

5-16-2015

Southern Illinois Ticks: An Ecological and Medical Overview

Madeleine A. Pfaff
m.pfaff@siu.edu

Follow this and additional works at: http://opensiuc.lib.siu.edu/uhp_theses

Recommended Citation

Pfaff, Madeleine A., "Southern Illinois Ticks: An Ecological and Medical Overview" (2015). *Honors Theses*. Paper 391.

This Dissertation/Thesis is brought to you for free and open access by the University Honors Program at OpenSIUC. It has been accepted for inclusion in Honors Theses by an authorized administrator of OpenSIUC. For more information, please contact opensiuc@lib.siu.edu.

SOUTHERN ILLINOIS TICKS: AN ECOLOGICAL AND MEDICAL OVERVIEW

MADELEINE A. PFAFF

A thesis submitted to the University Honors Program
in partial fulfillment of the requirements for the
Honors Degree

Southern Illinois University
MAY 12, 2015

TABLE OF CONTENTS

PART I. LITERATURE REVIEW

PART II. PERSONAL STORIES

PART III. EFFECTS OF PRESCRIBED BURNS ON SOUTHERN ILLINOIS TICKS

INTRODUCTION

As one of the most important disease vectors in the United States, ticks draw considerable attention and research to all aspects of their biology: physiology, epidemiology, and ecology. Ticks are of interest to researchers because of their capability to vector a large diversity of pathogens that cause influential and emerging disease globally. There are numerous textbooks written about many facets of tick biology, but here we will cover a brief overview of the literature that exists on ticks, focusing on characteristics that are important to understanding the biology and ecology of ticks in southern Illinois. Because southern Illinois is a region where residents are prone to certain tick-borne illnesses (see page 22), I have included personal accounts from several southern Illinois residents who have dealt/are dealing with tick related illness. The final part of this thesis is a discussion of my study on how tick abundance responds to prescribed burning in southern Illinois.

ACKNOWLEDGMENTS

I would like to thank Southern Illinois University and the University Honors Program for giving me another reason to challenge myself and write this thesis. I am grateful to the Saluki Scholar Research Opportunity program for providing funding for the primary research of this project, and Trail of Tears State Forest and Touch of Nature Environmental Center for allowing me to use their facilities to conduct field work. Valerie Pfaff and Jory Sturdevant assisted me in the field, put the “random” in random transects, and were great sports about hiking through tick-infested forests. Thank you to Steve Gariepy of Touch of Nature who was very gracious, enthusiastic and helpful when I needed to conduct field work. I would also like to thank Elliott Ziemann for assisting me with tick identification and tips for successful tick field work, and Sophia Bonjour, for assisting with statistical analysis. Scott Ferguson, Lori Merrill-Fink and Dr. Marjorie Brooks graciously shared their stories, and I would like to thank Elizabeth Parks, PA-C and Dr. Jeffrey Lehman for taking the time to discuss tick-borne disease and share their insights and knowledge. Finally, I am very grateful to Dr. Eric Schaubert, my advisor, who has challenged me to perform at a higher level than I thought I was capable of and has been patient and generous with his time and advice for as long as I have known him.

DEDICATION

To my parents, who freaked out when I had Rocky Mountain Spotted Fever, but still offer their unconditional support for me going back out to chase ticks. It’s their fault I’m passionate about the outdoors in the first place. Love you, Mom and Dad!

PART I. LITERATURE REVIEW

IXODIDA: CLASSIFICATION AND LIFE HISTORY

More than 12,000 species of arthropods within the order Parasitiformes have been described. Many of these species tend to be quite cryptic and subsist on a diet ranging from fungi, dust, and pollen to dung, carrion and rotten wood. Due in part to these idiosyncratic niches, it is estimated that there are 100,000-200,000 total species in Parasitiformes. Within this order of mites, ticks are classified in the suborder Ixodida, which includes three families. The family Nuttalliellidae contains only one species, while the other two, Ixodidae and Argasidae are classified as hard-bodied and soft-bodied ticks, respectively. Ixodid ticks are distinguished by a hard dorsal shield (scutum) and include about 670 species (Anderson 2002). There are 167 described species of Argasid ticks (1). All ticks have two distinct anatomical segments: a capitulum and an idiosoma. The capitulum is the “business end” as far as feeding goes and is the site of the mouthparts and palps. The idiosoma, posterior to the capitulum, serves as the attachment point for legs and genital pore (Anderson 2002). Larval ticks are distinguishable from nymphs and adults partly because they have six legs instead of the eight of their older counterparts.

Ticks are obligate exoparasites that ingest fluid (blood, lymph or digested tissue) from vertebrates almost worldwide. Mammals, birds and reptiles are all hosts to ticks. A blood meal is essential for the tick’s progression through its four-stage life cycle; at each life stage post-hatching (egg, larva, nymph, adult) the molt into the next life stage or is

regulated by a blood meal. Female ticks must ingest a blood meal to lay her eggs (Anderson 2002).

However, the time spent attached to a host feeding is very short relative to its full lifespan—in *Ixodes scapularis*, only about 2.5% of its life is spent feeding. When not actively parasitizing a host, a tick does not take in any food or environmental water but avoids desiccation by secreting hygroscopic saliva onto the surface of its hypostome. This saliva is salty relative to the air and absorbs atmospheric water to keep the tick hydrated (Francischetti et al. 2009). Similar to a leaf closing its stomata, a tick is able to regulate water loss by closing the openings to its tracheal system. This discontinuous ventilation cycling allows for brief openings of the spiracles that allow for small amounts of CO₂ loss followed by a “burst phase,” open spiracles that cause both CO₂ and water loss (Sonenshine & Roe 2014). To add to the tick’s durability in unfriendly environmental conditions, a tick can also survive for long periods of time underwater by means of plastron respiration, in which small hairs protect an air cavity and facilitate oxygen diffusion from the water into the tick’s respiratory system (Sonenshine & Roe 2014). A specimen of *Dermacentor variabilis* was reported to survive for 15 days underwater (Fielden et al. 2011).

For this paper, we will focus mainly upon Ixodidae because in Illinois, hard-bodied ticks have the greatest effects on humans. Argasid ticks generally live in nests and are less likely to come into contact with humans. That being said, Argasid ticks will feed on humans who come into contact with them in caves, cabins, or large burrows, usually in western states (Anderson 2002).

A hard-bodied tick can have a generation time of 5 to 6 years in northern climates, consuming three blood meals during that time. Further south, generation times average 1-2 years. An Ixodid tick spends only 3-5 weeks attached to a host, feeding, no matter how long it lives (Anderson 2002). The blood it consumes during an attachment period is a tick's only source of nutrients, so its metabolic rate is only 12% of that of other arthropods with similar body mass (Lighto & Fielden 1995). The prevailing hypothesis to explain a tick's ability to have such remarkable energy conservation is its mechanisms to conserve water.

FEEDING HABITS OF IXODID TICKS

For a tick, the key to its next life stage is a blood meal from a vertebrate host. Depending upon the tick species, this host may be a very specific taxa or part of a buffet of choices for the more indiscriminate ticks. Some ticks specialize on a different vertebrate host at each stage of its life cycle. Most ticks of medical importance in Illinois use a passive method of questing, or waiting for hosts. A questing tick waits in vegetation at a height correlating to the vertebrate it targets (lower heights for small mammals and reptiles while adult ticks are likely to quest from vegetation tips for large mammals (Sonenshine 1993)). A questing tick can sense potential hosts through vibration, shadows, body heat, or carbon dioxide, then grasps its host with tarsal claws upon direct contact (Anderson 2002).

The next step is the one with which humans are most concerned: when a tick makes contact with the bloodstream is when pathogen transfer can occur. Many ticks are very selective when choosing an attachment site. When the tick is ready to feed, it

cuts through the dermis and epidermis with its chelicerae and anchors itself to the skin using its teeth on the hypostome as well as a biological cement (Kemp et al. 1982). A pharyngeal pump is used to suck in fluids from the host, where the fluids and tick saliva mix in the buccal duct (Kemp et al. 1982). A tick's saliva is of enormous medical importance to humans, which will be discussed later.

For an ixodid tick to molt into its next developmental stage, it requires at least 2.5 days of feeding for each meal. Balashov (1972) found that larvae feed 3-5 days, nymphs feed 3-8 days, and adults feed 6-12 days. An adult female hard-bodied tick remains on its host until it reaches the engorgement phase, with body weight increasing to 125-150 times pre-feeding mass (Obenchain & Galun 1982). Adult male ticks do not feed, but may be found on host animals where they breed with females. After mating, the female leaves the host and lays 2,000-18,000 eggs in the duff layer on the ground, then dies (Anderson 2002).

TICKS AND PATHOGEN TRANSMISSION: HISTORY AND INTRODUCTION

Ticks, with their durable physiological characteristics, are a global presence. The earliest known record of ticks is an Egyptian papyrus scroll from 1550 BC that referred to "tick fever" (Obenchain & Galun 1982). Subsequently, the Greek poet Homer referred to ticks on Ulysses' dog, and Cato, Aristotle and Pliny the Elder all made reference to the vexatious ticks (Obenchain & Galun 1982; Hoogstraal 1970). Today, ticks are recognized as the number one vector for pathogens in domestic and wild animals in terms of disease diversity, and are second to mosquitoes for vector disease transmission to humans (De la Fuente et al. 2008).

While the ancient scholars certainly made reference to the troublesome nature of ticks, it was not until 1893 that a tick-borne disease transmission event was quantified (Smith & Kilbourne 1893). Theobald Smith was a doctor who worked in the Bureau of Animal Industry under Daniel Salmon, for whom the bacteria genus *Salmonella* is named. In fact, Smith performed the experiment that discovered the organism responsible for a strain of hog cholera, which was later named after Salmon since Salmon's name was listed as first author on the report (Dolman 1984). Besides that discovery, Smith is noted for a series of remarkable accomplishments in the field of infectious disease, including the discovery of anaphylaxis in 1903, *Brucellosis* infections, and observations of human and bovine tuberculosis. He also suggested a link between malaria and mosquitoes (Dolman 1984).

In 1893, Smith and veterinarian Frederick Kilbourne published the results of a series of experiments that demonstrated how Texas cattle fever is caused by a pathogen (*Babesia bigemina*) transmitted by an infected cattle tick (*Boophilus*) (Obenchain & Galun 1982, Smith & Kilbourne 1893). In the twentieth century, numerous papers were published reporting other tick-transmitted pathogens including filaria, protozoa, bacteria, rickettsiae and viruses (Obenchain & Galun 1982). Because of the medical and agricultural importance to humans, much of the work being done on ticks today involves molecular methods of studying the relationships between pathogens, ticks, and hosts (De la Fuente et al. 2008).

