated animals exploited land that had been cleared first by Native Americans, and then by pigs, of brush, undergrowth, acorns, deer, mice, innumerable insects whose life cycle included an

underground grub stage, and ticks. An ongoing cycle would be repeated as a new generation of pigs ventured further afield in an enlarging zone of pig and human occupation. When combined with the English settler's continuation of the medieval custom of "burning" fallen leaves and undergrowth, the bio-sanitizing system seems to have worked very well.

The Native American residents of Massachusetts' version of the PRZ was their collection of acorns to use as food and their protective slash and burn agricultural system. This ridded the area where they lived of not only underbrush but acorn eating mice and insect vermin as well. This system, if repeated regularly, also prevented endemic Lyme disease from ever reaching epidemic proportions.³⁵

This healthy cycle began to break down when the Native American population was decimated, English settlement begun to fragment the forests, and when once desirable pig behaviors became a nuisance that had to be controlled. This happened in stages. There are innumerable seventeenth century Massachusetts Bay Colony ordinances related to swine. The Massachusetts General Court tried many strategies to respond to an onslaught of complaints. They told complaining Native Americans to build fences. They tried fining the owners of wayward pigs. They tried fining people who complained about the Hog Laws. They appointed "Hogg reeves" to chase after pigs. They banned fish stealing pigs from the beaches near fishing platforms. They tried impounding pigs. They allowed pigs found in cornfields to be shot. They tried nose rings and yokes that enlarged the neck size of the pig to make it harder for them to exploit the weak areas of fences. By the late seventeenth century, rings and yokes were legally mandated in most areas.

Gradually, and by legal mandate, domestic swine were relegated to the fenced farmyard. By the 1690's pigs are mentioned as sometimes being confined within fences. Pregnant sows seem to have given birth within the confines of their owner's barnyard and owners were well aware of the number of off-spring and the health of their animals. One seventeenth century witchcraft deponent verifies that pigs have begun to be caged when he testifies that his bewitchment consisted of being thrown into a neighbor's sty.³⁶

Once the pigs were brought in from the forest for good, when there was a high population of deer, Lyme disease was still being controlled by prevailing cultural practices: acorn collection as hog food, the selective extraction of oak trees, and the burning of fallen leaves and undergrowth. By the mid twentieth century, New England's farming had declined and many previously cleared fields once again underwent the natural process of reforestation. Acorns were replaced by corn as the major feed for hogs. This left the new forest's floor littered with mast for birds, mice, and especially deer. The decline and regulation of recreational hunting led to a concurrent dramatic increase in the number of white tailed deer in those reforested woods.³⁷ In 1970, the Federal Clean Air Act suggested that states ban the yearly burning of fallen leaves to preserve air quality.³⁸ This removed the last existing control for tick populations. It immediately and dramatically expanded the over-wintering habitat for *Ixodes scapularis*. Portions of New England's burgeoning suburban population were poised on the brink of a new epidemic of Lyme disease that, if not for the availability of antibiotics, might have rivaled the "great sicknesses" of the early seventeenth century.

VII. WHAT HAPPENED IN 1692?

In seventeenth-century Massachusetts the landscape gradually developed an overlay of the physical, social, and cultural mindset of the new English settlers, first filling in land that had previously been cleared by a deceased Native population. Any attempt to give population figures for these colonies during the seventeenth century is an estimate. The number of settlers who immigrated to the Plymouth Colony is estimated to be a total of three hundred sixty two.¹ The estimates for migration into the Massachusetts Bay Colony between 1628 and 1640 (when it came to an abrupt end because of legal mandates in England) vary widely from 10,000 to 20,000. The colonies in New Hampshire and Connecticut were also largely populated from this "great migration."²

The estimates of the pre-disease Native American population for this same land area also vary widely. Daniel Gookin, writing during the seventeenth century, sets the Massachusetts tribe's pre-contact strength at 10,000 but there are questions about which groups he included or excluded in that number. Others have given estimates of 20,000 or more.³ Although Native and English land use patterns would have differed, it appears that a roughly equal number of English settlers may have replaced a similar number of deceased Natives on the same land. Since Native activity and land use often included using land until it was "worn out" and then moving on by clearing new fields for cultivation, it is quite likely that the English found fairly extensive amounts of cleared land to settle before they took to the forest in force.

It is highly likely that after an initial period of exploration, clearance of accumulated brush and undergrowth on the sites chosen for habitation, and building of homes and fences, the first colonists chose to interact infrequently with the forests. They

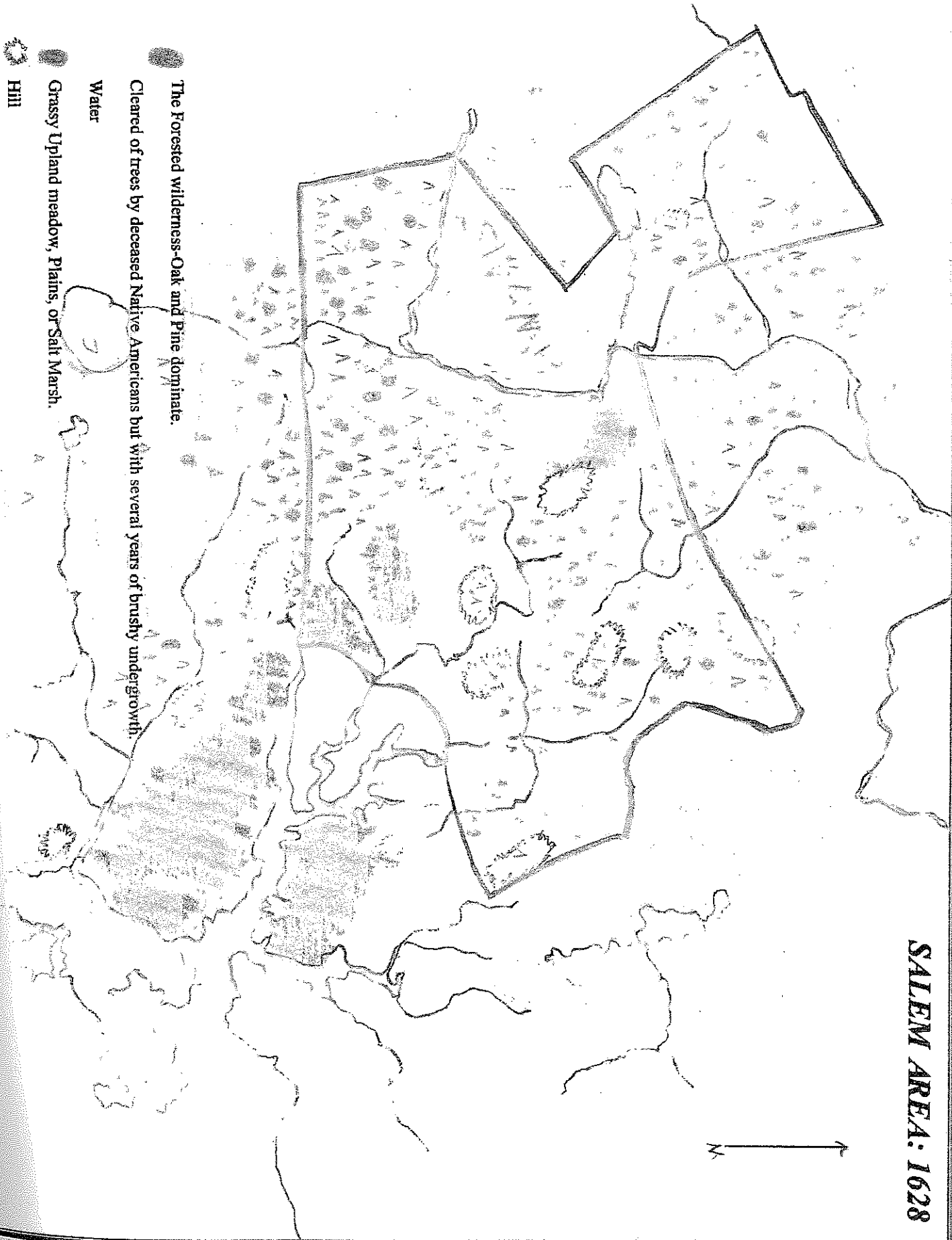
preferred to settle on land that was already cleared of trees, and avoided the dangers of wolves, hostile Native Americans, or getting lost that the wilderness represented. The business of the early seventeenth-century settler lay in cultivated places on the very fringe of the forest, sometimes selectively extracting pine and oak trees to use as timber for houses and for shipbuilding. This created a dispersed group of settlements in New England that tended to be along rivers with dense forest in between. Waterways were the great highways of seventeenth-century Massachusetts. Looking for good cleared land, the first group of settlers that went from Massachusetts to Connecticut went by sea. Travel between Plymouth and Boston was by water. Travel between Boston and Salem was often by water. One early observer commented on the frequent use of canoes for transportation in the Salem area, writing "there be more canoes in this town than in the whole patent, every household having a water horse or two."⁴ The typical early seventeenth century New England town either fronted the ocean or faced in on itself with a river nearby.

Because the English came from a land cramped island and practiced a system of manuring or "dressing" fields to promote continual fertility, along with a rotating set of pastures for horses and cows, even by the end of the "great migration" the English had probably not even begun to occupy all the cleared spaces on the map or intrude extensively into the forest yet. But by the 1650's and 60's the population on the outer edges of the Massachusetts Bay Colony began to have contact with the wilderness; forced by the need for more lumber, fields for crops and pasturage, clearing and fragmenting the forest in the Salem Village area, and intruding into the forest that ringed the frontier settlements of Andover and Billerica. The shipbuilding industry needed oak for timber

and pine for masts. Fledgling iron and copper works burned prodigious amounts of charcoal to process local metals.⁵ Potash and firewood were needed on a daily basis for cooking, soap making, and tanning leather.⁶

Historic Salem Village was located on the land that is now the modern town of Danvers, outside the town of Salem. Like Salem itself, the village was built partially on land that had been cleared by Native Americans. This area, called Naumkeag, was described by John Smith as having "a multitude of people." He wrote that the "sea coast as you passe, shewes you all along large corne fields, and great troupes of well proportioned people."⁷ By 1628 the multitude of well proportioned Native Americans were dead, all but eliminated in waves of plague and pestilence that had occurred before the first English settlers even arrived. At contact, the peninsula that is now Salem, the Northfields area, and a few outlying spots in Salem Village were clear of trees.⁸ The first English settlers in the area were a combination of the two hundred member Higginson Fleet that arrived in 1628 and about fifty men, including John Endicott and Roger Conant, who moved down from a fishing outpost that had been located earlier on Cape Ann.⁹ Francis Higginson described the landscape at contact. "Though all the countrey be as it were a thicke wood for the general, yet in divers places there is much ground cleared by the Indians and especially about the plantation." The soil was "in other places clay... in other gravel, in other sandy [as it is about our plantation at Salem]."¹⁰ The thick wood that Higginson noted, as is shown from pollen core sampling, was dominated by oak and pine trees. The site of Salem was chosen because it had access to the water, a good harbor, and was defensible against attack. A fort was built to protect the entrance to the

SALEM AREA: 1628



town on the land side, and during King Phillip's War a palisade was added, stretching across the base of the peninsula as protection from Native American attacks. While the land on the peninsula was not especially fertile, it was used for small gardens in the immediate vicinity of the first English houses. The mainland adjacent to the Salem peninsula featured salt and fresh marshes that were used almost immediately for the common grazing of cattle. This common land was called the Northfields. Beyond this zone, outside town, in what was to become the east side of Salem Village, there was an especially fertile plain. A few Native paths traversed the area and would become over time the roads that were used by the English settlers to travel between Ipswich and points to the north and Boston to the south.¹¹

Several land grants for the area outside town were made beginning in the 1630's. The first, in 1632, was for three hundred acres to Governor John Endicott for what became Orchard Farm. Another two hundred acres were later added to the grant in exchange for improvements to be made to the Ipswich Road. Land in the adjacent area was parceled out to several different settlers.¹² In 1639 the General Court of Massachusetts Bay Colony defined the outer limits of the village by stating that "wheras the inhabitants of Salem have agreed to plant a village near the river which runs to Ipswich, it is ordered that all the lands near their bounds, between Salem and the said river, not belonging to any other town or person by any former grant, shall belong to said village."¹³

