Climate Change and Emerging Diseases in New England

New or expanding diseases New England might face if CO_2, temperatures and rainfall levels continue to rise.

Sandra Martin

May, 2010

Climate change is resulting in rising temperatures and wetter conditions in New England, creating opportunities for new and expanding diseases and increasing mortality and morbidity from environmental and infectious diseases. While there is some controversy over the causes of climate change, few scientists dispute that CO_2 levels are increasing, temperatures in the more northern latitudes are rising and precipitation levels are becoming more chaotic. Rising CO_2 levels, rising temperatures and extreme rainfall events are all associated with faster plant growth, heat stress and a substantial increase in mosquito and tick vectors. Rising temperatures are also associated with increases in plant toxicity and disease growth rates. As populations expand and move into more rural areas and people travel more widely, diseases are more likely to spread among vulnerable populations such as those found in New England. Increasing direct diseases of interest in New England due to climate change include plant diseases, heat related stress, allergies, asthma and chronic lung conditions. Indirect diseases of interest include food borne diseases such as E. coli and salmonella and vector borne diseases such as Lyme, babesiosis, malaria, dengue fever, yellow fever and West Nile Virus. Public health officials in New England should consider these climate change trends when establishing local priorities and increase disease surveillance activities, emergency planning for likely emerging disease outbreaks and strategies for mitigating the impact of climate change on disease incidence in New England.

Climate Change

The climate of the earth is always in a state of change. Over millennia temperatures have risen and fallen due to multiple natural processes many of which are not well understood. Scientists theorize that past climate shifts have been caused by changes in the earth's orbit (NASA, 2010), tectonic plate movements, (Molnar and Martinod,1993) volcanic eruptions, asteroid strikes, sun spot cycles, and CO_2 build up from methane releases (Korhola et al., 2010, Luder and Stocker, 2003). During the Miocene Epoch even small temperature changes seemed to have precipitated extensive environmental changes, causing forests to become grasslands and large browsers to turn into smaller gazers (Kürschner et al., 2008). Short warming and cooling periods have been recorded even in recent history. In the Middle Ages, Europe experienced the Medieval Warm Period (MWP) and later in the mid 1600s what is known as the "Little Ice Age."

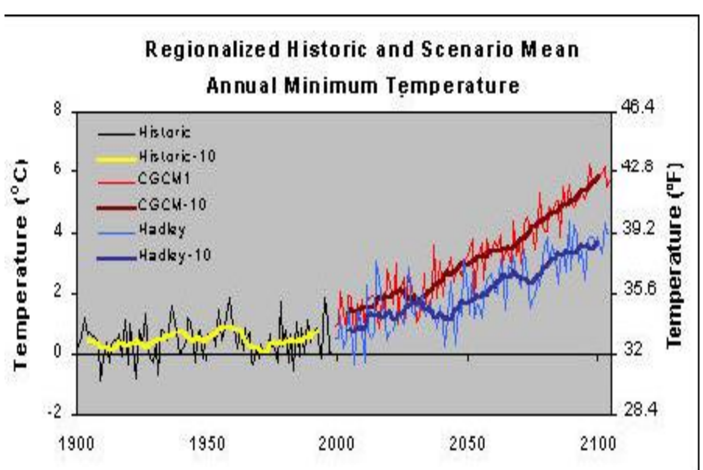
Currently there is a great deal of controversy over the use of the data documenting climate change. There are allegations that the Intergovernmental Panel on Climate Change (IPCC) may have suppressed information or improperly cited sources of information that contradicted the official projected rates of change (WWF, 2010). While the scope and causes of climate change may be controversial, there is little disagreement that sea levels, arctic ice, CO_2 levels and global surface temperatures are all changing by significant amounts (NASA, 2010) and are projected to continue to change (NERA, 2010).

New England has seen a weighted average temperature rise of 0.74 - 1.11° C over the last 100 years (NERA, 2010; Trombulak et al., 2004). Ledneva et al., (2004) studied the logs of a naturalist in Massachusetts and determined that 22 species showed earlier activity in the study area over the course of 30 years ending in 2002.

Figure 1 from the New England Regional Assessment (NERA) of the U.S. Global Change Research Project displays the historic regional temperatures in New England plus two models of projected temperature changes. The Hadley model projects an increase of 6° F in annual minimum temperatures and a 30% increase in participation. The Canadian Model projects a 10° F increase in minimum temperatures and some increase in precipitation. Figure 2 from the NERA (2010) project displays the regional weighted change in historic temperature changes in New England from 1895 to 1999. New Hampshire has seen the largest increase in temperatures rising 1.8° F over the 104 year period. The regional weighted temperature rise was 0.74° F (NERA, 2010).