Globally, there is a great diversity of diseases in humans that can be attributed to pathogens carried by ticks. These diseases vary in incidence and fatality rate and have

strong ties to local ecology. There is some concern with quantifying global data on tick-borne disease because many cases go unreported or misreported, and our expanding knowledge of pathogen identification casts doubt on the accuracy of historically recorded data on illness cause (Paddock 2009). Nevertheless, the International Institute of Medicine reports Lyme disease in the Holarctic as the most common tick-borne illness, followed by African relapsing fever, rickettsial (spotted fever) diseases, ehrlichiosis, anaplasmosis, Masters' disease, and Crimean-Congo fever (Bacon et al. 2008; Demma et al. 2005; Felsenfeld 1971; Openshaw et al. 2010; Rovey et al. 2008; Vial et al. 2006).

Cortinas et al. (2002) noted three conditions that must be fulfilled for the transmission of a pathogen from tick to human to take place. The first is tick survival: the tick needs food sources in order to advance in development and protection from extreme conditions in the environment. However, the tick is merely a vehicle for the actual disease-causing organism, so pathogen survival is another condition for tick-borne disease transmission. For a pathogen to persist, it needs a sufficient density of tick hosts, a reservoir host population, and opportunities for transmission between ticks and hosts in order to maintain the infection (Cortinas et al. 2002). Reservoir species are important because they harbor pathogens and are sources for disease outbreaks when aided by vectors and carriers. The competence of a reservoir can be quantified by the product of prevalence and infectivity. Prevalence is the probability that the host is infected, while infectivity is the probability that the infected host transmits the pathogen. This is termed "realized reservoir competence" (RRC) and describes the

probability that a host species will transmit a pathogen to an uninfected tick (Ostfeld et al. 2014; Brunner et al. 2008). The RRC value is useful in studies that examine which species play the largest roles in environmental persistence of pathogens. The final criterion for disease transmission to occur is ample encounters between humans and infected ticks.

When it comes to preferred host species, ticks can be specialist or generalist parasites. Generalist ticks that feed upon a different host during each life cycle stage are of the greatest concern for human health. While humans are dead-end hosts and do not proliferate tick-borne disease transmission, the reservoir hosts fed upon by ticks during earlier life stages carry pathogens that are then imparted to humans (Munderloh & Kurtti, 2010). Many of these reservoir hosts do not suffer from disease symptoms as humans do, which is where the problem lies. Since mortality due to infection does not occur in such reservoir species, the pathogen is maintained in the environment in a pool of animals that do not die off and eliminate the disease. The tick acts as a vector to move the pathogen between species.

That being said, the diversity of disease-causing organisms that ticks can transmit is vast. Multiple taxa of bacteria, viruses and protozoa are all capable of tick-aided transfer. Ticks can be infected with more than one pathogen, and co-infections do occur in humans (Swanson et al. 2006).

TICK FEEDING STRATEGY, SALIVA AND DISEASE TRANSMISSION

Certainly one of the most critical moments in a tick's life is attachment to its vertebrate host and subsequent feeding event. Ticks have evolved mechanisms to

facilitate feeding using salivary compounds. Francischetti et al. (2009) published a table that catalogues over 3,500 salivary proteins from various tick species. The variety and abundance of salivary compounds may indicate a kind of evolutionary “arms race” between the parasite and its host (Francischetti et al. 2009).

Tick salivary glands have two main roles: feeding and iron/water metabolism. As for water metabolism, ticks avoid desiccation by exchanging hygroscopic atmospheric water using saliva. When it comes to feeding, a tick must overcome the host’s immune system defense to feed. When bitten by a tick, the vertebrate immune system can induce hemostasis, inflammation, and immunity. Inflammation produces an itching response that helps the host remove the aggressor from its attachment site. Other components in tick saliva include anti-clotting, vasodilatory, anti-inflammatory, and immunomodulatory compounds to aid in overcoming the host’s immune response to the injured integument. Detailed descriptions of the feedback loops that occur during tick feeding and the host’s response can be found in Francischetti et al. (2009). Host resistance is a major mortality cause in ticks since blood is a tick’s only nutritious meal. Such resistance causes reduced blood ingestion, prolonged feeding, egg production, egg viability and molting inhibition (Anderson 2002). The best tick hosts do not develop an immune response to ticks and serve as important food sources for the parasite.

Because saliva is so important for ticks to be able to feed, pathogens have exploited this niche and cause disease transmission through tick saliva (saliva-activated transmission, SAT). Pathogens use the anti-immune system compounds secreted by ticks to travel into the host’s blood stream and eventually cause infection (Nuttall &

Labdua 2004). One example is the agent for Lyme disease, *Borellia burgdorferi*, which binds to the salivary protein Salp15. By binding to Salp15, the pathogen was protected from host antibodies and facilitated colonization in the host (Nandhini et al. 2005).

In short, with the great diversity of tick salivary compounds, many niches for pathogen exploitation exist. Research on the interaction of pathogens and their corresponding salivary proteins is an ongoing effort to better understand the mechanisms for transmission events to hosts.

NORTH AMERICAN/ILLINOIS TICK-BORNE DISEASES IN HUMANS

In North America, there are approximately 80 species of tick; however, only 12 are of major concern to human and animal health. The Center for Disease Control has identified 14 tick-borne diseases that affect humans as well as the genus of tick that transmits it.

Disease	Associated Tick Genus
Human Anaplasmosis	<i>Ixodes</i>
Babesiosis	<i>Ixodes</i>
<i>Borrelia miyamotoi</i>	<i>Ixodes</i>
Colorado tick fever	<i>Dermacentor</i>
Ehrlichiosis	<i>Amblyomma</i>
Heartland virus	<i>Amblyomma</i>
Lyme disease	<i>Ixodes</i>
Powassan disease	<i>Ixodes</i>
Rocky Mountain Spotted Fever	<i>Dermacentor, Rhipicephalus</i>
Masters' Disease (STARI)	<i>Amblyomma</i>
Tickborne Relapsing Fever	Argasid ticks
Tularemia	<i>Dermacentor, Amblyomma</i>
364D rickettsiosis	<i>Dermacentor</i>
Alpha-gal delayed anaphylaxis	<i>Amblyomma</i>
Tick paralysis	<i>Dermacentor</i>

According to the Illinois Department of Public Health, there are at least 15 species of ticks in Illinois (IDPH 2015). However, this number is likely changing due to ecological change and changes in habitat use by neotropical migrating birds (Hamer et al. 2012). Here we will describe four tick-borne diseases endemic to Illinois: Human Anaplasmosis, Ehrlichiosis, Rocky Mountain Spotted Fever and Lyme Disease (Hermann et al. 2014). All four are bacterial diseases with relatively nonspecific symptoms—60-75% of human cases of tick-borne disease are incorrectly diagnosed on the first visit to the doctor (Chapman et al. 2006). However, it should be noted that other tick-borne diseases do occur in Illinois as tick/associated pathogen distribution changes. Babesiosis and cytauxzoonosis are also included due to their emergence in southern Illinois. A brief description of each disease follows.

Human granulocytic anaplasmosis (HA) is an acute febrile illness caused by the obligate intracellular rickettsial bacterium *Anaplasma phagocytophilum*. In the United States, from 2000-2007 reported incidence of HA increased from 0.8 to 3.0 cases/million persons/year (Dahlgren et al. 2011). The first infections were reported in Wisconsin and Minnesota in 1994 and symptoms include fever, chills, headache, nausea and muscle pain (Dumler et al. 1994). *A. phagocytophilum* is found in small mammal reservoir hosts and vectored by *Ixodes scapularis* and *Ixodes pacificus* ticks.

Similar to HA, an ehrlichiosis infection exhibits a clinical presentation of generalized fever, nausea and muscle aches but is more likely to be accompanied by a rash than a HA infection. Both HA and ehrlichiosis laboratory results are characterized by leukopenia, thrombocytopenia, and elevated creatinine/serum aminotransferase

levels (Walker & Dumler 2005; Bakken et al. 1996; Eng et al. 1990). The rickettsial bacterium *Ehrlichia chaffeensis* is the causative pathogen of ehrlichiosis infections in humans, named after Fort Chaffee, Arkansas, where it was first documented in 1991 (Maeda et al. 1987; Dawson et al. 1991; Anderson et al. 1991). *E. chaffeensis* is transmitted to humans by *Amblyomma americanum*, the Lone Star tick, and the white-tailed deer (*Odocoileus virginianus*) is believed to be the major reservoir (Paddock & Yabsley 2007; Childs & Paddock 2003; Chapman 2006). While both diseases are treatable with antibiotics (tetracycline), untreated infections may involve complications like nervous system damage, renal failure, adult respiratory stress syndrome, and disseminated intravascular coagulopathy (Walker & Dumler 2005).

While still important health concerns, the prevalence of HA and ehrlichiosis pales in comparison to the number of Lyme disease infections in the United States. Lyme disease, with an estimated 300,000 cases per year, is the most common tick-borne disease in North American humans (“CDC provides an estimate...” 2013). After an outbreak of what was thought to be arthritis in residents of Lyme, Connecticut in 1975, investigators from Yale found a telltale bullseye rash called *erythema migrans* (EM) in many of the patients. That particular rash pattern had been associated with bacterial infections in Europe and had been linked to tick bites in Sweden. The Lyme infections were also temporally clustered from June-September and were more likely to afflict people who spent time in rural and wooded areas. Steere and Malawista suggested the link between the arthritis and ticks in a 1978 paper (Steere et al. 1978; Elbaum-Garfinkle 2011). In 1982, Willy Burgdorfer isolated and identified *Borrelia burgdorferi* as the

spirochete bacterium that causes Lyme disease (Burgdorfer et al. 1982). They did so by collecting and dissecting *Ixodes scapularis* ticks from regions that had high infection rates of Lyme disease and discovered the bacteria in the tick midguts. A tick must be attached for at least 36-48 hours before pathogen transmission occurs (“Lyme disease transmission” 2013). Most disease transmission events occur at the nymph stage of the black-legged tick, after the larvae have obtained a blood meal from a reservoir host such as the white-footed mouse (Elbaum-Garfinkle 2011), although infection may occur at any life stage.