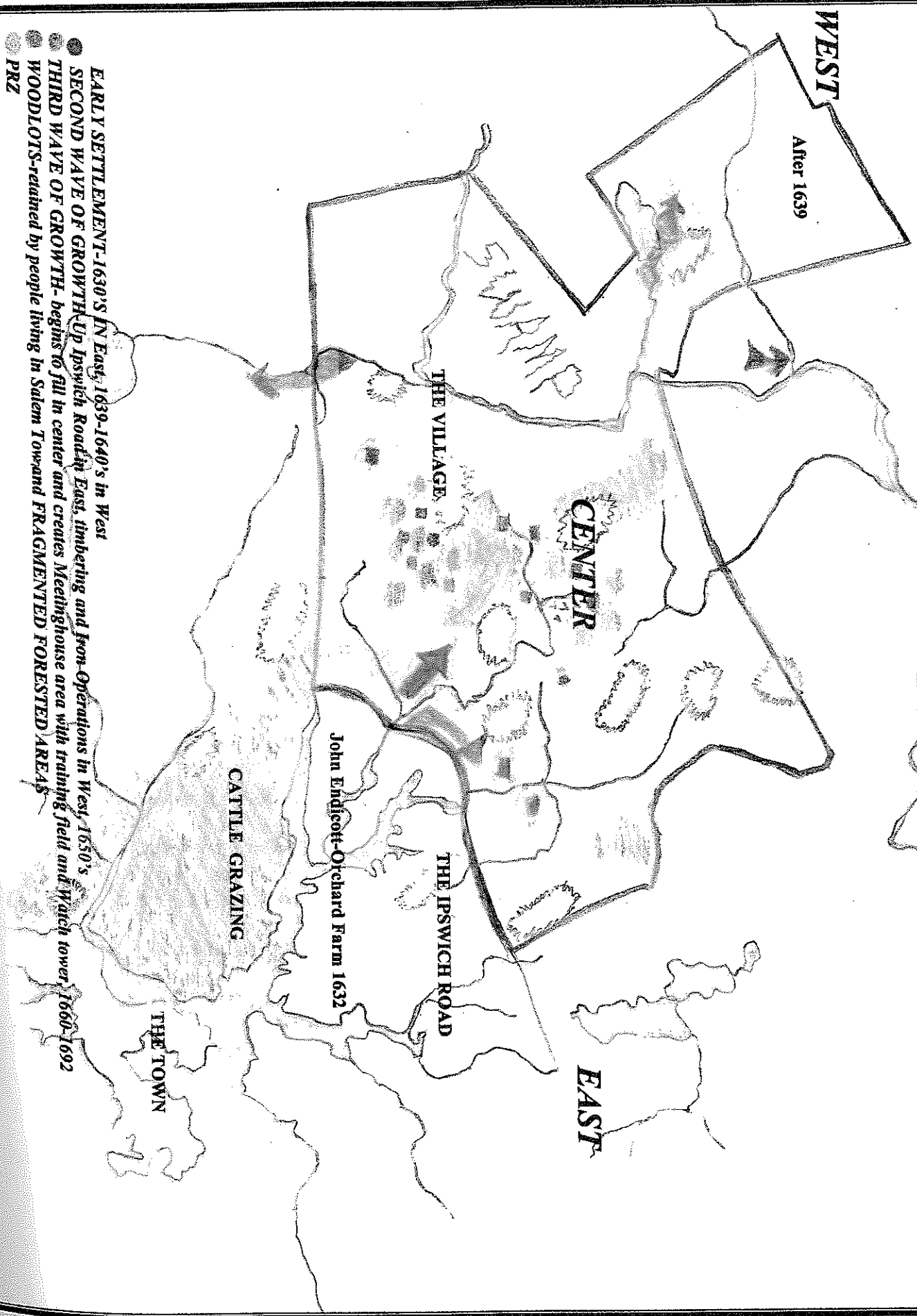
Immediately afterwards, grants were given for land on the Ipswich River, described as "a hill, an Indian plantation, and a pond" on the western side of the forest

that included "about one hundred or one hundred and fifty acres of meadow." Thomas and Nathaniel Putnam also bought one hundred and forty acres of upland and fourteen acres of meadow in this western zone. Bray Wilkins also bought land in this area in 1661.¹⁴

The village grew from these two areas-in from the east near Orchard Farm and then along the Ipswich Road, and also inward from the western Ipswich River edge towards the east. The wooded center of the village only began to fill in during the 1660's and 1670's in a splotchy crazy quilt development pattern that was created by the English land ownership system. As time went by, large land grants were divided up between the descendents of the original owners or pieces were sold. Farms were interspersed with woodlots, meadows, and various industries. In 1667 the village was described as a group of "houses distant from each other, some ten miles, some eight or nine miles from the town of Salem." The distance of the houses, one from another were "some a mile some further-that it is difficult sending a neighbor to another in dark nights in a wilderness that is so little cleared and [by] ways so unpassable." This wooded wilderness was a dangerous place, especially "considering what dreadful examples former times hath afforded in that respect, in this country, from Indians... in the night season."¹⁵

Service occupations like a doctor, a plate turner, the owners of public houses and the grist miller clustered near the eastern zone, especially along the Ipswich Road. Other, more forest consuming activities occupied residents of the Salem village and Andover areas. The Putnam family started an iron processing operation was located north of the village. Governor Endicott owned a copper mine and processing facility somewhere on

SALEM AREA growth and land development: 1628-1692



- EARLY SETTLEMENT-1630'S IN East, 1639-1640's in West
- SECOND WAVE OF GROWTH-Up Ipswich Road in East, timbering and Iron Operations in West, 1650's
- THIRD WAVE OF GROWTH- begins to fill in center and creates Meetinghouse area with training field and Watch tower, 1660-1692
- WOODLOTS-retained by people living in Salem Town and FRAGMENTED FORESTED AREAS
- PRZ

his land that processed eight tons of ore per week.¹⁶ Several sawmills were established along streams in the western zone to process wood.¹⁷

Deforestation began slowly, often with the selective extraction of oak. Houses, furniture, ships, barrels and fence rails were all made from this hardwood. The Endicott Orchard Farm, for example, was enclosed by a palisade that took 7,000 six inch wide trees to build.¹⁸ The iron operation set up north of town by the Putnam family would have burned five cords of wood (converted into charcoal) for each ton of iron that was produced. Another tree consuming set of iron works was set up in north of the village in Andover, on the Shawshin River, by the Chandler family.¹⁹

An acre of trees had to be cut for every two tons of potash that was produced and used as fertilizer or to create lye. Tanners, like the Parkers of Andover, and dyers, like William Shattuck of Essex Street in Salem Town, used bark for hide processing and coloring cloth and wool. The forest's tall and straight pine trees were cut for use as ship masts. Others were bled for their resin to make pitch, turpentine and other naval wares.²⁰

Cordwood went to Salem's brick kiln, lime kiln and its biscuit and bread ovens. Once homes had been built, they required prodigious amounts of wood for cooking and heating. When the Salem Village church hired its first minister, it contracted to supply him a total of thirty cords of wood for use in the new 20 by 30 ft. parish house.²¹

This deforestation, when combined with an English pattern of fixed land ownership, created a mosaic of farm, ecotone edge areas and forest fragments that can be very clearly be seen on the map. From the unwanted swamp in the southwest, a patchwork quilt of ownership and land uses spreads east across the land showing lots of

edge environments, orchard and riparian zones.²² When compared to modern maps, soil surveys and Geographical Information System vegetation surveys many aspects of this landscape were particularly likely to harbor ticks and a high risk for potential infection with Lyme disease.

The Salem Village growth pattern can be seen in some of the relict features of the Salem Village area that remain in modern Danvers. Some remaining structures represent successional growth and expansion patterns. These include the Joseph Putnam House, built in 1648, located in what was part of the west to east growth zone. The Endicott burial ground (which was a Native American camp before it became a cemetery) is in the east to west growth zone and has early graves that are from the 1650's. The Daniel Rea House (1660), the Samuel Houlton House (1670), the Joseph Holten House (1670), and the Benjamin and Sarah Houlton House (1670) are all part of a wave of growth in the center of the village. By 1671, Watch House Hill had been fortified and a militia training field was designated nearby. In 1672 Salem Village became a separate parish and a meetinghouse was built. The center of the village became more heavily occupied. A third wave of growth is represented by the house that was occupied by Rebecca and Frances Nurse, built in 1678, the Osborn House of 1680, the Haines House of 1681, the Zerubabel Endecott House of 1681, the (Parris) Parsonage of 1681, the Putnam Perry House of 1685, and the John Holten House of 1692.²³

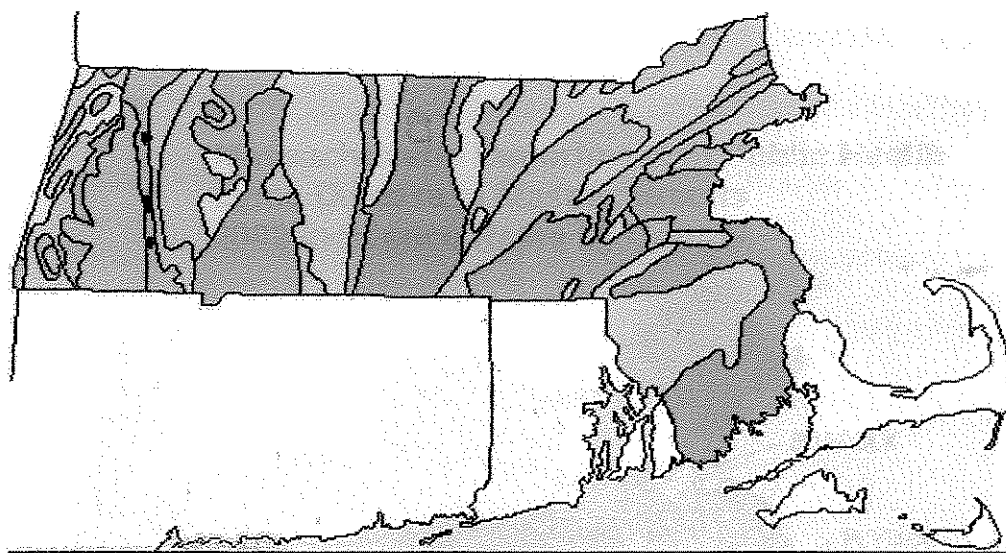
The first settlers on the great meadow at Andover, just to the north of Salem Village, and in Billerica to the west, also occupied a mosaiced landscape along the Shawshin and Ipswich river ways. In addition to grassland the area contained "woods" interspersed with grassy clearings, several hills, numerous creeks and rivers, meadows,

We now know that adult *Ixodes scapularis* (deer) ticks are brought into geographic areas primarily by birds (which can also carry immature stages of ticks into distant areas, especially during spring and fall migration) and deer, which are capable of ranging over wide areas, especially along riparian corridors. To become successfully established in an area, however, *Ixodes scapularis* ticks must have a suitable habitat for questing, molting, over- wintering, and oviposition. The vegetation, soil, topography and climate are strongly related to habitat suitability for these ticks.²⁸

Because leaf litter is a necessary component for the survival of immature stages of deer ticks, tick densities are highest in forests that are dominated by oak trees, followed by maple, and are lowest in coniferous forests.²⁹ Tick densities are also highest in areas with underlying sedimentary bedrock, which is associated with soil textures of increased particle size.³⁰

The most in-depth modern study of habitat suitability for *Ixodes scapularis* done to date is from Wisconsin. It found the highest level of ticks in deciduous forests with well drained soils dominated by the alfisol soil order category. Ticks were absent in clay soils and most abundant in sandy loam textured soil. Cambrian, Ordician, Silurian and Devonian sedimentary bedrock was found to be more likely to underlay these types of soils. Although they are found in all quaternary deposits, glacial moraine sites were most heavily populated by these ticks.³¹