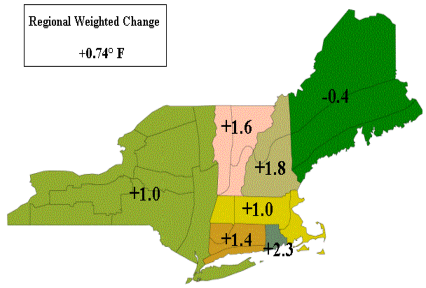
Vermont hardwood forests from 1965 to 2005 have moved upslope about 100 meters and changes in animal and bird migration patterns have been noted in the northeast (Global Change, 2010). Whether these changes are being caused by anthropomorphic activities or are the results of natural cycles, temperatures are rising. Increasing temperatures will likely cause changes to the environment and are strongly associated with increased morbidity and mortality rates (Chung, et al., 2009; Ford et al., 2009).

Figure 1: New England Regionalized Mean Annual Temperature Projections



NERA, 2010

Figure 2: New England & New York Temperature Changes (°F) Between 1895 and 1999



NERA, 2010

Warming trends are not occurring evenly around the globe. Higher latitudes, winters and nights are experiencing disproportionally more warming (Epstein, 2001). Every species has a viable temperature range or growing season. This range is likely to be narrower for juveniles of the species, making it more likely that even small temperature changes will impact many species (Jackson et al., 2009). Since species are interrelated, changes in one group can profoundly affect others. Climate changes have occurred rapidly in the past sometimes fluctuating widely over the course of a few decades. These kinds of fluctuations can sometimes alter population densities in a very short period of time such as occurred in the Southwest (Jackson et al., 2009) with the Anasazi.

CO_2 levels are increasing and are at levels not seen in the last 650,000 years (MacDonald, 2010). Increased levels of CO_2 have been shown to increase plant growth rates (MacDonald, 2010) and the production of pollens (Ladeau and Clark, 2006; Stinson and Bazzaz, 2006), including those associated with allergies.

Warming patterns are often associated with rising rain fall levels (Gardner, 2009), melting glaciers, rising sea levels (NOAA, 2010), and increased winds (Epstein, 2000, 2005). There will be direct and indirect effects from these changes. Direct effects include increased deaths due to hotter summers, more storms, more flooding (Kurane, 2009) and more droughts. Hotter summers mean more allergens and smog (Epstein, 2000, 2005). Heat-shock cases increase dramatically above 32° C. More people will die of heat-stroke and conditions exacerbated by heat and smog like cardiovascular and respiratory diseases (Kurane, 2009).

Indirect effects of climate change will include an increase in diseases (Epstein, 2001; Kurane, 2009) both infectious and environmental. Increased rain and flooding are associated with water and water supplies contaminated with bacteria (Ford et al., 2009) such as *Cryptosporidium* (Epstein, 2005) and *Vibrio cholera* (Epstein, 2000). Crops are vulnerable to contamination with *Escherichia coli,* though this is less likely in New England because of its good sanitary infrastructure (Kurane, 2009). Climate change could cause disruptions in food supplies as growing seasons and rain fall change. Animals could be affected by habitat changes. Malnutrition could make people and animals more susceptible to diseases (Epstein, 2000). Sewage and fertilizer washing into bodies of water could cause toxic algae blooms which could affect fishing areas and water supplies (Epstein, 2000). New diseases can cross over into animals causing them to lose weight and sometimes protective hair (Rand, 2007).

Extreme weather events may cause the most immediate public health risks, including lung diseases associated with fires during droughts, allergies due to increasing molds from flooding, mosquitoes borne diseases such as West Nile Virus (WNV) after extreme rain events and waterborne diseases such as *Cryptosporidium* infections after floods (Epstein, 2001).

Direct Effect Diseases of Interest

Heat Related Diseases

Rising temperatures create an increased risk of direct effect heat related deaths especially in the Northeast and Northwest regions of the United States (Reid et al., 2009). Without proper precautions such as access to air-conditioning, exposure levels to extreme heat for extended periods can exceed the body’s ability to self regulate its internal temperature, leading to increased death rates. Damage to the brain and other organs can occur when the body is unable to cool itself adequately (CDC, 2010). The Centers for Disease Control and Prevention (CDC) reported that 8,015 excessive heat related deaths occurred in the United States (USA) from 1979 to 2003. That is more people than died from floods, earthquakes, tornadoes, lightning and hurricanes combined (CDC, 2010).   Numerous studies document the rise in medical calls during prolonged heat waves (Basu and Samet, 2002) such as was experienced in Europe in 2003 (Garcia-Herrera et al., 2010).