Symptoms of Lyme disease are quite variable and not all cases present with the EM lesion. Common symptoms of early infections include severe fatigue, myalgia, arthralgias, lymphadenopathies, headaches and fever; neurologic, cardiac, and rheumatological symptoms may also occur (Elbaum-Garfinkle 2011). In later stages of the disease, arthritis is a common symptom. There is ongoing investigation into the affliction of chronic Lyme disease. Most Lyme disease infections, when caught early, can be treated with an antibiotic regimen (Elbaum-Garfinkle 2011). There is currently no available Lyme disease vaccine for humans; a vaccine was released in 1999 but was withdrawn from the market in 2002.

Another *I. scapularis* vectored pathogen is *Babesia microti*, a protozoan. Because it lives in the erythrocytes, thrombocytopenia and anemia are often noted in laboratory results. Symptoms of babesiosis, the clinical manifestation of *B. microti*, include muscle and joint pain, fever and dehydration (Nathavitharana & Mitty 2015). Elevated liver and kidney function tests are other laboratory findings in babesiosis patients. In grave cases,

symptoms can include respiratory, kidney and heart failure (Hatcher et al. 2001). Because *B. microti* is a protozoan, the treatment for babesiosis is not the antibiotics prescribed for bacterial tick-borne illnesses. Instead, most patients receive a course of atovaquone and azithromycin, or clindamycin and quinine for severe cases (Nathavitharana & Mitty 2015). In cases that exhibit a parasitemia level above 10%, liver, kidney or respiratory compromise, or hemolysis, an erythrocyte transfusion may be issued (Nathavitharana & Mitty 2015). Babesiosis is a reportable condition in 27 states (Illinois is not one of them), and >1,700 cases were reported nationwide in 2013 (“Parasites-Babesiosis” 2015).

The final endemic Illinois tick disease discussed here is Rocky Mountain Spotted Fever (RMSF). In Illinois, the foremost vector is American dog tick, *Dermacentor variabilis*. The pathogen, *Rickettsia rickettsii*, belongs to the spotted fever group of Rickettsia. Despite its name, the majority of RMSF cases occur in the southeastern United States (Nathavitharana & Mitty 2015). While ticks vector the pathogen to larger animals, humans and dogs are the only species that are known to display clinical signs of infection (Warner 2002). Upon infection in canines and humans, the *R. rickettsii* proliferate in tissues including the vascular endothelium and smooth muscle. In Illinois, the most common tick vector of *R. rickettsii* is *Dermacentor variabilis*, the American dog tick; *D. andersoni* is usually responsible in western states. There are three additional tick species that are suspected of being vectors for the pathogen for dogs and humans, including *A. americanum*, *Rhipicephalus sanguineus*, and *Haemaphysalis leporispalustris* (Warner 2002).

Cases of RMSF have been reported in all contiguous states except Maine and Vermont. Most cases occur from April to September and the CDC has reported 200-1,120 cases each year in the past fifty years (Dalton et al. 1995; Silber 1996; Treadwell et al. 2000). After the infective tick bite, the pathogen incubates 3-12 days and symptoms include fever, headache, fatigue, loss of appetite, muscle pain, nausea and vomiting. In some cases, a rash develops after onset of symptoms; this is where the term “spotted fever” comes from. 6-10 days after symptom onset is usually accompanied by diarrhea, joint pain, and severe headache. Due to the vasculitis that manifests itself in the form of a rash, multisystem septic shock can occur, as well as gastrointestinal hemorrhage and thrombotic stroke (Thorner et al. 1998). Untreated cases are fatal 15-30% of the time. Doxycycline is the antibiotic usually prescribed for RMSF patients (Warner 2002).

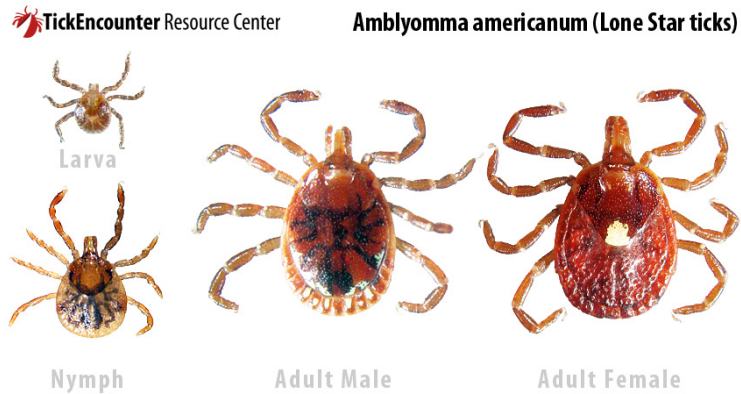
In addition to the pathogenic illnesses described here, another cause for concern when it comes to tick bites is the acquisition of delayed anaphylaxis to alpha-gal. Certain people who are bitten by *A. americanum* can become sensitized to the carbohydrate alpha-gal, which is found in mammalian meat. Due to circulating IgE antibodies to the carbohydrate, people who consume red meat post-tick bite can have an anaphylactic reaction three to six hours after ingestion. Such patients should also be aware that anaphylactic reactions to the anti-cancer drug cetuximab may be linked to the same allergy (Wolver et al. 2013; Saleh et al. 2012). Scientists were unaware of the tick-caused meat allergy until 2006, and not all people with IgE antibodies to alpha-gal have a reaction (Commins et al. 2011).

Naturally, humans are not the only victims of tick-borne illness. Many diseases afflict pets and livestock and are therefore of economic concern. Cytauxzoonosis is a disease in domestic cats that is most likely vectored by *Amblyomma* ticks (69). First described in 1976, the pathogen that causes cytauxzoonosis is the protozoan *Cytauxzoon felis*. Symptom onset is 10-14 days after transmission and causes anorexia, fever, dehydration, jaundice, and eventual death as macrophages obstruct vasculature. It is currently believed that bobcats are a reservoir for the disease and domestic cats, the dead-end host (69, 70). However, cats that survive a *C. felis* infection can serve as a reservoir for the disease, and it is not known how many infected bobcats perish upon infection. The best way to prevent a cytauxzoonosis infection is to keep pet cats indoors and administer flumethrin, a pyrethroid insecticide used to combat ticks and other exoparasites.

A news search on tick-borne illness turns up a bevy of reports of new and unfamiliar afflictions. Outbreaks of Heartland virus, Bourbon virus, Powassan virus, relapsing fever and *Borrelia miyamotoi* pop up throughout the US ranging from Kansas to Montana to New York. Public health entities and other organizations monitor emerging disease with surveillance programs. Current known diseases have been described as “the tip of the iceberg” since ticks are important vectors for a wide variety of pathogens, many of which are likely unknown to science.

ILLINOIS TICKS OF MEDICAL/VETERINARY IMPORTANCE

1. *Amblyomma americanum* (Common name: Lone Star tick)



(All photos sourced from: University of Rhode Island Tick Encounter Resource Center)

The Lone Star tick gets its name from the white spot on the back of the mostly brown adult female. All three life stages of *A. americanum* are aggressive and feed on humans. Adults are 1/8 inch long. The most common life stage found on humans is the nymph stage (“seed tick”), which is the size of a pinhead. Nymphs and adults are active during the warmer months of the year, usually April-August (Illinois Department of Public Health 2015). The Lone Star tick is of medical importance as the vector for the *Phlebovirus* that causes Heartland Virus infections, ehrlichiosis, STARI (Southern tick-associated rash illness), tularemia and an allergy to alpha-gal, a carbohydrate in red meat (“Tick diseases...” 2015; Savage et al. 2013).

2. *Ixodes scapularis* (Common name: Black-legged tick or deer tick)

 Tick Encounter Resource Center *Ixodes scapularis* (Blacklegged ticks or Deer ticks)



Adult *Ixodes scapularis* (formerly *Ixodes dammini*) are sexually dimorphic, dark in color with no light markings. Adult females have reddish or orange coloring behind the black anterior part of the body and are larger than males. Both sexes of adult *I. scapularis* are smaller than *Dermacentor variabilis* individuals. Adult females lay eggs earlier in the spring, and these hatch in June or July. Larval activity peaks in August with most larvae feeding on small mammals. After 3-5 days of feeding, larvae drop off into the leaf litter to overwinter and then molt into the nymph stage the following May. Nymphs feed for several days, drop off and molt into the adult stage by October. After a blood meal, adult females lay 1000-3000 eggs (Patnaude & Mather 2014). Black-legged ticks are noted as vectors of pathogens that cause Lyme disease, human babesiosis, and human granulocytic ehrlichiosis (Des Vignes & Fish 1997).

3. *Dermacentor variabilis* (Common name: American dog tick or wood tick)

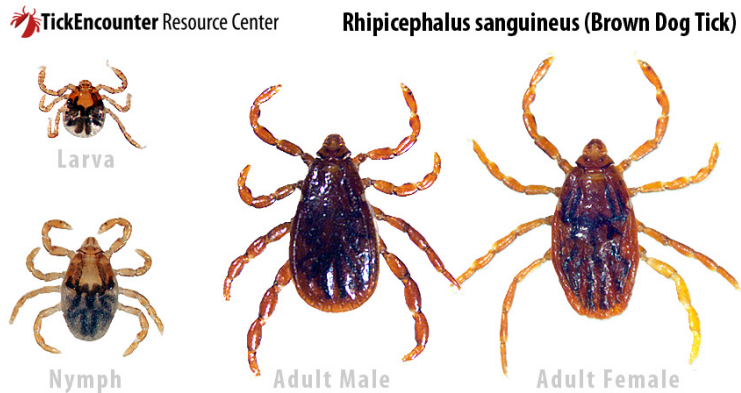
 TickEncounter Resource Center

***Dermacentor variabilis* (American Dog ticks)**



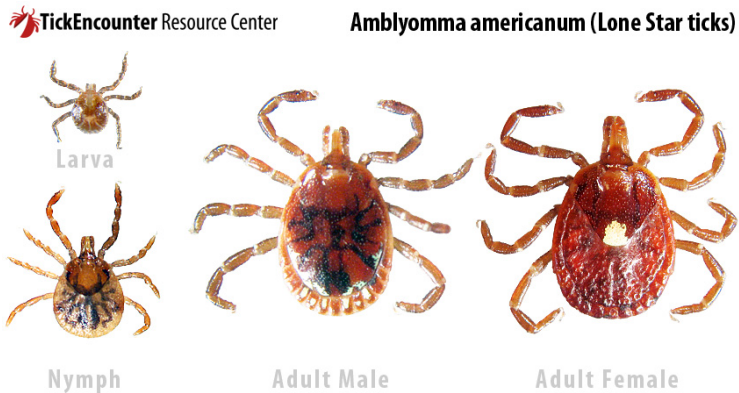
Dermacentor ticks are most likely to feed on humans and other large mammals during the adult stage, with nymphs and larvae focusing on smaller animals such as mice and birds. Reddish-brown in color, female ticks have a silver spot behind the head and males have silver lines on the body. Unfed adult ticks are 3/16-inch in length, but after feeding a female *Dermacentor* tick can be ½-inch long (Illinois Department of Public Health 2015). Peak adult activity occurs May-July and decreases in August. Peak larval activity is in September and early spring. In Missouri, the principal host of adult *Dermacentor* ticks is the raccoon (*Procyon lotor*) (Kollars et al. 2000). American dog ticks have been found to transmit the causative agents of Rocky Mountain spotted fever and tularemia (rabbit fever) to humans. This tick may also be responsible for causing “tick paralysis,” due to a neurotoxin in its saliva (Merck Veterinary Manual 2011).