This information corresponds favorably with information from both modern and historic Massachusetts. Cape Cod and the Plymouth area have a Pleistocene and Holocene sedimentary base and soil that is dominated by the sandy, well drained



Explanation

SEDIMENTARY ROCKS

 Pleistocene and Holocene sediments

 Ordovician sedimentary rocks

 Cambrian and Ordovician carbonates

 Cambrian clastics

IGNEOUS ROCKS

 Triassic volcanics

 Paleozoic mafic intrusives

 Middle Paleozoic granites

 Ordovician volcanics & metasediments


 Lower Paleozoic granites

 Precambrian granites

 ultramafic rocks

METAMORPHIC ROCKS

 Pennsylvanian metasediments

 Devonian metasediments

 Silurian and Devonian metasediments

 Silurian metasediments

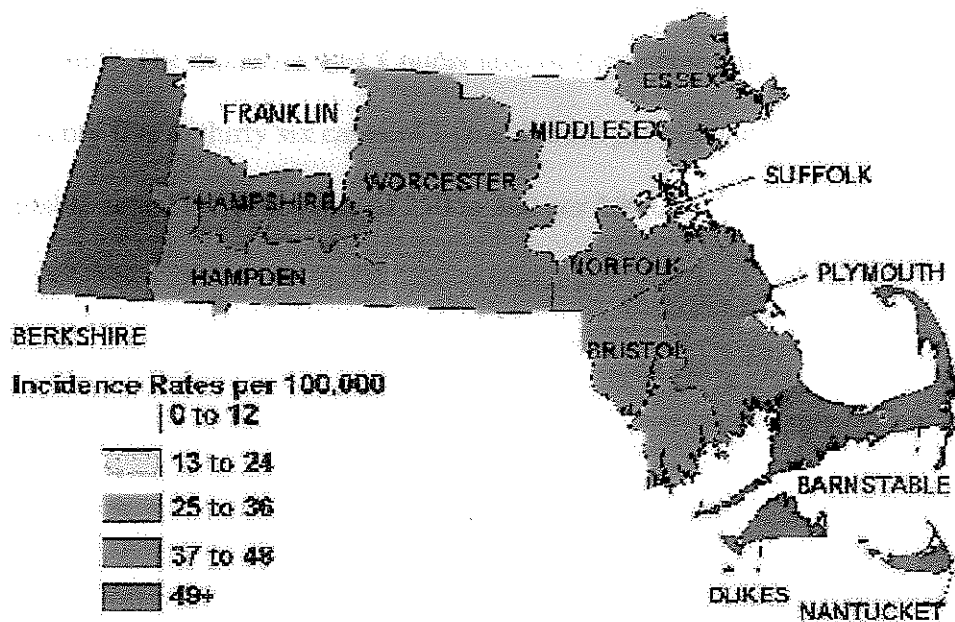
 Cambrian metasediments

 Precambrian gneiss and schist

Map 3. GEOLOGIC MAP OF MASSACHUSETTS. State of Massachusetts Public Information

Carver category. It takes thousands of years for significant changes to occur in the soil itself. The modern soil map, except for areas of urban disturbance, is comparable to the

**Massachusetts Department of Public Health
Lyme Disease in Massachusetts
2002 Incidence Rates by County**



Map 4. Massachusetts Department of Public Health.

soil map from the seventeenth century. The formation of soils is a continuing process and generally takes several thousands of years for significant changes to occur. The soil on Cape Cod is estimated be between 5,000 and 15,000 years old.³² Bog core pollen samples from Cape Cod show an oak/ scrub pine forest for 1620.³³ This corresponds to the written descriptions from both Bradford and Winslow. Forest edged environments are also described as well as a Native American altered landscape of forest and open patches. Cape Cod had some of the highest modern rates for Lyme infection in 2002. (See map above)

The Salem and Salem Village area has a more mixed bedrock consisting of granites and Silurian metasediments with a variety of sandy loam soils. Pollen analyses from Boston's Big Dig show a late seventeenth century forest dominated by oaks and pines with very few maple trees.³⁴ The map of property divisions for Salem reveals a mosaiced landscape with probable forest fragmentation, edge environments, and riparian corridors.³⁵ The modern Essex County area of Massachusetts had a reported infection rate of twenty five to thirty six cases of Lyme disease per population of 100,000 with a probable actual incidence rate of 250 to 360 per 100,000.³⁶

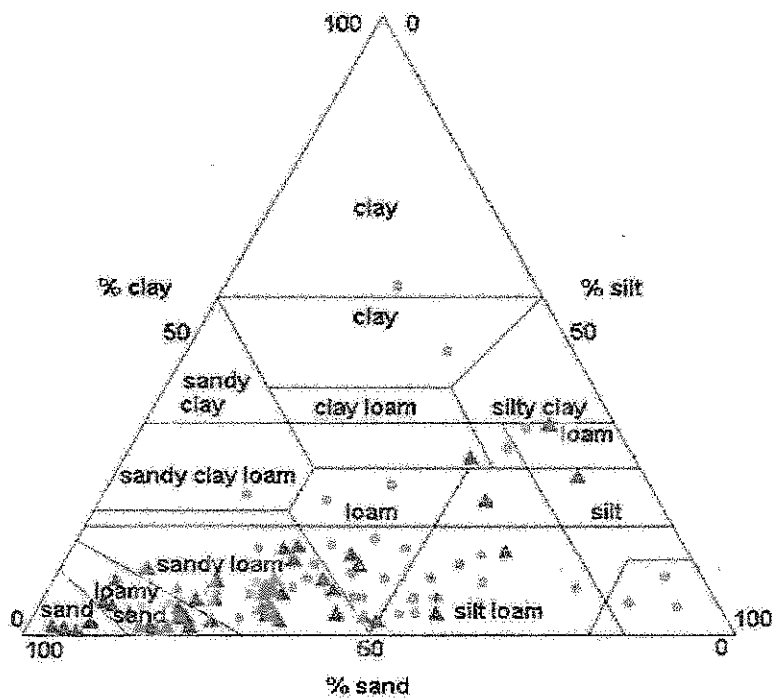


Figure 27. Soil Texture study from Wisconsin shows that *Ixodes scapularis* prefer sandy soils. Red triangle indicates sites with ticks present, green circle denote site with no ticks present. From "Predicting the Risk of Lyme Disease: Habitat Suitability for *Ixodes scapularis* in the North Central United States" in *Emerging Infectious Diseases* Vol. 8, No. 3, March 2002.

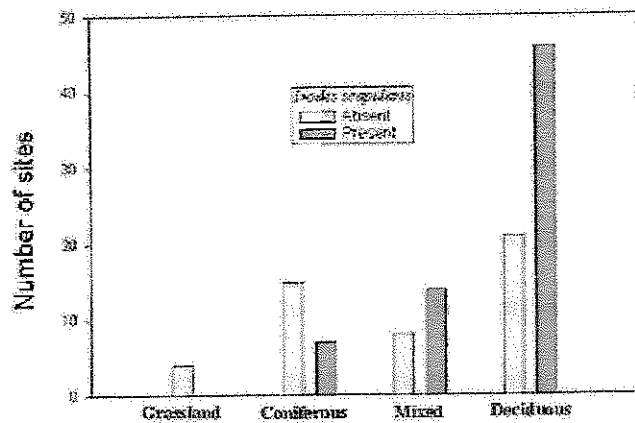


Figure 28. Land Cover categories and number of sites with *Ixodes scapularis* present and absent. Ticks favor deciduous forest. From "Predicting the Risk of Lyme Disease: Habitat Suitability for *Ixodes scapularis* in the North Central United States" in *Emerging Infectious Diseases* Vol. 8, No. 3, March 2002.

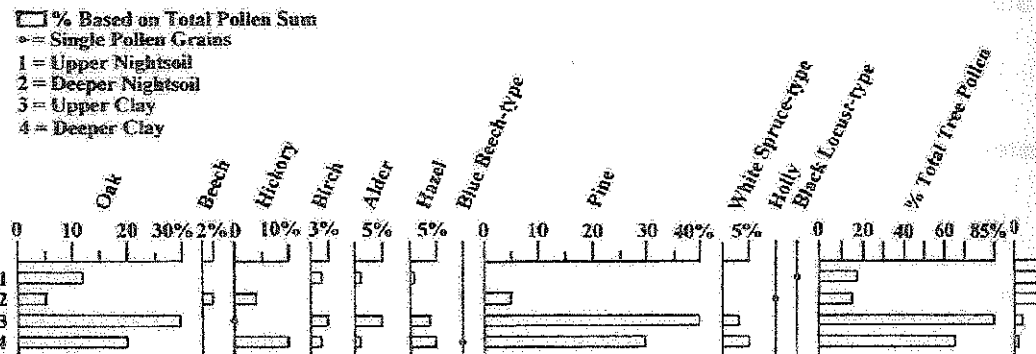


Figure 29. Feature 4 Boston privy vault exploratory core pollen percentages. Lower level 4 is +/- 1650 and upper level 1 is +/- 1690. It shows a forest dominated by an Oak/Pine mix and an interesting absence of Pine pollen in the upper most level. Has the pines been extensively harvested for naval wares by 1692, leaving an almost totally deciduous forest for a period? From Gerald Kelso "Pollen Analysis of the Feature 4 Privy at the Cross Street Back Lot Site, Boston, Massachusetts" in *Historical Archeology* Volume 32, No. 3, 1998.

One of the most useful tools available for modern Lyme disease epidemiologists has been the development of the Geographic Information System (hereafter GIS). Using a

Landsat Thematic Mapper, various elements can be factored into satellite geological system generated risk maps to predict the presence of the optimum tick vectors.³⁷

This has been done very effectively in Westchester County, New York, a Lyme disease endemic area. Information from the ground, developed through drag sampling and canine serology reports was compared and correlated with the satellite generated GIS map data for risk. In a study of 337 residential properties, high risk properties consistently tested as being both significantly greener than low risk properties. High risk properties appeared to contain a greater proportion of broadleaf trees, while low risk properties had more non-vegetative cover and open lawn.³⁸

The model that was developed was then tested further by comparing it with data about human infection that was obtained through a random questionnaire that was sent out to property owners in the area. By comparing predicted with observed (questionnaire responses) data, a 71% accuracy was found for the remote sensory prediction of risk.³⁹

Another study, also done in Westchester County, used canine seroprevalence rates to analyze the effect of residential adjacency to forest on Lyme disease risk. This study found that the rate of Lyme infected dogs was positively correlated with living adjacent to the forest.⁴⁰

A study done in Maryland used ground based zip codes to map annual risk for Lyme disease. It found that the greatest risk areas for actual infection occurred along green riparian features, especially along the watershed areas for Gunpowder River and Deer Creek near Baltimore. This was well correlated with a GIS assessment for the area.⁴¹

It is possible to use some of the information obtained in these modern studies to generally evaluate landscapes from the past. Several historic maps can be used for an analysis of Lyme disease risk in seventeenth-century Massachusetts. A map of Cape Cod 1620-26 with routes traveled can be imposed on a soil survey map.⁴² William Bradford and Edward Winslow gave on the ground and day by day accounts of the Pilgrims initial interaction with the Cape Cod landscape can be correlated with this map. For an examination of the farm layouts of the Salem Village witchcraft hysteria, the series of maps that were adapted from both the Charles Upham map of Salem Village in 1692 and the Sidney Perley Map of Salem in 1700 can be used.⁴³

Obviously, a satellite generated map of the seventeenth-century Massachusetts landscape is not available. The University of Virginia is in the process of creating a GIS examination of Salem Village and the surrounding area. Although certain features, most importantly waterways, have surely altered position in the past 300 years, there are some historic features that have survived to the present day. These include five houses, the known site of the Parris parsonage foundation, Wills Hill, Humphreys Pond Island, and Wenham Lake. By aligning these features with their placement on Upham's map, an analytical comparison was made.