Reid et al, (2009) created a cumulative national heat vulnerability index for the entire United States, mapping regions most at risk of health problems related to rising temperatures. Figure 3 displays a table showing the risk factors used by Reid et al. (2009) to develop their index. Exposures most associated with heat related health risks were a lack of air-conditioning, lower socio-economic status (SES), socially isolated individuals, a higher percentage of elderly and those with diabetes. Figure 4 displays the national map created by Reid et al. (2009) to display the increased health risks in regions with risk factors that were identified as highly associated with increasing temperatures. Much of southern New England is included in the most vulnerable areas. Reid et al. (2009) also concluded that although all large metropolitan areas will have much higher numbers of deaths due to prolonged heat waves, rural areas will show higher percentage increases. Much of New England can be classified as rural, making New England more vulnerable to heat associated diseases.

O'Neill et al. (2003) examined heat related deaths in a study of seven cities, including New Haven, Connecticut and found that overall there was a 5% increase in deaths when apparent temperatures were 29° C instead of 15° C. Out-of-hospital deaths increased over 10% and deaths for blacks increased 8.6% at this higher temperature. New Haven, Connecticut income is 55% of the state average. High school education levels are 10% lower than the state average of 84% (Census, 2010). These lower income and education levels indicate that this study may overstate the magnitude, but not the potential risk in New England from increasing temperatures.

Figure 3: Loading Factors for heat vulnerability variables.

Factor loadings for heat vulnerability variables for the four retained varimax-rotated factors based on data from 39,794 census tracts.

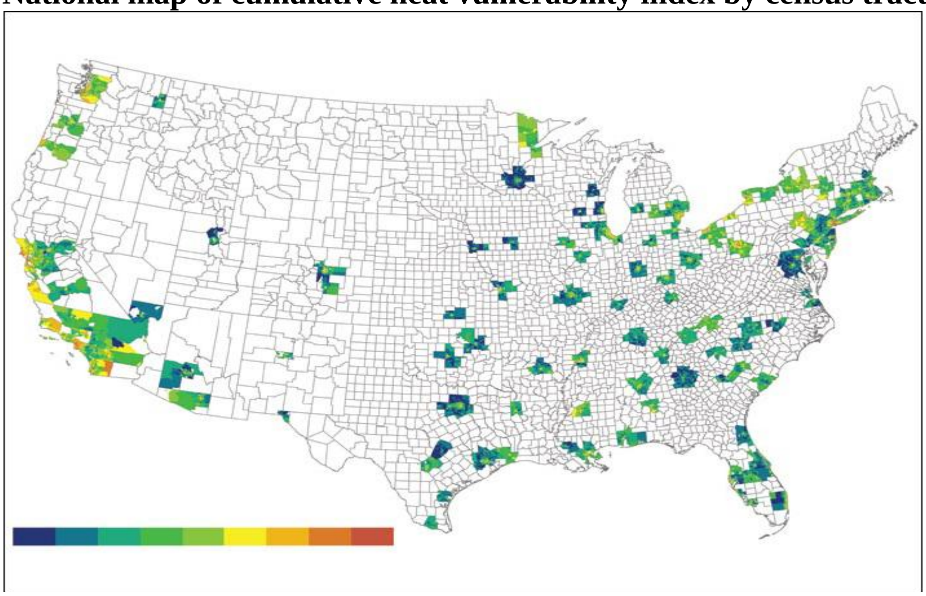
	Factor 1:social/environmental vulnerability	Factor 2:social isolation	Factor 3:prevalence of no AC	Factor 4:proportion of elderly/diabetes
Diabetes	0.37	–0.10	0.07	0.78
Below poverty line	0.87	0.18	–0.05	–0.03
Race other than white	0.85	–0.05	0.03	0.02
Live alone	–0.06	0.91	–0.002	0.16
Age ≥ 65 living alone	0.19	0.87	0.001	–0.06
Age ≥years	–0.32	0.38	–0.04	0.67
Less than high school diploma	0.85	–0.06	–0.05	0.07
Not green space	0.54	0.33	0.31	0.13
No central AC	0.02	0.02	0.92	0.06
No AC of any kind	–0.01	–0.03	0.92	–0.03

Absolute values > 0.4 are the most significant loadings on that factor.

Cumulative heat vulnerability index values7–101112131415161718–22

Reid et al., 2009

Figure 4. National map of cumulative heat vulnerability index by census tract (*n* = 39,794).