4. *Rhipicephalus sanguineus* (Common name: Brown dog tick)



R. sanguineus is not a species of major medical concern for humans in Illinois, although it can transmit RMSF in the southwestern United States and Mexico (Chapman 2006). It is, however, a common parasite of dogs in Illinois and can transmit canine ehrlichiosis and canine babesiosis. Globally, it is of medical concern to humans as a vector of *Rickettsia conorii*, the causative agent for tick typhus (Rovero et al. 2008). The brown dog tick is usually 1/8-inch long and engorged females can reach 1/2-inch in length. This tick is a tropical species and is unlikely to survive winter in Illinois outdoors, although it is able to reproduce indoors (Illinois Department of Public Health 2015). The best place to check for this tick on pet dogs is between the toes and around the ears.

5. *Amblyomma maculatum* (Common name: Gulf Coast tick)



A. maculatum is not common in Illinois, but it is possible to find specimens occasionally. The Gulf Coast tick is quite aggressive and is the vector of *Rickettsia parkeri*, which causes a spotted fever (Paddock et al. 2015). It is also considered to be the principal vector for the causative protozoan pathogen of canine hepatozoonosis in the US as well as canine parasitism. A three-host tick, all three life stages of *A. maculatum* have been found on coyotes (*Canis latrans*), which is likely related to the persistence of canine disease-causing agents in the environment (Kocan et al. 1999; Teel et al. 2010). The Gulf Coast tick is characterized by its reddish-brown color and large mouthparts and is mostly found in the southern United States.

DATA ON TICK-BORNE DISEASE IN ILLINOIS

The epidemiology of many tick-borne diseases has been notoriously difficult to study for several reasons. The first is that the symptoms of many tick-borne illnesses are relatively nondescript or similar to influenza and are either unreported or misdiagnosed. Additionally, tick-borne disease awareness may be higher in some areas and then are

more commonly reported. For this reason, a person who has spent time in wooded areas and come down with flulike symptoms should be advised to tell his or her healthcare providers that there is a potential that he or she may have been exposed to ticks. It can also be difficult to analyze historical records of tick-borne disease because our rapidly expanding knowledge of the diversity and scope of such illnesses can negate old data.

Despite these challenges, Hermann et al. (2014) compiled a study of tick-borne disease distribution in Illinois. They found records of 1,289 human tick-borne disease cases from 2000-2009 in the state and focused on the 4 endemic tick-borne diseases: anaplasmosis, ehrlichiosis, Lyme disease and Rocky Mountain spotted fever (RMSF). 92 of 102 Illinois counties reported at least one case of tick-borne disease during the study period. From 2000-2009, Lyme disease was more of a concern in the northern part of the state than in southern Illinois. The *B. burgdorferi* vector, *Ixodes scapularis* (black-legged tick) was first detected in Illinois in 1987 in Jo Daviess county, which is in northwestern Illinois (Bouseman et al. 1990). The geographic distribution of *I. scapularis* expanded throughout the 1990s in the northern part of Illinois, usually associated with forested areas (Bouseman et al. 1990; Guerra et al. 2002). By 2009, *I. scapularis* was confirmed in 26 Illinois counties and is likely in an additional 8 (Herrmann 2014). The highest Lyme rates reported in the study were in Jo Daviess and neighboring Carroll county with 294.4 and 94.4 per 100,000 people. The most Lyme cases reported in the decade were in Cook County (which also has the most people) and DuPage with 214 and 97, respectively. It is four times as likely for a Lyme disease case to occur in the northern

2/3 of Illinois as the southern 1/3. Most Lyme cases occurred in patients younger than 20 and older than 40, with the median age for all tick-borne illnesses 43 (Herrmann 2014).

While Lyme is of higher concern for residents of northern Illinois, people in the southern 1/3 of the state have other foes: Rocky Mountain spotted fever and human ehrlichiosis. A given case of RMSF is 35 times as likely to occur in southern Illinois than northern Illinois, and 22.5 times for ehrlichiosis. The most RMSF cases reported from 2000-2009 were in Jackson county with 32, followed by Williamson with 26. Ehrlichiosis diagnoses followed a similar pattern with 17 cases in Jackson county and 14 cases in Williamson county (Herrmann et al. 2014). Due to the high relative populations of those counties, Pope county experienced the highest rate of both diseases, with 442.58 cases/100,000 residents for RMSF and 93.17/100,000 for ehrlichiosis. Union county and Hardin county were second for disease rates of RMSF and ehrlichiosis, respectively (Herrmann et al. 2014). As for dogs, 88% of those diagnosed with tick-borne disease had not been treated with flea and tick preventive medication.

The differences in disease prevalence between northern and southern Illinois is linked to the species distribution of the host tick. As *I. scapularis* expands its range throughout the state, the risk of Lyme disease will likely increase for southern Illinois residents. Because ticks are associated with rural and wooded areas that have high host densities, the forested southern Illinois is a suitable habitat for them and could be related to the higher disease prevalence of RMSF and ehrlichiosis in those areas. It will take continued disease monitoring in all counties of the state to improve our knowledge

of scope and distribution of ticks and the illnesses they carry. This information is important because it can lead to better awareness in healthcare providers who treat patients for the general symptoms that may actually be a tick-borne disease.

ECOLOGY AND TICK-BORNE DISEASE TRANSMISSION: AN INTRODUCTION

When investigating the origins of emerging diseases, it can be useful to take into account ecology and potential ecological disturbances that play a role in changing disease dynamics and emergence. The factors that dictate how diseases emerge and spread are quite intricate and can be geological, climatic, anthropogenic, and ecological in nature. Each of these factors that influence a tick's life history determines the likelihood that a pathogen transmission event can occur; pathogen survival and chance of contacting a host are determined by such details.

When it comes to ecology, there are several important parameters that affect tick-borne disease. These include vector competency; population dynamics of the tick species; seasonal and diel activity of the tick; tick geographic range; host specificity and host ecology; and habitat requirements of hosts and ticks (Sonenshine & Mather 1994).

ECOLOGY AND TICK-BORNE DISEASE TRANSMISSION: VECTOR COMPETENCY

Vector competency when applied to ticks is defined as the capability for a tick to acquire a pathogen and transmit it to a vertebrate host. Certain tick species are better vectors for certain pathogens. Vector competency is related to contact with reservoir hosts, which are species that accommodate a pathogen or pathogen community from which the vector can acquire pathogens (Sonenshine & Mather 1994).

ECOLOGY AND TICK-BORNE DISEASE TRANSMISSION: HOSTS

In addition to reservoir hosts, ticks may also feed upon animals classified as dilution hosts. Dilution hosts have a lower susceptibility to tick-borne pathogens and do not facilitate transmission as readily as the pathogen-friendly reservoir species. The “dilution effect” reduces overall disease prevalence in a community. Species diversity (but not evenness) is correlated with a lower disease risk since competing species have a negative effect on dominant reservoir hosts (Schmidt & Ostfeld 2001). However, since community effects are contingent on how individual species interact with one another, the dilution effect is not a generalized principle of disease ecology but rather one that can be applied to particular conditions (Salkeld et al. 2013).

One such example is the use of domestic ruminants as dilution hosts. Cattle have been used as a tool to protect people from malaria (“zooprophylaxis”) because infected mosquitoes bite the cattle instead of humans (Service 1991). However, the World Health Organization no longer recommends the use of cattle as a malaria prevention tool because in certain cases cattle can increase mosquito abundance around people by attracting mosquitoes to an a populated area or providing breeding grounds via urine and feces (Dobson et al. 2006). This situation illustrates one of the caveats of the dilution effect: sometimes it works, sometimes it doesn’t; it depends on the surrounding circumstances.

Ticks can be host generalist or specialist to various degrees. Many ticks are specialized to only a few host taxa. Host specificity is regulated by physiology: the tick’s ability to respond to specific compounds associated with a host and the components of a tick’s saliva that allow it to feed on its host by avoiding host immunoresponse to the

bite (Sonenshine & Mather 1994). Target hosts also determine aspects of tick behavior, such as the height of vegetation ticks climb to quest for hosts. Although a tick may specialize on a particular non-human (or even non-mammalian) host, disease transmission to humans is still possible from these species. For example, the pigeon tick *Argas reflexus* caused an outbreak of disease in Germany although it is not a common human exoparasite (Dautel et al. 1991).

Some hosts can also serve as a vehicle for tick range expansion. In addition to mammals, *I. scapularis* parasitize birds. In Chicago, *I. scapularis* ticks carrying *B. burgdorferi* have been observed on migratory and residential birds. It is likely that neotropical migrant species of birds carry neotropical tick species and associated pathogens that do not become established warranting to unsuitable North American habitat (Hamer et al. 2012).

Due to the irreplaceable role that hosts play in tick development as well as pathogen transmission, much of understanding environmental pathogen dynamics is contingent on understanding host ecology and tick interactions.

ECOLOGY AND TICK-BORNE DISEASE TRANSMISSION: HABITAT

Ticks are quite particular about habitat and any given species has habitat requirements that are not ubiquitous. Vegetation, hosts that occupy the habitat, and microclimate all play a role in determining tick habitat preference.

For a tick-borne disease to occur, human exposure must be in an environment where the pathogen, reservoir host, and vector-competent ticks overlap (Barbour & Fish 1993). Biotic, abiotic and human use characteristics determine the means for this to

happen. Changes in North American human land use in the last two centuries have drastically contributed to ecosystem change and manipulation of how ticks use the environment and contact humans. One such landscape change is forest fragmentation. Fragmentation is the division of a continuous patch of area into smaller, isolated areas with a modified matrix among them. The core habitat is the area unaffected by fragmentation. Fragmentation has considerable effects on organism populations and communities living there (Bennett & Saunders 2010).