This analysis found that there were some inaccuracies in the placement of the known still extant feature points on Upham's map, ranging from 50.387 meters off mark for the Edward Bishop House to 221.724 meters for Will's Hill. It found that comparative distance measures on Upham's map were most accurate in the east and less accurate in the west. However, the relative shape distribution of the points on the map and the GIS control map matched fairly well: there were no dramatic surprises. Distances were

sometimes off a bit in the comparison between the Upham map and known control points. Actual distance seems to have been much farther in some cases than the Upham map depicts. However, in the center of the map, Upham placed more distance between features than actually existed.⁴⁴

With this in mind, working with the maps that are available, but realizing their flaws, and then adding in descriptions about landscape features written by people on the ground in their historic time, using pollen studies for a determination of forest composition, and most importantly, soil surveys for a determination of underlying soil composition, it is possible to generally map areas of approximate high risk for Lyme disease infection- a geography of risk for the seventeenth century. This results in four historic risk maps.

MAP 1. CAPE COD, 1620 -1626, is a map of Pilgrim contact with Cape Cod with contact descriptions written by William Bradford and Edward Winslow as an overlay. It includes comments made by the second set of colonists to arrive at Plymouth, who also made a short stop on Cape Cod in 1621 before proceeding to the established village.

THE 1620 PILGRIM COLONISTS were placed on the map of Cape Cod in areas where they interacted as an additional layer of information. The result was a 98 % correlation between HIGH RISK geographic areas and affliction or death in 1620. This number is considered to be statistically significant.

DESCRIPTIONS OF INITIAL INTERACTIONS WITH THE LANDSCAPE OF CAPE COD

1ST EXPLORATORY JOURNEY-November 15-17, 1620

Edward Winslow in Mourt's Relation:

The harbor was "encompassed about to the very sea with oaks, pines, junipers, sassafras, and other sweet wood. The wood was "for the most part open and without under wood, fit either to go or ride in." We came to a deep valley, full of brush, wood-gaile and long grass. There we saw a deer and found springs of fresh water. We went in another valley. We found a fine clear pond. There grew many vines and fowl and deer haunted there. From thence we went on and found much clear ground. Some though it best to go down and travel by the sea sand... We found new stubble, of which they had gotten their corn this year and many walnut trees....As we wandered we came to a tree...In the end we got out of the wood...where we saw three bucks...we came along the creek

William Bradford in Of Plymouth Plantation:

Leaving the sands they turned another way into the woods. They fall into such thickets as were ready to tear their clothes and armour in pieces. By the way they found a pond. They found cleared ground where the native has formerly set corn. They found the river to be more like creeks.


Second Exploratory Journey: December 6-12, 1620.


Edward Winslow:

We landed between two creeks and marched some four or five miles by the greater of them... Our men were tired from marching up the steep hills and deep valleys... We followed certain beaten paths and tracks of the Indians into the woods... we light upon a very broad beaten path, well nigh two feet across..., but in the end we found it to be only a path made to drive deer in...we...marched five or six miles in the wood...

William Bradford:

They marched through the woods.
They hasted out of the woods.
They ranged up and down all day.

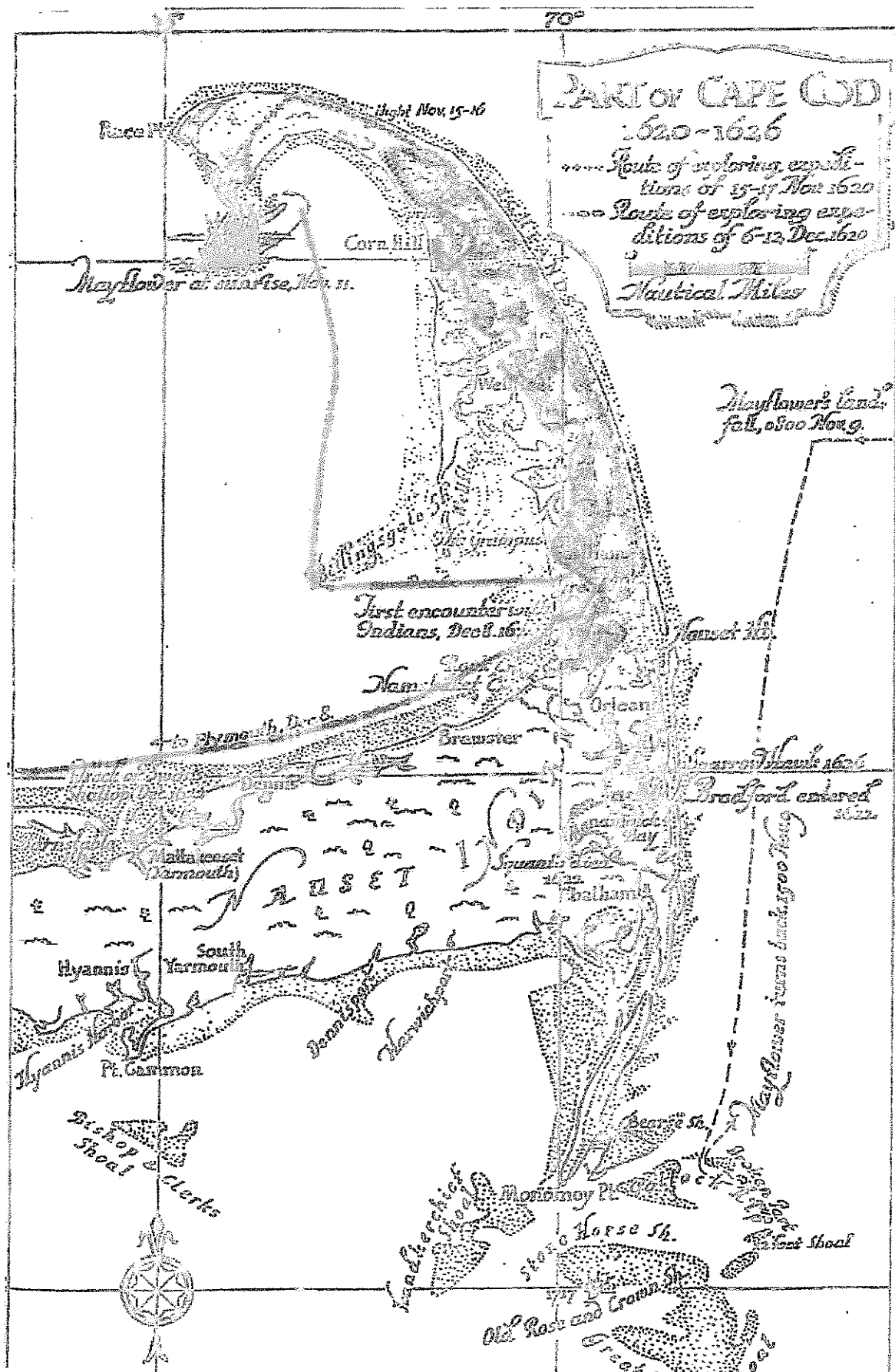
 1st Journey

 2nd journey

 Areas of risk for tick infestation

Map: Page 40 from The Times of Their Lives.

2000 by James and Patricia Scott Deetz.



MAP 2. PORTIONS OF ESSEX COUNTY MASSACHUSETTS, 1692, includes Salem Village, and small parts of Salem, Topsfield and Boxford. This was done because the witchcraft afflictions were spread across this fairly large geographic area, rather than being limited to Salem Village. It uses a soil survey, the Upham, and the GIS generated maps as reference areas that might include tick habitat are marked in red. An overlay was then created that mapped the residences of both humans and animals that were physically “afflicted” with Lyme-like symptoms, who testified as accusers or were testified about, as an additional information layer.

KEY TO MAP 2: Residence of afflicted Between February 1689 and February 1692.

1. Several members of the Wilkins family were afflicted. Sam testified in the Witchcraft trials. Daniel died mysteriously and his death is ruled to be preternatural. Bray Wilkins also suffered from symptoms that sound like either a bladder infection or prostate enlargement.
2. In the Putnam family, the mother Ann Putnam (Sr.) and a daughter, Ann Putnam (Jr.) suffer from seizures and other afflictions. Mary Walcott and Mercy Lewis also lived with this household. Mary Walcott had a possible ECM in the shape of a bite mark on her wrist. Mercy Lewis was an afflicted servant and appears to have moved into Boston sometime late in 1692.
3. In the Parris household, Elizabeth Parris and Abigail Williams suffered from seizures and other afflictions. A son was mentally ill for life.
4. In 1689 Benjamin Holton (Houlton) had suffered from the same type seizures as the afflicted girls. He later died.
5. Animals afflicted
6. Animals afflicted

Outside the Salem Village bounds:

7. Animals afflicted at Giles Corey’s farm
8. Mary Warren- afflicted girl
9. Elizabeth Hubbard- afflicted girl
10. Goody Trask- had the same type seizures as the girls
11. Shattuck with afflicted children lived in the town area



Map 3 shows how the Native American settlement in the area had been situated. Native American's tended to live and farm on rich lowland areas. This occupation zone was surrounded by an upland open forested buffer zone that was subjected to regular burning that cleared both undergrowth and vermin. This zone was in turn surrounded by heavily forested areas. This same area was settled by members of the Putnam family and over the course of time had been split into several farms.

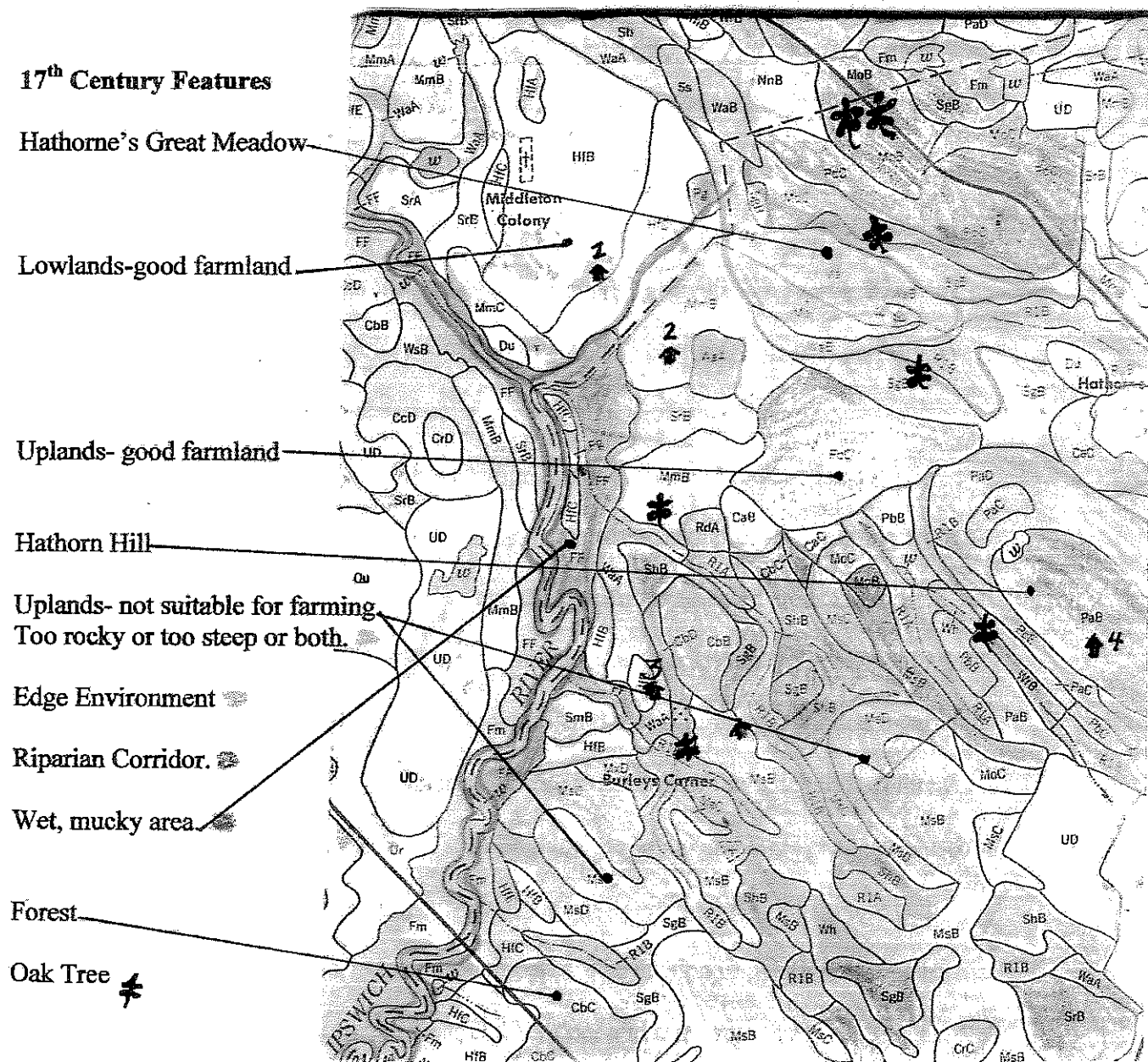
Map 4 places 17th Century features onto the soil survey map to create a depiction of the land in 1692. By then it included the farms of the Thomas Putnam, Edward Putnam, and their cousin John Putnam Jr. The Rea Farm was situated upon Hathorn Hill. These families included the afflicted Ann Putnam, Sr. and Ann Putnam, Jr., several of the afflicted girls and Hannah Putnam. They all suffered from neurological "fits." Ann Putnam, Sr. may have suffered a miscarriage and the eight month old infant of John Jr. and Hannah died of the same symptoms. Edward Putnam described what may be an ECM on the skin of one of the afflicted. An overlay was created based on written descriptions of part of the property, including specific marker trees, from the 1685 will of Thomas Putnam, Sr. This map shows a HIGH level of Lyme disease risk for all members of this family living in this area. It includes numerous edge ecotones, forest fragments, orchards, and a riparian corridor. Numerous oak trees are mentioned.