Such effects can be positive or negative depending on specific habitat effects for species. A beneficial edge effect increases diversity at edges due to greater vegetation complexity, microclimatic factors and multiple habitat availability (Gray et al. 1999; Nelson et al. 2000; Ewers & Didham 2006). Negative edge effects include isolation of patches and corridors that inhibit distribution/dispersal and increased predation/parasitism (Gray et al. 1999; Yahner 1988). To a tick, the most important aspect of fragmentation is how its host population is effected. For example, the white-footed mouse (*Peromyscus leucopus*) is a reservoir for a variety of pathogens as well as a suitable host for nymph-stage ticks. Mouse abundance is found to be higher in small, fragmented patches than in large habitat areas. The combination of its suitability as a reservoir and host leads to higher infected nymph density (Lewellen & Vessey 1998; Nupp & Swihart 1998; Allan et al. 2003).

Furthermore, historic disturbance has been correlated with tick-borne disease prevalence. In the late 18th and early 19th centuries, there was widespread deforestation of old-growth forest by settlers in the northeastern United States. By the late 19th

century, many farms in that area were abandoned, leading to forest succession providing quality habitat for white-tailed deer (*Odocoileus virginianus*) and their parasites, *Ixodes scapularis*. Such historic land use changes are believed to have stimulated Lyme disease emergence in the 20th century (Spielman et al. 1985). A disease outbreak among Soviet military personnel who spent time in recently abandoned brushy areas is thought to be due to an increase in tick loads on European hares that flourished when Crimean farms were abandoned and overgrown during World War II (Hoogstraal 1979).

In another study on Crimean-Congo hemorrhagic fever, high habitat fragmentation with high connectivity was positively correlated with case incidence (Estrada-Peña 2010). Some host populations do well at close proximity to humans, and thrive in suburban environments. Patterns of urbanization can influence geographic distribution of tick-borne disease such as Rocky Mountain Spotted Fever. RMSF was originally transmitted by *Dermacentor andersoni*, endemic to northwestern and north-central USA (Wolbach 1919). After a geographical shift, the RMSF pathogen is transmitted by *D. variabilis* in the more highly populated southeastern United States. Changes in tick habitat suitability can also occur with changes in human land use. Reforestation is generally considered to be good for ticks, and the hostile microclimate of monoculture agriculture can hinder tick success (Pfaffle 2013).

In conclusion, a compelling argument can be made for the understanding of ecological influence on ticks and tick-borne disease as an important tool for disease reduction. In this paper, I have barely scratched the surface of all the research done and

theories formulated about tick-environmental interaction because it is outside the scope of this paper. Biologist Richard Ostfeld argues for a more comprehensive view of ecology and ticks, doing away with reductionist, over-simplified views of these systems (Ostfeld 2011). Not only can an understanding of tick ecology help us to understand pathogen-tick dynamics, but will also allow us to develop management strategies for reducing disease risk.

TICK PREVENTION AND CONTROL

The most effective way to prevent tick-borne illness is to be aware of situations where tick exposure is a possibility. The following recommendations are provided by the Center for Disease Control. Exercise caution near wooded/brushy areas by wearing long pants and shirts, preferably treated with permethrin or >20% DEET repellent. Avoiding direct contact with brush and long grass can reduce tick exposure; ticks do not jump or drop down from trees. After spending time outdoors, check for ticks on the body, paying close attention to ears, behind knees, inside belly button, and in the hair. Upon discovering a tick, remove it as close to the skin as possible using tweezers. Clean the area with soap and water or rubbing alcohol. Dispose of the tick of by submersing it in rubbing alcohol, flushing it down the toilet, or placing it in a sealed container. Avoid crushing the tick with fingers. Examine clothes, packs and pets for ticks. Tumbling clothes in the dryer can kill undetected ticks. Extra caution should be taken from April to September, when ticks are most active in North America.

PART II. PERSONAL STORIES

TICK TALES: SOUTHERN ILLINOIS RESIDENTS SHARE THEIR TICK EXPERIENCES

For being so tiny, ticks have a resounding impact on people around the world as a nefarious creature. The diversity in tick species and their associated pathogens have vastly different impacts on people worldwide from paralysis in Australia to the neurologic effects of chronic Lyme disease in North America. While much of Illinois is monoculture grain fields, southern Illinois is unique in that it is mostly forested. The Shawnee National Forest has many recreational opportunities and people enjoy living in homes close to the forest. While such activities add to the quality of life of southern Illinois residents, they can also bring them into close proximity with disease-carrying ticks. For some people, a bite can mean a pesky itch... or an event that has life-changing effects.

I first became interested in tick-borne illness after a project trapping small mammals where I was spending a lot of time in the woods at Touch of Nature Environmental Center (TONEC). Despite religious DEET application, I ended up with dozens of tick bites over the course of several weeks. In September, I began to feel fatigued all day no matter how much sleep I got at night and had some generalized aches and pains. When the fatigue did not subside after almost two weeks, I went to the doctor. They tested me for several conditions including mononucleosis and anemia, but I suggested that they might want to test for a tick-borne disease due to the number of bites I had. The test results came back just before my twenty-first birthday: my blood

had tested positive for Rocky Mountain Spotted Fever. After a round of doxycycline, I felt much better; my case had been mild compared to many. My minor bout with Rocky Mountain Spotted Fever did pique my interest in ticks and how they work, and was the inspiration for this project. The following are accounts from several people who have dealt with tick-borne illness. I am very gracious for their time and willingness to share their experiences for this project.

The drive from Carbondale down Giant City Road to the state park is a scenic route through rolling hills bordered on both sides by hardwood and pine forest. It is more common than not to see wild turkey and white-tailed deer along the roadside. Much of this picturesque land is part of Southern Illinois University's Touch of Nature Environmental Center. TONEC was the brainchild of past SIU president Delyte Morris, who encouraged the university to purchase 150 acres of land along Little Grassy Lake in 1949. The land, which has increased to more than 3,000 acres, is used for outdoor education and programming for SIU students and the community. TONEC provided the first camp for disabled people in the world. It also provides facilities for a wide array of research for SIU students and faculty from fisheries to deer studies.

In addition to all the benefits TONEC imparts to people, ticks also seem to flourish on the site. Scott Ferguson has been working at TONEC since 2000, lives on-site and has had more than his share of tick encounters. Eight years ago, Scott had a headache and did not feel well, but took ibuprofen and enjoyed his Fourth of July holiday in Makanda. He had been nursing an open wound on his calf, and after the Independence Day celebration he wasn't feeling any better. He spent the week sick

(including a 104 degree fever), and after his ability to walk and eat were compromised he visited the hospital. The medical staff at the hospital treated him for a possible infection of his calf wound, but it was not until an infectious disease doctor examined his case that he was diagnosed with Rocky Mountain Spotted Fever. *Ehrlichiosis* antibodies were also present in his blood work. The doctor prescribed Scott the antibiotic doxycycline and he felt better after two days.

During his illness, Scott lost thirty pounds and sustained some nerve damage. Living on-site at TONEC, Scott also takes precautions to protect his pets from tick-borne illness. His veterinarian recommends changing the medication used to combat ticks regularly. Frontline (fipronil and (S)-methoprene) “stopped working here.” His pets currently take chewable tablets for tick-borne disease prevention. Scott believes that the high deer density affects the large tick population at TONEC. Fellow TONEC staff member Steve Gariepy remembers finding fawns with enormous tick loads on their ears. Steve, who coordinates programs at TONEC, makes sure that the children he works with are aware of ticks and encourages their parents to inspect them for ticks after spending time outside at TONEC.

Scott is not the only SIU employee to have felt the effects of a tick bite. Back on the main Carbondale campus, away from the lush forests of Touch of Nature, Morris Library sits at the edge of Thompson woods. Lori Merrill-Fink, the director of the University Honors Program, has her office in the library with a window facing the woods. Her home is also close to the forest, which is where she was probably bitten by a Lone Star tick almost three years ago. Around Christmas 2012, Lori enjoyed one of her

favorite treats: a Steak and Shake hamburger. That evening, she was overcome with severe stomach pain and itchiness on her arms, chest, legs and neck. Because she is allergic to bee stings, Lori's husband administered her EpiPen, which "took off the edge" but she went into the Emergency Room and was given intravenous Benadryl.

After her trip to the ER, Lori was referred to an allergist, who diagnosed her as the first confirmed case of a red meat allergy in Carbondale. Lori describes her allergy as more of an "inconvenience," especially since her husband and son enjoy eating meat so much, and she does miss Steak and Shake burgers. Before her allergic reaction, Lori seldom ate red meat in large quantities. Today, she is very cautious to avoid red meat; "It's not worth the discomfort from the reaction," she says. One point of interest, though, is that cured meats do not seem to bother her. Bacon, prosciutto and salami are safe for her to eat without adverse consequences. I did not find any information in the literature about cases in which alpha-gal allergy patients are able to eat cured meat, but perhaps the curing process might contribute to the breakdown of the carbohydrate in red meat that triggers an allergic reaction.

Dr. Marjorie Brooks is an aquatic ecologist and assistant professor in the SIU Zoology department. In 2009, she was living in Wyoming but visited Carbondale to interview for the position she currently holds. While in Illinois, she visited Touch of Nature and was bitten by a tick. Within 10 days of the bite, Dr. Brooks began to experience joint aches, fatigue, cognitive impairment and had trouble with word retrieval. She saw her general practitioner, who diagnosed her with Lyme disease and prescribed her antibiotics. Dr. Brooks was on intravenous antibiotics for six months and

had an intravenous port for self-administration. Today, six years after her diagnosis, she takes daily oral antibiotics as a prophylactic treatment of her Lyme. She said that one doctor advised her to discontinue the antibiotics for a period of time, but she felt very ill and has been taking them daily ever since. As a biologist, Dr. Brooks spends considerable time in the outdoors and has seen other people in her life deal with challenges related to tick-borne illness. When most Americans think of tick-borne illness, Lyme disease is likely the first one that comes to mind. However, due to the relatively low abundance of *Ixodes scapularis* ticks in southern Illinois, it has not been common here in the past. As a side note, "Southern Lyme," also known as Masters' disease/STARI, is vectored by *Amblyomma*, a common southern Illinois tick. STARI causes Lyme-like symptoms. As *I. scapularis* continues its range expansion into the region, though, Lyme disease cases do occur and may increase in prevalence as more people come into contact with *I. scapularis* ticks.