(Includes some modern feature designations)⁴⁶



Based upon the concepts of the Native American Inhabitation discussed in *The Great Meadow-Farmers and the Land in Colonial Concord*⁴⁷

MAP 4. Soil Map of Putnam Farms Area
(Includes some modern feature designations)⁴⁸



1. Edward Putnam House
 LR. Edward described possible ECM as "the mark being in a kind of a round rink and three strokes across the ring."
2. Thomas Putnam House
 LR. Residents of this farm suffered from "fits" and seizures and lethargy. Ann Putnam, Sr. may have suffered a miscarriage.
3. John Putnam, Jr. House
 Wife Hannah has fits, and an 8 month old child died of the same fits.
4. Daniel Rea House

VIII. CONCLUSIONS:

Cotton Mather was not totally off mark when he wrote about the "wonders of the invisible world." He saw in human affliction the work of the hand of evil. His invisible world, we now know, is inhabited by bacteria and viruses that can cause affliction and death. If the hand of evil is redefined as being the results of infection a new understanding of the past can be formulated. The idea that Lyme disease affected the English settlers in Massachusetts fits logically with modern scientific research, historic writings, and major scholarly works about the seventeenth century. This is especially true for the witchcraft episode of 1692.

Alan MacFarlane argues in *Witchcraft in Tudor and Stuart England* that witchcraft outbreaks occur historically during periods where societies are evolving from a communal to an individualistic ethic.⁴⁵ In northern deciduous forested areas the most important environmental effects of this social movement is the tendency for the particular culture involved to spread out spatially--often onto land that is already inhabited by an established *Ixodes* tick population. This may be true in several of the witchcraft outbreaks that occurred in Europe before the 1690's. In England the social and legal changes caused by the Norman Conquest of 1066 may have had the environmental result of reforested fragments being created as upper class hunting parks. The sudden imposition of upper class ownership onto wild mammals, most notably deer and boar, may have led to rapid population fluctuations that benefited the *Ixodes* life cycle.⁴⁶ In New England, activities related to commerce, usually based on an individualistic desire for profit, moved people and their animals out of the modified environments of Plymouth, Boston and Salem Town into forested edge ecotones and sometimes into the forest itself. The afflictions of

Lyme disease may be an almost unavoidable "side effect" to any movement of this type in northern deciduous forest areas. The neurological nature of some of Lyme disease's symptoms fit seamlessly with a diagnosis of preternatural affliction.⁴⁷

English settlers in seventeenth-century Massachusetts interacted with New England's landscape. They perceived the events of their lives through the lens of their own particular culture. Women and children wore their culturally prescribed long skirts and were inadvertent tick collectors. They used brass or iron and brass pins that coincidentally were the same color as the *Ixodes scapularis* tick. If a circular rash developed on their bodies, it was easily perceived as a sign of the scurvy or a preternatural bite mark. Neurological afflictions that challenged the mind for comprehension were perceived of as the strange and cunning work of the invisible demonic realm. Seizures and lameness was part of the etiology of witchcraft affliction. The defensive response to human agony caused Salem Village's society to crack along an east/west dividing line. Like a flawed ceramic pot that was made with a hairline crack, once it split it would be difficult to put back together in a new way that made sense.⁴⁸

Everything that happened in life needed to be fit neatly into the way that the world was mentally ordered for these English citizens. Bacteria caused their hops to become beer. It curdled their milk. They lived their lives surrounded by bacteria but were unaware. It would be centuries into the future before the germ theory for disease fit neatly into anyone's mental world order. And we still have much to learn about the workings of the invisible bacterial world.

Lyme disease is caused by a bacteria that has both a complex life cycle and an extremely successful evolutionary history—it parasitizes but usually does not kill its hosts.

Accepting an extended history for this disease would challenge some of our most basic perceptions. It may be that we have lived with this bacterium for eons and that it helped shape our social history. Women and young children, who were at such a disadvantage in the seventeenth century because of the style of clothing worn, were likely to suffer from both the acute form and the chronic form of Lyme disease in greater numbers than their male counterparts. This Lyme induced 'weaknesses' may have carried a social price with it. Woman bore the burden of both more witchcraft accusations and affliction in the seventeenth century. The anti-fertility properties of Lyme disease, especially increased spontaneous abortion rates, seem to be embroidered into tales of witchcraft from the past. Other symptoms and facts concerning the dissemination of the Lyme bacteria seem to be subtly encoded into witchcraft folklore. In witchcraft an otherworldly thing (be it devil or tick) attaches to the body in hidden places like "under the left arm" or "on her right side near her ribs" or in "the more secret parts" and suckles blood.⁴⁹ The witch has a mark on her body that is numbed and feels no sensation. The Lyme victim also does not usually feel an attached tick because of the anesthetic quality of tick saliva. A round bull's eye rash sometimes develops. In 1692 the afflicted sometimes developed round rashes on various parts of their bodies that are almost always described as being round like a demon's bite mark.

It is important to remember that in 1692 the society that inhabited Massachusetts Bay was at war with a French and Native American alliance. Salem Village had absorbed a stream of human refugees from Maine. The stress suffered during some of the horrendous events that some of the young afflicted girls had experienced must have been nearly unbearable. Stress has a strong affect on Lyme disease victims. It can reawaken

dormant spirochetes in birds. It may have exacerbated the symptoms in victims like Mercy Lewis and Sarah Churchill who had fled from the warfare that was raging in Maine.⁵⁰

There are frustratingly unattainable 'facts' that may have made for a high rate of Lyme disease infection in 1692. The forest may have experienced an "acorn year" in 1690 or 1691 which nourished a mouse population explosion and carried a subsequent higher than normal rate of Lyme disease. There may also have been an influx of refugee deer from Maine and New Hampshire that migrated down into the relatively more tranquil areas of Andover, Ipswich, and Salem Village. Deer are skitterish creatures that flee from fire and the retort of guns⁵¹. The Putnam family may have decided to pen all their pigs that year to avoid complaints. The previous winter may have been warm. This led to a high survival rate for deer, mice, ticks and bacteria.

Lyme disease may have affected the early demographics of both the Plymouth and Massachusetts Bay colonies. By sickening and depleting the human population it caused significant delays in development at Plymouth, Salem, and Boston. The death of almost all the women in Plymouth Colony during the winter of 1620-21 led to an abrupt halt in reproduction and an almost total dependence on emigration (especially of fertile women) for any future growth at all. Reproductive rates were also decreased in the Massachusetts Bay Colony where many women died. By infecting cows, Lyme disease may have been an important triggering factor behind the original expansion of Puritan settlers into the Connecticut River Valley. In the mid 1630's this move was highly debated before the Massachusetts General Court but was allowed. This created an inland expansion of the English culture area in New England that would never be turned back.

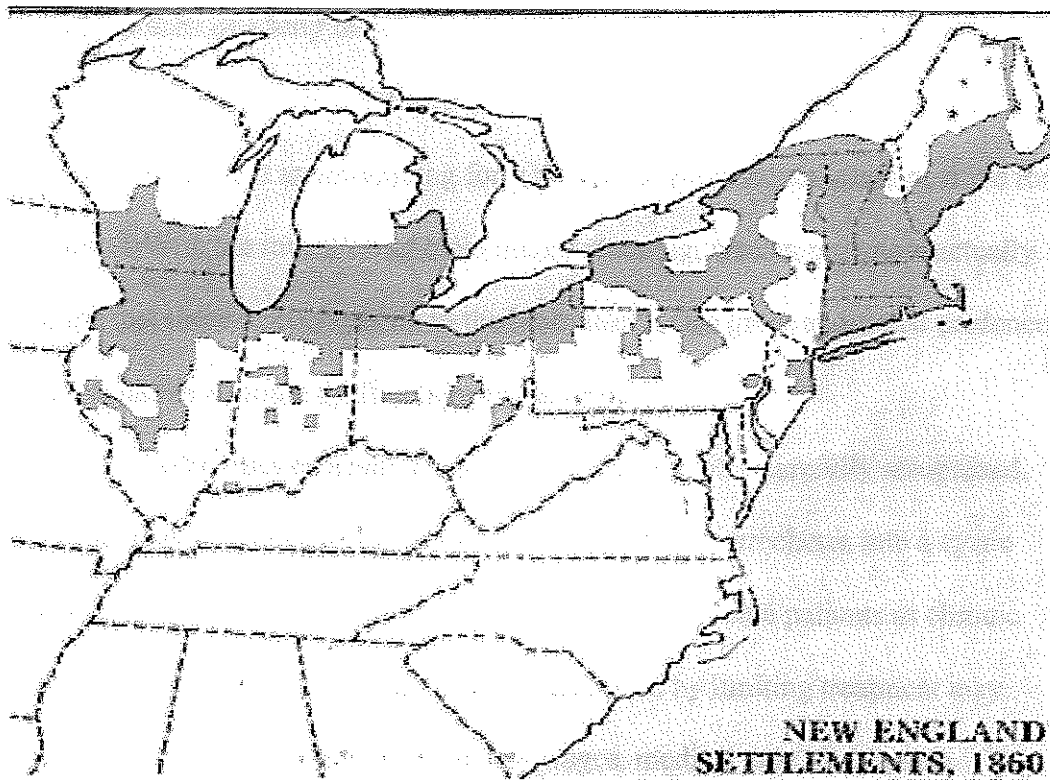
Unlike other more virulent diseases, the history of Lyme disease has been a subtle trail of affliction that did not begin to be defined in America until the nineteen seventies. But even with the brutally high mortality rates that the plague created for centuries it took until 1894 (540 years after it killed off thirty three percent of Europe's population) for the plague bacteria *Yersinia pestis* to be discovered, and three more years for the flea and rat vectors to be determined. This modern insight into the mechanism of spread for the "Black Death" was then circumstantially applied to events that had occurred centuries earlier.⁵² It took until 2004 for DNA amplification of material from plague pit burials in England and France to verify that the dead had indeed been infected with *Yersinia pestis*. Similar research is now being done on the remains of the victims of Justinian's plague from 600 A.D.⁵³ The same should be done for Lyme disease. While not as deadly, it is an infectious factor that has had arguably profound affects on human history.

By afflicting people in the Salem Village area, Lyme disease caused reactive violence to be inflicted in the name of the government of Massachusetts Bay which in turn created a cultural response that has never really ended. The division between church and state that was incorporated into the United States Constitution, as well as the inclusion of the Fifth Amendment in the Bill of Rights, may be in part a reaction to the events of 1692.⁵⁴ And the "Witchcraft Hysteria" is still the subject of modern fascination, research, and scholarship.

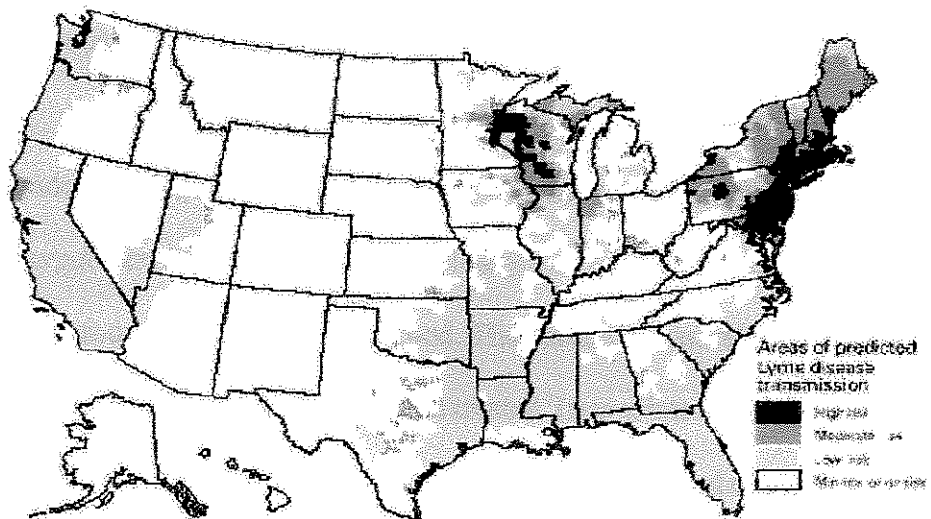
As the United States grew, the English cultural area spread out of New England into the Mid-Atlantic States, the upper areas of the Mid-West, and eventually to parts of the Pacific Coast, especially the gold rush areas around San Francisco that were heavily colonized out of New England. All of these areas had high rates of both prevalence and

risk for Lyme disease in 2002. Comparing a map of the pre-civil war population expansion out of New England with a modern Lyme disease epidemiological prevalence map shows that there are some interesting overlaps of geographical range. Could some of the *Borrelia burgdorferi* bacteria have been spread by humans and their domesticated animals when they moved to farms in the Midwest or retired to Florida? This underscores a need for further research into the topics of human vector competence and sexual and congenital transfer as possible amplifying factors in Lyme disease's epidemiological transmission models. Far from being a modern, easily curable disease of insignificant effect that readily responds to antibiotics, Lyme disease may hold an important, complex, and continuing role as a variable factor of influence in America's cultural history.

Accepting the fact that Lyme disease has a past may give modern researchers new insights into the present. Meeting the *Healthy People 2010*⁵⁵ goal of a forty percent decrease in the prevalence of Lyme disease in the United States will take a Herculean effort. It will require some shifts in approach. There is a great need for all interested parties, including patients, physicians, and medical researchers, to respect each other and be tolerant of differences of opinion. If Lyme disease is seen as an old affliction, it might make chronic Lyme disease and neurological Lyme disease the focus of much more research. Better reporting methods are needed to develop true prevalence rates. The CDC's own formula of one reported symptomatic case for every ten actual occurrences, and then the doubling of it using Dr. Steere's early finding of one asymptomatic case for every symptomatic case, gives an astounding infection rate for the most endemic areas of modern Massachusetts!



Map 9. Population movement from New England by 1860. From *Albion's Seed* by David Hackett Fischer.



Map 10. National Lyme disease risk map with four categories of risk. From the CDC Division of Vector Borne Diseases at www.cdc.gov

The acceptance of a long history for Lyme disease may also shed some much needed light into the area of mental illness. One blood sample survey done in a modern mental institution found that an astounding forty percent of the patients tested positive for Lyme infection.⁵⁶ With the invention of brain image scanning, we can only now actually see the damage that these spirochetes do to the brain itself. This gives additional credence for the infective nature of some dysfunctions of the brain.

The role of antibiotics as a cure for this disease needs to be carefully scrutinized. Inadequate doses may in reality do little more than convert the spirochetes into a cystic state where they can persist, causing a lifetime of affliction for some patients as chronic Lyme disease. Saving a few dollars at the onset of infection by limiting treatment may result in higher societal costs in the future: in lost wages, medical care, and human suffering. Doctors who aggressively treat and actually report cases to the CDC should not be subjected to harassment.

The earlier hopes for a safe vaccine seem to be unrealistic, especially as our understanding of the extremely complex and relapsing life cycle for the *Borrelia burgdorferi* bacteria comes more clearly into focus. The role of the immune system in this process is not fully understood. The chronic but usually controllable nature of the infection needs to be accepted and treated. Immediate research strategies need to shift away from the dogged pursuit of a lucrative vaccine and refocus on environmental remediation to prevent infection in the first place. It appears that this will be more difficult than it seems.

One of the latest NIH funded studies to be released suggests that we may be able to decrease the number of infected nymphal ticks in the forests of New England by 27%

by vaccinating the entire white tailed mouse population. The logistics of this new plan are difficult to fathom. There must be millions of mice that would need to be trapped, vaccinated, and released. If this new mouse vaccine is anything like the old ones, it will require annual booster shots, which would require more trapping and probably some form of tagging system in order to figure out which mice had gotten its shot. It would certainly be less bother to trap and kill these rodents. But then the pharmaceutical companies would not be able to supply and profit from millions of doses of mouse vaccine. The fact that this scheme is being presented as a viable approach to the Lyme disease problem is absurd.⁵⁷

What we truly need to do is to decrease the tick population in endemic areas and do it in a way that does not scourge our bodies and the earth with pesticides or improve the bottom line of the drug companies. Old ideas like regular leaf burning, pannage, and acorn removal could be coupled with experiments that find ways to use acorn derivatives as bio-fuel or building materials. Removing acorns would help limit the population of mice and deer by removing a primary food source. While it is controversial and not likely to happen, the return of a native predator, the wolf, would be an excellent deer population control measure. Looking backward to understand the ways that earlier inhabitants of tick risky areas interacted with their environment, lived their lives, and coped with disease may help to plot a course forward. If we get it right we can create a future where the needs of the afflicted are met, the cycle of infection is broken, and the spread of Lyme disease is contained.

APPENDIX: THE AFFLICTED AND THEIR SYMPTOMS

Name	Date	DIA	Description of Symptoms	ECM	FLS	LA	NL	CNP	RP	BM/D	MEA
Algonquin Speaking tribes Oral History: Eastern Woodlands 1. Kreeh		Precontact	Prerenatural Rheumatism after Deer Hunting		Y				Y		Y
Jamestown settlers Virginia 2. John Smith, General Historie of Virginia, 1624.	1607	Starvation, Scurvy, ?	Irritability, fevers, lethargy (laziness), swellings	Y		Y			Y		Y
Plymouth settlers Massachusetts 3 Bradford, Winslow	1620	Starvation, Scurvy	Fevers, throat problems, joint pain, lethargy	?	Y	Y			Y	Y	Y
Massachusetts Bay Settlers 4. Winthrop, Higginson, Wood	1628-30	Scurvy	Fevers, lameness, lethargy	?	Y	Y			Y	Y	Y
Massachusetts and Connecticut settlers 5. Winthrop	1630's	Bad grass	Spontaneous abortions in Cows						Y	Y	Y
Elizabeth Brown, ¹ Salisbury 6. ECASWVI, 63-64	1662	Prerenatural	Sensation of bird pricking her with motion of ..wings ...in her throat a bunch like a pullet egg								
Ann Cole ² Hartford, Conn. 7. Narratives, 18	1662	Prerenatural	Had fits ,uttered matter unintelligible		Y		?		Y		Urban
John Pressy ³ Ambsbury, Mass. 8. ECASWVI, 64	1668	Prerenatural	Became disoriented in Woods, Hallucination		?				Y		Y
Elizabeth Knap ⁴ Groton, Connecticut 9. Narratives, 21-23	1671	Prerenatural	Tongue drawn up like a semi-circle to the roof of her mouth, not to be removed. Tongue drawn out of mouth to extraordinary length Voice was not her own		Y		XII		Y		Y
James Carr ⁵ Salem Area 10. ECASWVII, 38	1672	Behagged	I was taken after a strange manner as if every living creature did run about every part of my body ... continued for about 3 quarters of a year		Y				Y		Y

DIA-diagnosis, ECM-skin rash, FLS- flu like symptoms, LA- Lyme arthritis, NL-neurological Lyme disease, CNP- cranial nerve pair number, RP-spontaneous abortion or congenital birth defects, BM/D- lives on bird migration route or deer noted, MEA- lives in what is a modern endemic area for Lyme disease.

APPENDIX: THE AFFLICTED AND THEIR SYMPTOMS

Name	Date	DIA	Description of Symptoms	ECM	FLS	LA	NL	CNP	RP	BM/D	MEA
Samual Gray ⁶ Salem 11.ECASWV1, 38	1678	Pretermatural	Hallucination "between sleeping and waking.... athriving child Did pine away and was never well, although it lived some months After, yet in a sad condition and so dyed."				Y		Y		Urban
Morse boy ⁷ Newberry, Mass. 12.Narratives, 23-32	1679	Pretermatural	Barked like a dog, clock't like a hen, in fit his tongue likewise hung out of his mouth, lay miserable lame creeping on one side, lost speech, grown antle		Y		Y	XII	Y		Y
Shattuck child ⁸ Salem Town 13.Narratives, 225,226,380	1680	Bewitched	first child taken with a drooping condition,taken in a terrible fit, his mouth and eyes drawne aside and gaspedat Length we perceived his understanding decayed-Symptoms lasted 17 or18 months.				Y	X, Bells Palsy	Y		Urban
Bernard Peach ⁹ Salisbury 14.ECASWV1,68	1682	Pretermatural	One of the best cows ...was insuch a mad fright that two men had much ado to get her...she did run and fly about..						Y		Y
Hannah Perley ¹⁰ Ipswich, Mass. 15.ECASWV1, 64,146	1682	Pretermatural	was so sore, fell in a dreadful fit, complaining of being pricked with pins and falling down into dreadful fits... so pined a wai to skin and bone and ended her sorrowful life. Also, had a cow "taken strangeli runing about like a mad thing...		Y		Y		Y		Y
Howen boy ¹¹ Boston Area 16.Narratives, 37	1682	Pretermatural	taken in same way as Goodwin children(see 1688 below)				Y	X	Y		Urban
Mary Hortado ? 17. Narratives, 36	1683	Pretermatural	bitten on both arms black and blue and one of her breasts scratched, the impression of the teeth being like a man's teeth.. were plainly seen by many...	ECM							
John Louder ¹² Salem Area 18.ECASWV1, 41.	1684	Pretermatural	felt as if a great weight were pressing on body, struck dumb				Y	VII, X	Y		Y
Shattuck child Salem Town 19. See above	1685	Bewitched	was taken in a strange and unusual manner as if his vitals would have broke out his breast boane drawn up to gather to his upper part of his brest his neck and ey(e)s drawne so aside as if they would never come to right againe..(is) generally in such an uneasie and restless frame almost always runing too and fro acting so strange.	SEE 1680 ABOVE							

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Name	Date	DIA	Description of Symptoms	ECM	FLS	LA	NL	CNP	RP	BM/D	MEA
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John Roger¹³
Billerica
20.ECASWV1,139

1685 Prematural a cow "of a sudden she would give little or none [milk].

Y Y

Jarvis Ring¹⁴
Salisbury, Mass.
21.

1685 Prematural som thing coming upon him when he was in bed and did sorely afflict him by lying upon him and he could neither move nor speake while it was upon him.
the print of the bite (of a woman) is yet to be seen on the little finger of his right hand for it was hard to heal

Y Y Y

?Trask Goodwife¹⁵
Salem bounds
bordering Beverly, Mass.
22.ECASWV1,96-97

1686 Distracted Had fits that were the same as the "afflicted girls", may have committed suicide by slashing throat with scissors.