The healthcare providers that serve the southern Illinois community have seen their share of tick-borne illness, as well. Elizabeth Parks, a Physician Assistant in Herrin, believes that overall her patients and people in the region are educated about ticks and their associated risks. Elizabeth has been a PA for six years and says that the number of cases of tick-borne illness varies from year to year. Tests for Lyme disease and other tick-borne illnesses can be delicate because there are many blood test criteria to be met for a positive diagnosis. Currently, the CDC recommends an enzyme immunoassay (EIA) or immunofluorescence assay (IFA) as the first part of a two-step testing process; if either test comes back positive then an IgG Western blot (> 30 days), or IgM and IgG

Western blot (<30 days) are ordered. When she does see a patient with a tick-borne illness, Elizabeth prescribes an antibiotic (doxycycline) or refers the patient to an infectious disease specialist for further treatment. Some patients visit her office in order to get a tick removed or because they sustained a tick bite and are concerned about potential disease.

Dr. Jeffrey Lehman is another southern Illinois healthcare provider who works with patients affected by ticks. As an allergist, Dr. Lehman sees patients with IgE to alpha-gal from Lone Star ticks bites. He first became aware of the allergy in 2007, soon after it was discovered, while working in Springfield, IL. After moving to Carbondale in 2012, he diagnosed his first patient with the allergy in early 2013 and since then has found about 40 clinically significant cases. "I have seen an increase in prevalence only because I am looking for it more often. This has likely been around for a while but we just discovered it in 2007," he noted. Dr. Lehman said that most of his patients who are diagnosed with the allergy are aware that they were bitten by a tick, but some patients did not have a known tick bite. Additionally, there is some evidence to suggest that chigger bites might also play a role in IgE to alpha-gal. In addition to sensitivity to red meat, Dr. Lehman said that some patients are unable to tolerate dairy since it contains some alpha-gal. On the other hand, it is also possible to lose sensitivity to alpha-gal if the person avoids tick bites for some time.

PART III. PRIMARY RESEARCH: EFFECTS OF PRESCRIBED BURNS ON TICKS

There have been many global efforts to control tick populations with varying levels of success. Deer eradication attempts have produced widely variable effects on tick abundance (Ostfeld 2011). Corn-filled troughs surrounded by rollers coated in acaricide forces hungry deer to coat their necks with the insecticide in order to feed, killing 100% of ticks on deer (Pound et al. 2000; Solberg et al. 2003). Tests of these 4-poster feeders have shown their effectiveness, but only ticks that feed on deer are affected. For people or agencies that are reluctant to apply chemical acaricides to the environment, another option for tick control is ecological manipulation such as brush control. One way to do so is by implementing a controlled burn of potential tick habitat. But does prescribed burning actually work as a long-term tick control tactic or should it be used with other possible results in mind?

Fire is a tool used to manage vegetation communities' structure and composition, affecting succession and dominant plant species. It has indirect effects on animals because it is a habitat modifier. Fire can also be used to reduce fuel loads for wildfire risk (Stephens & Ruth 2005). Hoch et al. (1972) measured a decreased abundance of Lone Star ticks immediately after burning in oak-hickory forests due to direct incineration and reduction of the duff layer and herbaceous tick habitat. Some sites are burned on a regime of with burns occurring at intervals of one to several years. In plots burned annually, *Amblyomma* abundance is reduced, but if fire is omitted for a

year than larval abundance can increase to pre-burn levels or higher (Davidson et al. 1994). In the Missouri Ozarks, which have a forest composition similar to that of southern Illinois (oak-hickory), Allan (2009) linked patterns of fire and tick abundance to white-tailed deer use of sites and found that larval *A. americanum* populations recovered to abundances >6 times those of pre-burn numbers.

Objective

The primary research component of my honors thesis is to study how ticks respond to prescribed burning. The objective of this portion of the project is to compare tick density between burn units that differ in time since the last burn.

Methods

My study sites were Touch of Nature Environmental Center (TONEC) in Makanda, IL and Trail of Tears State Forest (TOT) near Jonesboro, IL. Three units at TOT were included in my study, and I sampled six units at TONEC. I sampled on days with similar meteorological conditions (mostly sunny and between 65-75 degrees Fahrenheit) to nullify effects of tick diel activity. I used a 1-m² flannel drag cloth as my collection device and walked 3 random 100-m transects in each burn unit. Upon completion of each transect, I inspected the drag cloth for ticks and saved specimens in vials filled with 70% isopropyl alcohol.



Figure 1. Map of study site locations, southern Illinois



Figure 2. Inspecting drag cloth for ticks

With assistance from SIU graduate student Elliott Ziemann, I identified ticks to life stage and species under a dissecting microscope. I divided the number of ticks collected at burn unit by 300 (three 100-m transects with a 1-m² drag cloth) to obtain a value of ticks per square meter. I divided up time since burn into 4 time classes: 0-2 years post-burn, 2-5 years post-burn, 5-15 years post-burn, and >15 years post-burn. For each time class, I averaged the tick densities from all surveys conducted in corresponding burn units. I then ran a one-way analysis of variance (ANOVA) to determine whether the time since burn had a significant effect on tick density.

0-2 years since burn	2-5 years since burn	5-15 years since burn	>15 years since burn
TOT South Burn	TON 7 Ridges	TON East Creek	TOT Control
TOT North Burn	TON Camp 1	TON West Creek	TON U40 Control
TON Camp 2			

Table 1. Burn units sampled and the time class since last controlled burn.

Results

I collected $n = 79$ ticks from the 9 study sites. Of the collected ticks, 91.1% were of the species *Amblyomma americanum* while the remaining 8.9% were *Dermacentor*

variabilis. Those numbers do not necessarily reflect species abundances relative to one another since *A. americanum* ticks are known to be more aggressive questers than *D. variabilis* ticks (Hair & Howell 1970). All *D. variabilis* specimens collected were adults, with 57.1% male and 42.8% female. Of the larval *A. americanum* ticks collected, all 22 were collected in October. Of the total ticks collected, 37.9% were nymphs and 29.1% were adults. The data shown here includes all tick life stages collected.

Densities of ticks collected in all burn and control units ranged from 0 to 0.053 ticks per m² (Mean = 0.0228; SD = 0.022). The results of the one-way ANOVA showed that the time since burn did not have a significant effect on tick density ($F_{3,7} = 2.726$; $p = 0.124$).

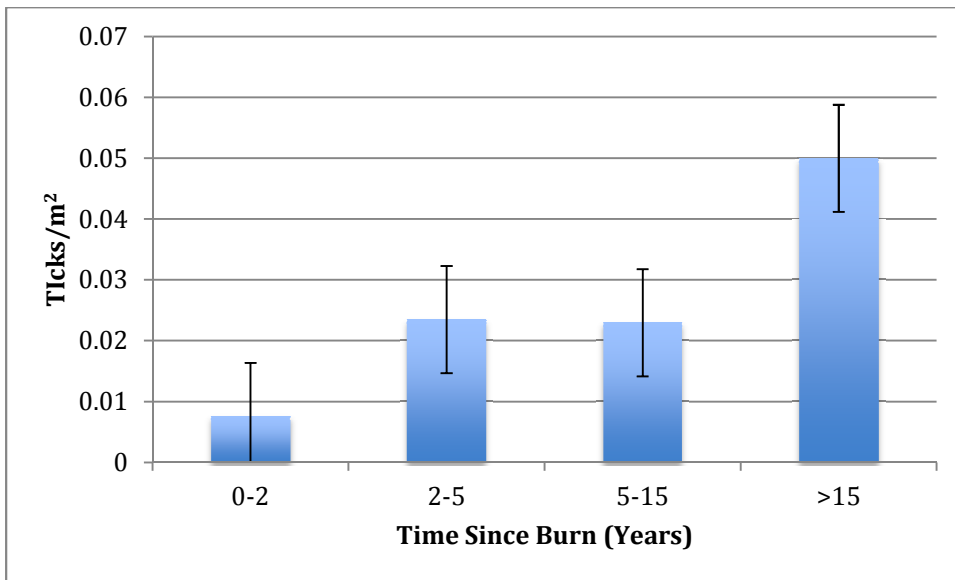


Figure 3. Time since burn (years) vs. tick density

Discussion

In order to best interpret the data, it is important to understand certain aspects of the sites that were sampled. Camp 1 at TONEC burned in 2007 and again in 2010. It is

being managed for grass and there is very little herbaceous vegetation in the understory. Zero ticks were found there. Camp 2 at TONEC burned in 2007 and most recently in 2014, but had more low and medium height vegetation than Camp 1. One tick was found there. The four units at TONEC close to Indian Creek (East Creek, West Creek, U40 Control and 7 Ridges) were characterized by a larger quantity of vegetation than the Camp 1 & 2 units, although the vegetative characteristics were not quantified. Additionally, the deer density at TONEC has been noted to be quite high. While sampling for ticks, I noted 4 deer in the West Creek unit and 3 deer in the 7 Ridges unit (as well as one coyote). I saw zero deer at Trail of Tears while sampling for ticks.

Another variable when considering how burning affects ticks is the characteristics of the burn event itself. A very hot burn that destroys most or all of the duff layer may kill more ticks initially than a less destructive burn event. Rate of vegetative succession may also affect how ticks and their hosts respond to a burn event.

According to these data, tick density increases with time since burn. Further experimentation would require a larger sample size and a broader ecological perspective that quantifies other variables such as host use and vegetative characteristics of the burn units. Since the ANOVA showed that time since burn did not have a significant effect on tick density, it is inconclusive whether prescribed burning has a major effect on ticks. However, according to the raw data there seems to be a trend toward lower tick densities after more recent burns.

LITERATURE CITED

1. Allan, B.F.; Keesing, F.; Ostfeld, R.S. 2003. Effect of forest fragmentation on Lyme disease risk. *Conservation Biology* 17:267–272.
2. Anderson, J.F. 2002. The natural history of ticks. *Medical Clinics of North America* 86:(2)205–218.
3. Anderson B.E.; Dawson, J.E.; Jones, D.C.; Wilson, K.H. 1991. *Ehrlichia chaffeensis*, a new species associated with human ehrlichiosis. *Journal of Clinical Microbiology* 29:2838–2842.
4. Bacon, R.M.; Kugler, K.J.; Mead, P.J. 2008. Surveillance for Lyme disease—United States, 1992–2006. *MMWR* 57(No. SS-10):1-9.
5. Bakken, J.S.; Krueth, J.; Wilson-Nordskog, C.; Tilden, R.L.; Asanovich, K.; Dumler, J.S. 1996. Clinical and laboratory characteristics of human granulocytic ehrlichiosis. *Journal of the American Medical Association* 275:199–205.
6. Balashov, Y.S. 1972. Bloodsucking ticks (Ixodoidea): Vectors of diseases of man and animals. *Entomological Society of America* 8:1–376.
7. Barbour, A.G. & Fish, D. 1993. The biological and social phenomenon of Lyme disease. *Science* 260:1610–1616.
8. Bennett, A.F. & Saunders, D.A. 2010. Habitat fragmentation and landscape change. S. Navjot, S. Ehrlich, P.R. Ehrlich (Eds.), *Conservation Biology for All*, Oxford University Press, Oxford pp. 88–106.
9. Birkenheuer A.J.; Marr, H.R.; Warren, C.; Acton, A.E.; Mucker, E.M.; Humphreys, J.G.; Tucker, M.D. 2008. *Cytauxzoon felis* infections are present in bobcats (*Lynx rufus*) in a region

where cytauxzoonosis is not recognized in domestic cats. *Veterinary Parasitology*. 153(1-2):126-30.