Y Y Y

The Goodwin Children¹⁶
Boston, Mass
23. Narratives,99-128

1688 Bewitched strange fits, seized sometimes deaf, dumb or blind, tongues down throat, pulled out upon chins to prodigious length, mouths opened to such a wideness that their jaws went out of joint, shoulder blades, elbows, wrist, several of joints stiff, no stirring of heads, heads would be almost twisted around, lost hearing, complain that they were in a hot oven, red streaks on body, could not move head, stiff then limber, they would be twisted into such postures

Y Y Y
II,
VIII,
X
XII

Benjamin Holton¹⁷
Salem Village
24.

1689 Prematural Had fits that were the same as the "afflicted girls" died. was "much pained at his stomach and was often struck blind."

Y Y Y

Samual Abbey¹⁸
Salem Village
25. ECASWV1,8

1689 Prematural Lost several (cattle) in drooping condition..had a cow that could not rise alone

Y Y

Thomas Gadge¹⁹
26.ECASWV1,8

1689 Prematural Cows died in a sudden, terrible & strange unusual manner

Y Y

Margaret Reddington²⁰
Salem?
27.ECASWV1,125

1689 Prematural I was excedingly elle and that night godey esty apeared to me

Y Y Y

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APPENDIX: THE AFFLICTED AND THEIR SYMPTOMS

Name	Date	DIA	Description of Symptoms	ECM	FLS	LA	NL	CNP	RP	BM/D	MEA
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Prisilla Stacey ²¹ ? 28.ECASWV1,37	1690	Pretermatural	felt "pinching and bruising of her till her ears and other parts of her body looked black by reson of her soer pinching of her in the time of her sickness.Died		Y				Y		Y
---	------	---------------	---	--	---	--	--	--	---	--	---

Dorcas Good 29.Homeless?	1692	witch	had a deep red spot about the b ignedness of a fleabite	ECM				Y	Affected for life		Y
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Benjamin Abbott ²² Andover 30. ECASWV1,138	1691	Pretermatural	I was taken with a swelling in my foot& then was taken with a payne in my side which bred to a sore..some of the cattle would come out of the woods with their tongues hanging out of their mouths in a strange and affrightening manner.			Y			Y		Y
---	------	---------------	---	--	--	---	--	--	---	--	---

William Beale ²³ Marblehead 31.NewYork Public Library	1691	Pretermatural	A greate & wracking paine had seized upon my body... Hallucination of shade or shape. My nosed gushed out bleedinge in a most extraordinary manner.						Y		Y
--	------	---------------	---	--	--	--	--	--	---	--	---

Sam Wilkins and Daniel Wilkins ²⁴ Salem Village 32. ECASWV1,101	1691 1691	Pretermatural Pretermatural	I was also pinched by an unseen hand Death					Y		Y	Y
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Phoebe Chandle ²⁵ Andover 33.ECASWV1,140	1692	Pretermatural	There was a voice in the bushes... one half of my right hand was greatly swollen and exceedingly painful & also part of my face...several times since I have been troubled with a great weight upon my breast & upon my legs...soe that I could hardly go..I had a strange burning at my stomach & then was struck deaf		Y				Y		Y
---	------	---------------	--	--	---	--	--	--	---	--	---

Ann Putnam, Sr. ²⁶ Ann Putnam, Jr. Salem Village 34. Narratives, 157	1692	Pretermatural	Sickness caused by some pretermatural cause, lethargy, fits Eldest daughter also was taken in a sad manner...		Y				Y		Y
--	------	---------------	---	--	---	--	--	--	---	--	---

John How ²⁷ ? 35.ECASWV1,151	1692	Pretermatural	Sow died. Hand was so numb and full of pain ..it is not wholly well now		Y				Y		Y
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John Parker ²⁸ Josiah Eaton Andover? 36.Suffolk Court Records Case 2710,48	1692	Pretermatural	'he was pressed almost choked ...his son was strangely handled and sometimes blind in one ey and sometimes in the other ey ".					Y	Y	Y	Y
--	------	---------------	---	--	--	--	--	---	---	---	---

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APPENDIX: THE AFFLICTED AND THEIR SYMPTOMS

Name Date DIA Description of Symptoms ECM FLS LA NL CNP RP BM/D MEA

Stephen Bittford ²⁹ ?	1692	Pretermatural	I was in grate pain of my neck and could not stir my head nor spake a word. I rcv'd a "blow on my chest" I was piched and nipt by sumthing invisible for sum time.	Y	Y	Y	Y	
Abraham Wellman ³⁰ ?	1692	Pretermatural	Cow was taken with fits.. when she see any person coming to milk her She would run and let no one come near her for a week....		Y		Y	
38.Suffolk Court Records Case 2712,48								
Benjamin Gould ³¹ Salem Village 39.ECASWV2,42	1692	Pretermatural	I had two pences upon my side.. i had then such a paine in one of my feet that I could not ware my shue for 2 or 3 days.	Y	Y		Y	
Giles Corey ³² Salem 40.	1692	Pretermatural	I fetched an ox out of the woods7 he laying down in the yard. I went to raise him to yoake him but he could not risse but dragged his hinter parts as if he had been hiptshott. I had a cat last week strangely taken.		Y		Y	
Dog Salem Village 41.Narratives, 372	692	Pretermatural	afflicted, put to death		Y		Y	
Mary Parker ³³ Andover 42.	1692	Witch	Mrs. Parker did lay upon the durt and snow...they thought she was dead... a neighbor said she saw her before in such kinds of fits...she rises up & laughs in or faces...I have seen said Parker in such condition several other times...	Y	Y		Y	
Mercy Short ⁴⁴ Charlestown, Boston Area 43. Narratives,	1692	Pretermatural	fits, pins on body,	Y	Y		Y	
Elizabeth Parris Salem Village 44. Narratives,	1692	Pretermatural	afflicted girl, fits	Y	Y		Y	
Elizabeth Hubbard Y Salem 45. ECASWV1,9	1692	Pretermatural	afflicted girl,.. afflict me by pinching and pricking me...	Y	Y		Y	
Mary Walcut Salem Village 46. Narratives1,53	1692	Pretermatural	had marks of teeth on wrist	Y	Y		Y	
			ECM					

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APPENDIX: THE AFFLICTED AND THEIR SYMPTOMS

Name	Date	DIA	Description of Symptoms	ECM	FLS	LA	NL	CNP	RP	BM/D	MEA
Abigail Williams Salem Village 47 Narratives, 153	1692	Preternatural	mark like print of orange on her skin	ECM			Y		Y		Y
Mercy Lewis Salem 48. Narratives, 153	1692	Preternatural	afflicted girl				Y		Y		Y

Abbreviations:

Narratives-Narratives of the New England Witchcraft Cases, edited by George Lincoln Burr
ECASWV1 or 2-Essex County Massachusetts Archives Salem Witchcraft, Vol 1 and Vol 2.

DIA-diagnosis, ECM-skin rash, FLS- flu like symptoms, LA- Lyme arthritis, NL-neurological Lyme disease, CNP- cranial nerve pair number, RP-spontaneous abortion or congenital birth defects, BM/D- lives on bird migration route or deer noted, MEA- lives in what is a modern endemic area for Lyme disease.

ENDNOTES

INTRODUCTION

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2. Center for Disease Control, *Lyme disease* (Atlanta: U.S. Publishing Office, 2001) pamphlet.
3. See fatalities in K.B. Leigner and C.R. Jones, "Fatal progressive encephalitis." (Abstract presented at the International Conference on Lyme disease and other Tick Borne Diseases at Munich, Germany, June 1999).
4. Center for Disease control, op.cit.
5. This issue is outlined in the Lyme Disease Association's Position Paper *Conflicts of Interest in Lyme disease: Laboratory Testing, Vaccination, and Treatment Guidelines* (April 2001) available at www.lymenet.org.
6. Ibid, Section XII, subsection 5.
7. A.C. Steere, E. Dwyer, and R. Winchester, "Association of Chronic Lyme arthritis with HLA-DR4 and HLA-DR2 alleles," *The New England Journal of Medicine* 323 (July 26, 1990): 219-223.
8. Lyme Disease Association, op. cit., Section IX, "Size of the Lyme Disease Market in U.S. Dollars." See also National Institute of Allergy and Infectious Disease News Release (12/04/2004) "Broad-based Vaccination of Wild Mice Could Help Reduce Lyme Disease Risk in Humans" at www.niaid.nih.gov.
9. CDC, *Lyme Incidence Report* for 2002.
10. Gregory A. Poland, "Prevention of Lyme disease: A review of the Evidence," *Mayo Clinic Proc.* 76 (2001): 717-19.
11. See, for example, www.actionilyme.com, Kathleen Dickson's case may be an extreme one but it highlights both the physical and psychological challenges involved with Lyme disease. Kathleen has gone from testifying as an expert witness before the F.D.A. to being arrested and sent to a psychiatric ward. She proclaims a long list of problems attaining treatment and long list of accusations against the Connecticut Medical establishment. She has filed a racketeering complaint against several drug companies and physicians for conspiring to change the definition of Lyme disease. She has applied for political asylum in Ireland. Dismissed by many as a kook, her case highlights the need to find both better therapies for the disease and public education about its psychological and physical effects.
12. Ann Putnam made her speech in 1706. Betty Parris grew up, married, and lived until 1760. Her brother Noyes, however, is reported to have died young and was described as suffering from mental illness. Abigail Williams is thought to have also died young, perhaps by suicide, and was described as being "followed by diabolical manifestations to her death." see Frances Hill, *Hunting for Witches* (Beverly, Mass: Commonwealth Editions, 2002), 71.
13. See Michael Christopher Carroll, *Lab 257, The Disturbing Story of the Government's Secret Plum Island Laboratory* (New York: Morrow, 2004) for an

overview of research at Plum Island. Other theories for the origins of Lyme disease in North America include the Vanderbilt family bringing it into New York Harbor on animal pelts from Siberia, a Nazi project described in John Loftus' *The Belarus Secret: The Nazi connection in America* (New York: Knopf, 1982) that studied dropping infected ticks from airplanes as an early form of biological warfare, and a later Soviet tick "weaponizing" program. Other evidence suggests that the appearance of the disease in predates all these activities.

14. W. Harvey and P. Salvato, "Lyme disease: Ancient Engine of an unrecognized *Borrelia* pandemic," *Medical Hypothesis* (2003): 742-759.

15. Robert Aronowitz, "Lyme disease-the Social Construction of a New Disease and Its Social Consequences" in *Making Sense of Illness, Science, Society and Disease* (Cambridge U.K.: Cambridge University Press, 1999), 57-83.

16. Dr. Kenneth Leigner, "Lyme disease presentation" at the 12th Annual International Conference on Lyme disease and other Spirochetal and Tick Borne Disorders, New York, April 9, 1999.

17. The symptoms of neurasthenia are listed as psychological plus fatigue, weakness, headache, sweating, polyuria, tinnitus and vertigo, photophobia, fear, easy exhaustion on the slightest effort, inability to concentrate, irritability and complaint of poor memory, poor sleep, numerous constantly varying aches and pains, vaso-motor disturbances. From *Taber's Cyclopedic Medical Dictionary* (Philadelphia: F. A. Davis Co, 1952). This is a classic description of a chronic Lyme patient.

18. Polly Murray, *The Widening Circle* (New York: St. Martin's Press, 1996), 31-45.

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21. This is from personal observation of attached ticks on myself, my son and my mother. They can look like little dark brown splinters or sticker thorns.

22. Cotton Mather, "A Brand Pluck'd Out of the Burning" in George Lincoln Burr, *Narratives of the New England Witchcraft Cases* (Mineola, New York: Dover Publications, 2002), 264.

I. THE AFFLICTION OF LYME DISEASE

1. Brian Fallon and Jenifer A. Miels, "Lyme Disease: Neuropsychiatric Illness," *American Journal Psychiatry* 151 No.11 (November 1994): 1571-1580.

2. Center for Disease Control, *Lyme disease* (Atlanta: U.S. Publishing Office, 2001) pamphlet.

3. Polly Murray, *The Widening Circle* (New York: St. Martins Press, 1996). For a vivid description of her experiences see pages 18-30.

4. Amy Tan quoted in J.J. McCoy, "Lyme diagnoses can miss 'bull's-eye': Author Amy Tan finds herself among Unconventional Cases," *Maine Sunday Telegram*, (August 17, 2003): 1G.

5. Denise Lang, *Coping with Lyme disease* (New York: Holt, 2004), 122.

6. Ibid., 137-138.

7. Ibid., 111 and George Lincoln Burr, *Narratives of the New England Witchcraft Cases*. (Mineola, New York: Dover Publications, 2002) see especially 158-160.

8. Dr. Fallon's work is listed at www.columbia-lyme.org. Preliminary findings of a SPEC brain scan study of Lyme patients done at Columbia University and Columbia Presbyterian Hospital in New York City is due to be completed by the end of 2005.

9. List of symptoms from Karen Vanderhoof-Forschner, *Everything You Need To Know about Lyme Disease and Other Tick Borne Disorders* (New York: John Wiley & Sons, 1997), 47-64.

10. Many of the extant witchcraft trial papers are in Parris' handwriting. This is evident when you compare the writing with sermon books that Parris wrote during the same time period.

11. John Demos, *Entertaining Satan: Witchcraft and the Culture of Early New England* (New York: Oxford Press, 1983).

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13. Mary Beth Norton, *In the Devil's Snare* (New York: Vintage, 2003).

14. Linda Caporael, "Ergotism: The Satan Loosed in Salem?" *Science* 192 (1976).

15. Laurie Winn Carlson, *A Fever in Salem* (Chicago: Ivan R. Dee, 1999).

16. Thurman Sawyer and George Bundren, "Witchcraft, Religious Fanaticism and Schizophrenia—Salem Revisited," *The Early American Review* 3 No. 2 (Fall, 2000): Article 5.

17. Norton, *Snare*, Introduction.

18. James Carr testimony, Essex County Archives, *Salem Witch Trials* VII, 38.

19. Amy Tan quoted in J.J. McCoy, "Lyme diagnoses can miss 'bulls-eye': Author Amy Tan finds herself among Unconventional Cases," *Maine Sunday Telegram* (August 17, 2003): 1G.

20. Polly Murray, *The Widening Circle* (New York: St. Martins Press, 1996).

21. All information from Center for Disease Control, "Vector Ecology" in *Lyme disease* (Atlanta: U.S. Publishing Office, 2001) pamphlet.

22. G. Burke, et al., "Hypersensitivity to Ticks and Lyme disease Risk in Heavily Tick Infested Areas," *Journal of Emerging Infectious Diseases* 11 No.1 (January 2005): 36-41. Being bitten by ticks repeatedly elicits a response that limits the antithetic feature in tick spit making the bite more likely to be felt and the tick to be scratched off by the host human before it can pass on the *Bb* bacteria.

II. ENGLISH SETTLEMENT IN NEW ENGLAND

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Lyme disease spirochete edition that I could get my hands on. It defines scurvy as a disease characterized by mucosa hemorrhage, prostration, and ecchymoses -a form of macula (spot, blemish or stain) appearing in large irregular shaped hemorrhagic areas of the skin. This description is similar to modern descriptions of sore throat, fatigue and various rashes and skin reactions in Lyme disease.

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25. Robert Atkins, *Dr. Atkins New Diet Revolution* (New York: Avon Books, 2002), 47-55.

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CHAPTER III: WAS IT LYME DISEASE?

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16. Daniel Gookin, unpublished papers at The Massachusetts Historical Society, *Historical Collections of the Indians of New England* (1674), 7-12.
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26. Keynote address "The complexity of Vector-borne spirochetes" by Dr. Willy Burgdorfer given on April 9, 1999 at the *Twelfth International Conference on Lyme Disease and Other Spirochetal and Tick borne Diseases*, see also O. Brorson and S. Brorson, "A Rapid Method for Generating Cystic forms of *Borrelia burgdorferi* and their reversal," *APMIS* 106 No.12 (December, 1998):1131-1141.

27. A. Gylfe, et al., "Reactivation of *Borrelia* infection in birds," *Nature* 403 (February 17, 2000): 724-725.

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30. Vanderhoof-Forschner, *Everything*, 47-64.

31. Brian Fallon and J.A. Nields, "Neuropsychiatric symptoms," *The American Journal of Psychiatry* 151, No.11 (November, 1994): 1571-83.

32. Vanderhoof-Forschner, *Everything*, 47-64.

33. Daniel Elliott, Stephen Eppes and Joel Klein, "Teratogen Update: Lyme disease" in *Teratology* 64 (2001): 276-281.

34. Four out of the 13 pregnant women who participated in the Lymerix trials experienced spontaneous abortions: www.lymenet.org.

35. John Winthrop wrote a gossipy detailed account of Mary Dyer's stillborn daughter in his journal and in several letters: "It was a woman child, stillborn, about two months before the just time, having life a few hours before...it was of ordinary bigness; it had a face, but no head, and the ears stood upon the shoulders and were like an ape's; it had no forehead, but over the eyes four horns, hard and sharp, two of them were above one inch long, the other two shorter; the eyes standing out, and the mouth also; the nose hooked upward all over the breast and back, full of sharp pricks and scales, like a thornback; the navel and all the belly with the distinction of the sex, were where the back should be; and the back and hips before, where the belly should have been; behind, between the shoulders, it had two mouths, and in each of them a piece of red flesh sticking out; it had arms and legs as other children; but, instead of toes, it had on each foot three claws, like a young fowl, with sharp talons." Mary Dyer lived outside Boston at Romney Marsh. Cotton Mather's wife also delivered a deformed child on March 28, 1693, that died within a matter of days, followed by the death of the mother within a few weeks. While no elaborate description survives, Samuel Sewall stated that the child had no anus. Cotton Mather had multiple contacts with "afflicted" persons during the 1680's and 90's, including bringing some "afflicted" children to live in his home.

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CHAPTER V: SCURVY DEJA VU?

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3. Willison, *Saints*, 270-272.
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12. Winslow, *Mourt's*,
13. Nathaniel Shurtleff, *Records of the Governor and Massachusetts Bay in New England* (New York: AMS Press, 1968), 232.
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20. See *Livestock of Plymouth colony 1620-1692 research* report by Craig S. Chartier at Plymouth Archaeology Rediscovery website <http://plymoutharch.tripod.com> for an extensive analysis of archeological sites and 257 probate records.
21. David Cressy, *Coming Over*, 280.
22. Ibid., 279.
23. Nathaniel Shurtleff, *Records*, 337.
24. Ibid.
25. Ibid., 232.
26. Ibid., 293, repealed later, 309.
27. Ibid., 148.
28. Ibid., 37.
29. Ibid., Section 4, 37.
30. Ibid., 208
31. Bradford, *Plymouth*

32. Winslow, *Mourt's*, 36.
33. Kenneth Chase, *Firearms: A Global History to 1700* (New York: Cambridge University Press, 2003), 23-26 and Shurtleff, *Records*, inventory list 1630.
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CHAPTER VI. THE PIG IN THE PROMISED LAND

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2. William Wood, *New England's Prospect* (Amherst: University of Massachusetts Press, 1977), 57, and Bradford, *Plymouth*.
3. John Smith, *General Historie of Virginia, New England and the Summer Isles*, (London, 1624) Reprinted in Philip Barbour, ed., *Complete Works of Captain John Smith*, Volume 2 (University of North Carolina Press, Chapel Hill. 1986), 472-473.
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10. California Department of Environmental Science Policy and Management, *Wild Pigs* (Berkeley, California: California Printing Office, 1998).
11. California's feral Pig, op. cit.
12. F.J. Singer, D.K. Otto, A.R. Tipton, C.P. Hable, "Home range movements and habitat use of Wild Boar," *Journal of Wildlife Management* 5 (1981):263-270 and J.J. Meyer and I.L. Bribin, Jr., *Wild Pigs of the United States-their history, morphology, and current status* (Athens, Georgia: University Press, 1991), 313.
13. Gervase Markham, *Good Cheape Husbandries* (London Reprint of 1614 edition).
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22. Shurtleff, *Records*, 90.
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13. Paul Boyer and Stephen Nissenbaum, *Salem-Village Witchcraft*, 236.
14. Phillips, *Salem*, 111-114.
15. Paul Boyer and Stephen Nissenbaum, *Salem-Village Witchcraft*, 230-231.
16. Upham, *Witchcraft*, Vol. 1, 147.
17. Charles Upham, *Salem Witchcraft*, Map of Salem Village in 1692.
18. Ibid., 31.
19. Sarah Loring., *History of Andover*, 1880.
20. Lapping, *Furrow*, 96-97.
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43. Maps from Boyer and Nissenbaum, *Witchcraft*, Appendixes.
44. The University of Virginia Salem Witchcraft GIS project information can be found at <http://lewis.iib.virginia.edu>.
45. Map from James and Patricia Scott Deetz *The Times of Their Lives* (2000), 40.
46. See *Soil Survey of Essex County, Massachusetts, the Southern Part*, 1984, Sheet 7.
47. see Brian Donahue, *The Great Meadow-farmers and the Land in Colonial Concord* (New Haven: Yale University Press, 2004) for an in depth discussion of Native American land usage practices.
48. *Soil Survey*, sheet 7.

VIII. CONCLUSIONS

1. Alan MacFarlane, *Witchcraft in Tudor and Stuart England*. New York: Harper and Row, 1970, 158-164, 192-206.
2. The link of affliction with the Norman conquest of England is noted in Christopher Lee, *1603* (New York: St. Martin's Press, 2003), 120-121. The Normans imposed a series of new property laws and wild mammals became the property of the king.

3. Ipswich, which is directly north of Salem Village had similar problems earlier. By 1658 the residents complained that "for some time suffered losses in their estates, and some affliction in their bodies also...which doth not arise from any natural cause" in Boyer and Nissenbaum, eds., *Salem-Village Witchcraft: A Documentary Record of Local Conflict in Colonial New England* (Boston: Northeastern University Press, 1993), 429.
4. The most in depth study of the social aspects of the 1692 event is Paul Boyer and Stephen Nissenbaum's *Salem Possessed: the Social Origins of Witchcraft* (Cambridge, Massachusetts: Harvard University Press, 1974).
5. Dgar Peel and Pat Southern, *The Trials of the Lancashire Witches: A Study of Seventeenth Century Witchcraft* (New York: Taplinger, 1969), 115.
6. Carol Karlsen, *The Devil in the Shape of a Woman* (New York: Vintage, 1987), 227-228. See also Mary Beth Norton's *In the Devil's Snare* for a lengthy discussion of the warfare connection.
7. B. Harvey, ed., *Living and Dying in England 1100-1540* (Oxford, U.K.: Clarendon Press, 1993), 236-38.
8. Thomas H. Maugh II, "Scientists Use DNA in Search for Answers to Sixth Century Plague," *Los Angeles Times* (May 6, 2002) available at UCLA School of Public Health website www.ph.ucla.edu.
9. In the convoluted legal format of the Salem Witch Trials a confession of guilt often brought a reprieve from capital punishment and a not guilty plea was often followed by a death sentence. When Giles Corey refused to plead at all he was crushed to death. This part of American history may have directly influenced the inclusion of the Fifth Amendment in the Bill of Rights. It offers protection against self incrimination. See also Richard P. Conti, "The Psychology of False Confessions," *the Journal of Credibility Assessment and Witness Psychology* 2 No.1 (1999):14-36.
10. United States Department of Health and Human Services, *Healthy People 2010* (Washington, D.C.: Government Printing Offices, 2000) and Gregory A. Poland, "Prevention of Lyme disease," *Mayo Clinic Procedures* 76 (2001): 713-724.
11. Denise Lang, *Coping with Lyme disease* New York: Henry Holt, 2004) 15.
12. The entomologist Durland Fish is one of the authors of this study. See the National Institute of Allergy and Infectious Disease News Release (12/04/2004) "Broad-based Vaccination of Wild Mice Could Help Reduce Lyme Disease Risk in Humans" at www.niaid.nih.gov.

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