10. Bouseman, J. K.; Kitron, U.; Kirkpatrick, C.E.; Siegel, J.; Todd, K.S. 1990. The status of *Ixodes dammini* (Acari: Ixodidae) in Illinois. *Journal of Medical Entomology* 27:556–560.
11. Brunner, J.L.; LoGuidice, K.; Ostfeld, R.S. 2008. Estimating reservoir competence of *Borrelia burgdorferi* hosts: prevalence and infectivity, sensitivity, and specificity. *Journal of Medical Entomology* 45(1):139-47.
12. Burgdorfer, W.; Barbour, A.G.; Hayes, S.F.; Benach, J.L.; Grunwaldt, E.; Davis, J.P. 1982. Lyme disease—a tick-borne spirochetosis? *Science* 216(4552):1317-9.
13. CDC provides estimate of Americans diagnosed with Lyme each year. (2013, August 19). Retrieved from <http://www.cdc.gov/media/releases/2013/p0819-lyme-disease.html>.
14. Chapman, A.S. 2006. Tickborne Rickettsial Diseases Working Group. CDC Diagnosis and management of tick borne rickettsial diseases: Rocky Mountain spotted fever, ehrlichiosis, and anaplasmosis – United States. *MMWR Recommendations and Reports* 55(RR04):1–27.
15. Childs, J.E. & Paddock, C.D. 2003. The Ascendancy of *Amblyomma americanum* as a vector of pathogens affecting humans in the United States. *Annual Review of Entomology* 48:307–337.
16. Commins, S.P.; James, H.R.; Kelly, L.A. 2011. The relevance of tick bites to the production of IgE antibodies to the mammalian oligosaccharide galactose- α -1,3-galactose. *Journal of Allergy Clinical Immunology* 127(5):1286-93.

17. Cortinas, M.R.; Guerra, M.A.; Jones, C.J.; Kitron, U. 2002. Detection, characterization, and prediction of tick-borne disease foci. *International Journal of Medical Microbiology* 291(33):11-20.
18. Dahlgren, F.S.; Mandel, E.J.; Krebs, J.W.; Massung, R.F.; McQuiston, J.H. 2011. Increasing Incidence of *Ehrlichia chaffeensis* and *Anaplasma phagocytophilum* in the United States, 2000–2007. *The American Journal of Tropical Medicine and Hygiene*, 85(1), 124–131.
19. Dalton, M.J.; Clarke, M.J.; Holman, R.C. et al. 1995. National surveillance for Rocky Mountain spotted fever, 1981–1992: epidemiologic summary and evaluation of risk factors for fatal outcome. *American Journal of Tropical Medicine and Hygiene* 52:405–413.
20. Dautel, H.; Kahl, O.; Knulle, W. 1991. The soft tick *Argas reflexus* (F). (Acari: Argasidae) in urban environments and its medical significance in Berlin (West). *Applied Entomology* 111:380-390.
21. Davidson, W. R.; Siefken, D.A.; Creekmore, L.H. 1994. Seasonal and annual abundance of *Amblyomma americanum* (Acari: Ixodidae) in central Georgia. *Journal of Medical Entomology* 31: 67–71.
22. Dawson, J.E.; Anderson, B.E.; Fishbein, D.L.; Sanchez, J.L.; Goldsmith, C.S.; Wilson, K.H.; Duntley, C.W.. 1991. Isolation and characterization of an *Ehrlichia* species from a patient diagnosed with human ehrlichiosis. *Journal of Clinical Microbiology* 29:2741–2745.
23. Demma, L. J.; Holman, R.C.; McQuiston, J.H.; Krebs, J.W.; Swerdlow, D.L. 2005. Epidemiology of human ehrlichiosis and anaplasmosis in the United States, 2001-2002. *American Journal of Tropical Medicine and Hygiene* 73:400-409.

24. De la Fuente, J.; Estrada-Pena, A.; Venzal, J.; Kocan, K.; Sonoenshine, D. 2008. Overview: ticks as vectors of pathogens that cause disease in humans and animals. *Frontiers in Bioscience* 13:6938-6946.
25. Des Vignes, F.; Fish, D. 1997. Transmission of the agent of human granulocytic ehrlichiosis by host-seeking *Ixodes scapularis* (Acari: Ixodidae) in southern New York State. *Journal of Medical Entomology* 34: 379-382.
26. Dobson, A.; Cattadori, I.; Holt, R.D.; Ostfeld, R.S.; Keesing, F.; Krichbaum, K.; Rohr, J.R.; Perkins, S.E.; Hudson, P.J. 2006. Sacred cows and sympathetic squirrels: the importance of biological diversity to human health. *PLoS Medicine* 3:714-718.
27. Dolman C.E. 1984. "Theobald Smith, 1859-1934: A Fiftieth Anniversary Tribute" (PDF). *ASM News* 50: 577–80.
28. Dumler, J.S.; Bakken, J.S.; Eckman, M.R.; Vanetta, L.L.; Chen, S.M.; Walker, D.H. 1994. Human granulocytic ehrlichiosis: a new, potentially fatal tick-borne infection diagnosed by peripheral blood smear and PCR. *Laboratory Investigations* 70:A126–A126.
29. Elbaum-Garfinkle, S. 2011. Close to Home: A History of Yale and Lyme Disease. *The Yale Journal of Biology and Medicine* 84(2):103–108.
30. Eng, T.R.; Harkess, J.R.; Fishbein, D.B.; Dawson, J.E.; Greene, C.N.; Redus, M.A.; Satalowich, F.T. 1990. Epidemiologic, clinical, and laboratory findings of human ehrlichiosis in the United States, 1988. *Journal of the American Medical Association* 264:2251–2258.
31. Estrada-Peña A.; Vatansever, Z.; Gargili, A.; Ergönul, O. 2010. The trend towards habitat fragmentation is the key factor driving the spread of Crimean-Congo haemorrhagic fever. *Epidemiology and Infection* 138:1194-1203.

32. Ewers, R.M. & Didham, R.K. 2006. Confounding factors in the detection of species responses to habitat fragmentation. *Biological Review of the Cambridge Philosophical Society* 81:117-142.
33. Felsenfeld, O. 1971. *Borrelia*: strains, vectors, human and animal borreliosis. W. H. Green, St. Louis, MO. 180 pp.
34. Fielden, L.J.; L.M. Knolhoff; Villarreal, S.M.; Ryan, P. 2011. Underwater survival in the dog tick *Dermacentor variabilis* (Acari: Ixodidae). *Journal of Insect Physiology* 57:21-6.
35. Francischetti I.; Sa-Nunes, A.; Mans, B.; Santos, I.; Ribiero, J. 2009. The role of saliva in tick feeding. *Frontiers in Bioscience* 14:2051-2088.
36. Gray, J.S.; Kirstein, F.; Robertson, J.N.; Stein, J.; O. Kahl. 1999. *Borrelia burgdorferi* sensu lato in *Ixodes ricinus* ticks and rodents in a recreational park in south-western Ireland. *Experimental and Applied Acarology* 23:717–729.
37. Guerra, M. A.; Walker, E.; Jones, C.; Paskewitz, S.; Cortinas, M.R.; Stancil, A.; Beck, L.; Bobo, M.; Kitron, U. 2002. Predicting the risk of Lyme disease: habitat suitability for *Ixodes scapularis* in the north-central U.S. *Emerging Infectious Diseases* 8:289–297.
38. Hair, J.A.; Howell, D.E. 1970. Lone star ticks: their biology and control in Ozark recreation areas. Oklahoma State University, Agricultural Experiment Station, Bulletin No. B-679, 47 pp.
39. Hamer, S.; Goldberg, T.; Kitron, U.; Brawn, J.; Anderson, T.; Loss, S.; Walter, E.; Hamer G. 2012. Wild birds and urban ecology of ticks and tick-borne pathogens, Chicago, Illinois, USA, 2005-2010. *Emerging Infectious Diseases* 18(10):1589-1595.

40. Hatcher, J.C.; Greenberg, P.D.; Antique, J.; Jimenez-Lucho, V.E. 2001. Severe babesiosis in Long Island: review of 34 cases and their complications. *Clinical Infectious Diseases* 32:1117-25.
41. Herrmann, J.; Dahm, N.; Ruiz, M.; Brown, W. 2014. Temporal and spatial distribution of tick-borne disease cases among humans and canines in Illinois (2000-2009). *Environmental Health Insights* 8(2):15-27.
42. Hoch, A. L.; Semter, P.J.; Barker, R.W.; Hair, J.A. 1972. Preliminary observation on controlled burning for lone star tick (*Acari: Ixodidae*) control in woodlots. *Journal of Medical Entomology* 9: 446–451.
43. Hoogstraal, H. 1979. The epidemiology of tick-borne Crimean- Congo hemorrhagic fever in Asia, Europe, and Africa. *Journal of Medical Entomology* 15:307–417.
44. Hoogstraal, H. 1970. Bibliography of Ticks and Tickborne Diseases From Homer (about 800 B.C.) to 31 December 1969, Vol 1. United States Naval Medical Research Unit Number Three, Cairo, Egypt.
45. Illinois Department of Public Health. Prevention & Control: Common Ticks. (15 April 2015). Retrieved from: <http://www.idph.state.il.us/envhealth/pccommonticks.html>.
46. Kemp, D.H.; Stone, B.F.; Binnington, K.C. 1982. Tick attachment and feeding: Role of the mouthparts, feeding apparatus, salivary gland secretions, and the host response. FD Obenchain, R Galun (Eds.), *Physiology of Ticks*, Pergamon, Oxford pp. 119–168.
47. Kocan, A.; Breshears, M.; Cummings, C.; Panciera, R.J.; Ewing, S.A.; Barker, R.W. 1999. Naturally occurring hepatozoonosis in coyotes from Oklahoma. *Journal of Wildlife Disease* 35: 86-89.

48. Kollars, T.M.; Oliver, J.H.; Master, E.J.; Kollars, P.G.; Durden, L.A. 2000. Host utilization and seasonal occurrence of *Dermacentor* species (Acari:Ixodidae) in Missouri, USA. *Experimental and Applied Acarology*. 24(8):631-43.
49. Lewellen, R.H. & Vessey, S.H. 1998. The effect of density dependence and weather on population size of a polyvoltine species. *Ecological Monographs* 68:571–594.
50. Lighto, J.R.B; Fielden, L.J. 1995. Mass scaling and standard metabolism in ticks: a valid case of low metabolic rate in sit and wait strategists. *Physiological Zoology* 68:43-62.
51. Lyme disease transmission. (2013, January 11). Retrieved from <http://www.cdc.gov/lyme/transmission/>.
52. Maeda, K.; Markowitz, N.; Hawley, R.C.; Ristic, M.; Cox, D.; McDade, J.E. 1987. Human infection with *Ehrlichia canis*, a leukocytic *Rickettsia*. *New England Journal of Medicine* 316:853–856.
53. Merck Veterinary Manual. 2011. Tick Paralysis. Accessed July 17, 2012. Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc. Whitehouse Station, NJ.
54. Munderloh, U.G. & Kurtti, T.J. 2010. Emerging and re-emerging tick-borne diseases: New challenges at the interface of human and animal health. Institute of Medicine Committee on Lyme Disease and Other Tick-Borne Diseases: The State of Science.
55. Nandhini R.; Narasimhan, S.; Pal, U.; Bao, F.; Yang, X.F.; Fish, D.; Anguita, J.; Norgard, M.V.; Kantor, F.S.; Anderson, J.F.; Koski, R.A.; Fikrig, E. 2005. The Lyme disease agent exploits a tick protein to infect the mammalian host. *Nature* 436:573-577.

56. Nathavitharana, R.R.; Mitty, J.A. 2015. Diseases from North America: focus on tick-borne infections. *Clinical Medicine*. 15(1):74-77.
57. Nelson, D.R.; Rooney, S.; Miller, N.J.; Mather, T.N. 2000. Complement-mediated killing of *Borrelia burgdorferi* by nonimmune sera from sika deer. *Journal of Parasitology*, 86:1232–1238.
58. Nupp, T.E. & Swihart, R.K. 1998. Effects of forest fragmentation on population attributes of white-footed mice and eastern chipmunks. *Journal of Mammalogy* 79:1234–1243.
59. Nuttall P.A. & Labdua, M. 2004. Tick-host interactions: saliva-activated transmission. *Parasitology* 129:177-189.
60. Obenchain, F.D.; & Galun, R. 1982. *Physiology of Ticks*, Pergamon Press, Oxford. pp. 213-244.
61. Openshaw, J. J.; Swerdlow, D.L.; Krebs, J.W. et al. 2010. Rocky Mountain spotted fever in the United States: interpreting contemporary increases in incidence. *American Journal of Tropical Medicine and Hygiene* 83:174-182.
62. Ostfeld, R.O. 2011. *Lyme Disease: The Ecology of a Complex System*. Oxford University Press.
63. Ostfeld R.S.; Levi, T.; Jolles, A.; Martin, L.; Hosseini, P.; Keesing, F. 2014. Life history and demographic drivers of reservoir competence for three tick-borne zoonotic pathogens. *PLoS One*. 9(9):e107387.
64. Paddock, C.D. & Yabsley, M.J. 2007. Ecological havoc, the rise of White-tailed deer, and the emergence of *Amblyomma americanum*-associated zoonoses in the United States. *Current Topics in Microbiology* 315:289–324.

65. Paddock, C. D. 2009. The science and fiction of emerging rickettsioses. *Annals of the New York Academy of Sciences* 1166:133-143.
66. Paddock, C.D.; Denison, A.M.; Dryden, M.W.; Noden, B.H.; Lash, R.R.; Abdelghani, S.S.; Evans, A.E.; Kelly, A.R.; Hecht, J.A.; Karpathy, S.E.; Ganta, R.R.; Little, S.E. 2015. High prevalence of "Candidatus *Rickettsia andeanae*" and apparent exclusion of *Rickettsia parkeri* in adult *Amblyomma maculatum* (Acari: Ixodidae) from Kansas and Oklahoma. *Ticks and Tick Borne Disease*.
67. Parasites- Babesiosis, 2013 Data and Statistics. (2015, January 13). Retrieved from <http://www.cdc.gov/parasites/babesiosis/data-statistics/index.html>
68. Patnaude, M.R.; Mather, T.N. 2014. Blacklegged tick or deer tick. University of Florida Publication Number EENY-143.
69. Pfaffle, M.; Littwin, N.; Muders, S.V.; Petney, T.N. 2013. The ecology of tick-borne diseases. *Zoonoses* 43(12-13):1059-1077.
70. Pound, J. M., Miller, J.A.; George, J.E. 2000. Efficacy of amitraz applied to white-tailed deer by the "4-poster" topical treatment device in controlling free-living lone star ticks (Acari: Ixodidae). *Journal of Medical Entomology* 37:878-884.
71. Rizzi, T.E.; Reichard, M.V.; Cohn, L.A.; Birkenheuer, A.J.; Taylor, J.D.; Meinkoth, J.H. 2015. Prevalence of *Cytauxzoon felis* infection in healthy cats from enzootic areas in Arkansas, Missouri, and Oklahoma. *Parasitology Vectors*. 8:13.
72. Rovey, C.; Brouqui, P.; Raoult, D. 2008. Questions on Mediterranean spotted fever a century after its discovery. *Emerging Infectious Diseases* 14:1360-1367.

73. Saleh H.; Embry, S.; Nauli, A.; Atyia, S.; Krishnaswamy, G. 2012. Anaphylactic Reactions to Oligosaccharides in Red Meat: a Syndrome in Evolution. *Clinical and Molecular Allergy* 10(1):5.
74. Salkeld, D.J.; Padgett, K.A.; Jones, J.A. 2013. A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. *Ecological Letters* 16:679-686.
75. Savage, H. M.; Godsey, M.S.; Lambert, A.; Panella, N.A.; Burkhalter, K.L.; Harmon, J.R.; Nicholson, W.L. 2013. First Detection of Heartland Virus (Bunyaviridae: *Phlebovirus*) from Field Collected Arthropods. *The American Journal of Tropical Medicine and Hygiene* 89(3):445–452.
76. Schmidt, K.A. & Ostfeld, R.S. 2001. Biodiversity and the dilution effect in disease ecology. *Ecology* 82:609-619.
77. Service, M.W. 1991. Agricultural development and arthropod-borne diseases—a review. *Revista De Saude Publica* 25:165-178.
78. Silber, J.L. 1996. Rocky Mountain spotted fever. *Clinical Dermatology* 14:245–258.
79. Smith, T.; Kilbourne, F.L. 1893. Investigations into the nature, causation, and prevention of Texas or southern cattle fever. U.S. Department of Agriculture Burden Animal Industry Bulletin 1:1–301.
80. Solberg, V. B.; Miller, J.A.; Hadfield, T.; Burge, R.; Schech, J.M.; Pound, J.M. 2003. Control of *Ixodes scapularis* (Acari: Ixodidae) with topical self-application of permethrin by white-tailed deer inhabiting NASA, Beltsville, Maryland. *Journal of Vector Ecology* 28:117-134.
81. Sonenshine, D.E. 1993. *Biology of Ticks, Vol 2*. Oxford University Press, New York.

82. Sonenshine, D.E. & Mather, T.E. 1994. *Ecological Dynamics of Tick-Borne Zoonoses*. Oxford University Press.
83. Sonenshine, D.E. & Roe, R.M. 2014. *Biology of Ticks, Volume 1* (2nd ed.). Oxford University Press.
84. Spielman A.; Wilson, M.L.; Levine, J.E.; Piesman, J. 1985. Ecology of *Ixodes dammini*-borne human babesiosis and Lyme disease. *Annual Review of Entomology* 30:439-460.
85. Steere, A.C.; Broderick, T.F.; Malawista, S.E. 1978. Erythema chronicum migrans and Lyme arthritis: epidemiologic evidence for a tick vector. *American Journal of Epidemiology* 108(4):312-21.
86. Stephens, S. L.; Ruth, L.W. 2005. Federal forest-fire policy in the United States. *Ecological Applications* 15:532–542.
87. Swanson, S.J.; Neitzel, D.; Reed, K.D.; Belongia, E.A. 2006. Coinfections acquired from ixodes ticks. *Clinical Microbiology Review* 19(4):708-27.
88. Teel, P.D.; Ketchum, H.R.; Mock, D.E.; Wright, R.E.; Strey, O.F. 2010. The Gulf Coast tick: a review of the life history, ecology, distribution and emergence as an arthropod of medical and veterinary importance. *Journal of Medical Entomology* 47(5): 707-722.
89. Thorner, A.R.; Walker D.H.; Petri, W.A. 1998. Rocky Mountain spotted fever. *Clinical Infectious Disease* 27:1353–1359.
90. Tickborne diseases of the US. (2015, January 15). Retrieved from <http://www.cdc.gov/ticks/diseases/>

91. Treadwell T.A.; Holman, R.C.; Clarke, M.J. et al. 2000. Rocky Mountain spotted fever in the United States, 1993–1996. *American Journal of Tropical Medicine and Hygiene* 63:21–26.
92. Vial, L.; Diatta, G.; Tall, A. et al. 2006. Incidence of tick-borne relapsing fever in West Africa: longitudinal study. *Lancet* 368:37-43.
93. Walker, D.H. & Dumler, J.S. 2005. (*Ehrlichia chaffeensis* (human monocytic ehrlichiosis), *Anaplasma phagocytophilum* (human granulocytic anaplasmosis), and other ehrlichieae). In: Douglas and Bennett's Principles and Practice of Infectious Diseases. Sixth Edition. Mandell GL, Bennet JE, Dolin R, editors. Volume 2. New York: Elsevier; 2005. pp. 2310–2318.
94. Warner, R.D. 2002. Rocky mountain spotted fever. *Journal of the American Veterinary Medicine Association* 221:1413–7.
95. Wolbach, S. 1919. Studies on Rocky Mountain spotted fever. *Journal of Medical Research* 41:1–218.
96. Wolver S.; Sun, D.; Commins, S.; Schwartz, L. 2013. A peculiar cause of anaphylaxis: no more steak? *Journal of General Internal Medicine*. 28(2):322-325.
97. Yahner, R. 1988. Changes in wildlife communities near edges. *Conservation Biology* 2:333-339.