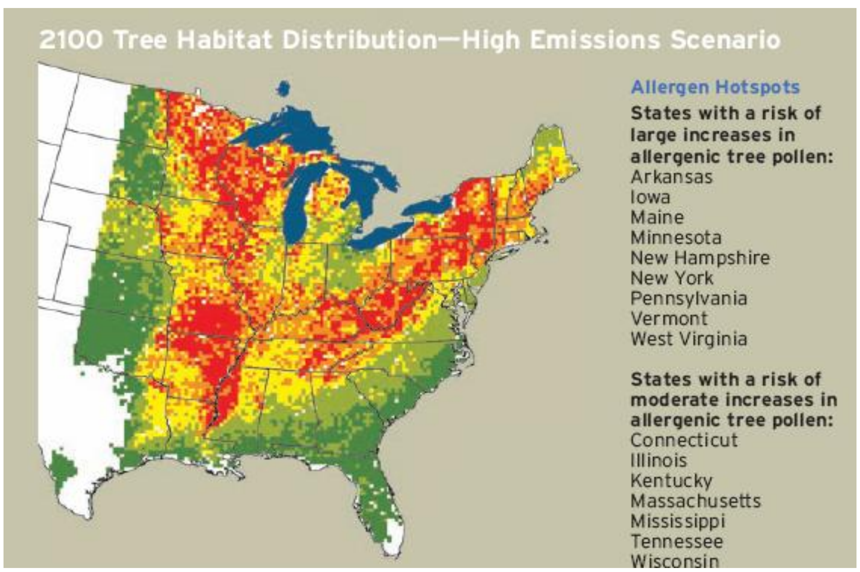
Reid et al. 2009

Allergies

Allergy seasons have been extended due to earlier spring buds (Miller-Rushing et al., 2008) and later frosts (Bradshaw and Holzapfel, 2001). Houle (2007) found that forests in the

Northeast and eastern Canada are flowering two to six days earlier than they did 100 years ago. Allergies are positively associated with atmospheric pollen counts (Frentz, 2001). Allergies and asthma cause suffering, increased sick days and are estimated to cost the USA more than 32 billion dollars annually (Staudt et al., 2010). About 40 million people in the USA suffer from plant based hay fever allergies (Gilmour et al., 2006). Figure 5 is taken from the World Wildlife Federation's 2010 report on climate change and allergies and shows the projected impact on New England if greenhouse emissions continue at a high rate for the next 100 years (Staudt et al., 2010).

Figure 5: Projected increases in allergenic tree pollen with high greenhouse gas emissions



Staudt et al., 2010

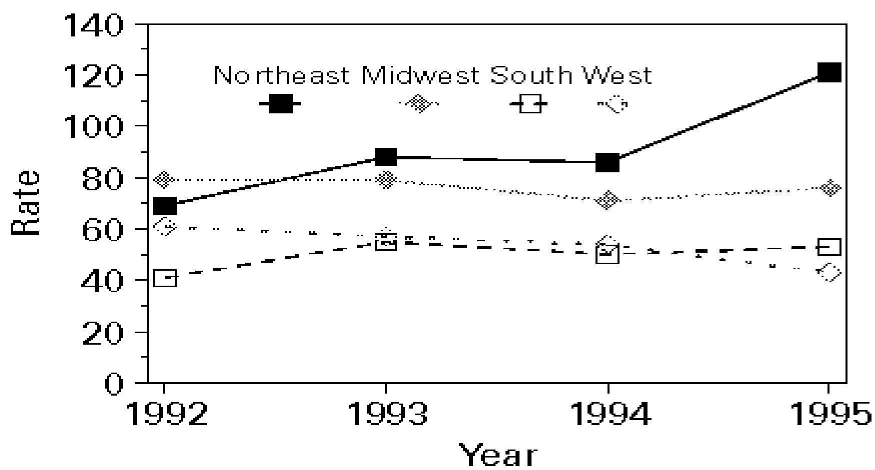
Because of its role in photosynthesis plants are directly affected by CO_2 levels. Since 1960, CO_2 levels have risen 22% (Ziska et al., 2009). Most plants do not receive the optimum amount of CO_2 needed to function at peak efficiency (Ziska et al., 2009) so increases in CO_2 are projected to amplify plant growth rates. Wayne et al., (2002) found that doubling CO_2 levels resulted in a 61% increase in pollen produced by ragweed, *Ambrosia artemisiifolia* L. (Linnaeus). Poison ivy likewise could play a substantial role in increased morbidity rates. There are 350,000 cases every year in the USA of contact dermatitis (Staudt et al., 2010). Increased CO_2 levels have been shown to increase growth rates and toxicity levels of poison ivy (Staudt et al., 2010), potentially creating higher rates of severe contact dermatitis.

Asthma and Chronic Lung Diseases

Asthma is a chronic condition characterized by inflamed airways and episodes of difficulty breathing. Symptoms include wheezing, coughing, and shortness of breath. The exact etiology of asthma is not well understood. CDC estimates that over 23 million Americans currently have asthma (CDC Fastats, 2010). Since 1992, the Northeast region has consistently experienced higher rates of asthma and asthma related Emergency Room (ER) visits (Moorman et al., 2007). Figure 5 shows the estimated rates of ER visits primarily due to asthma by region.

Figure 5: ER visits primarily for Asthma by region and year

FIGURE 6. Estimated average rates* of emergency room visits for asthma as the first-listed diagnosis, by region and year — United States, National Hospital Ambulatory Medical Care Survey, 1992–1995

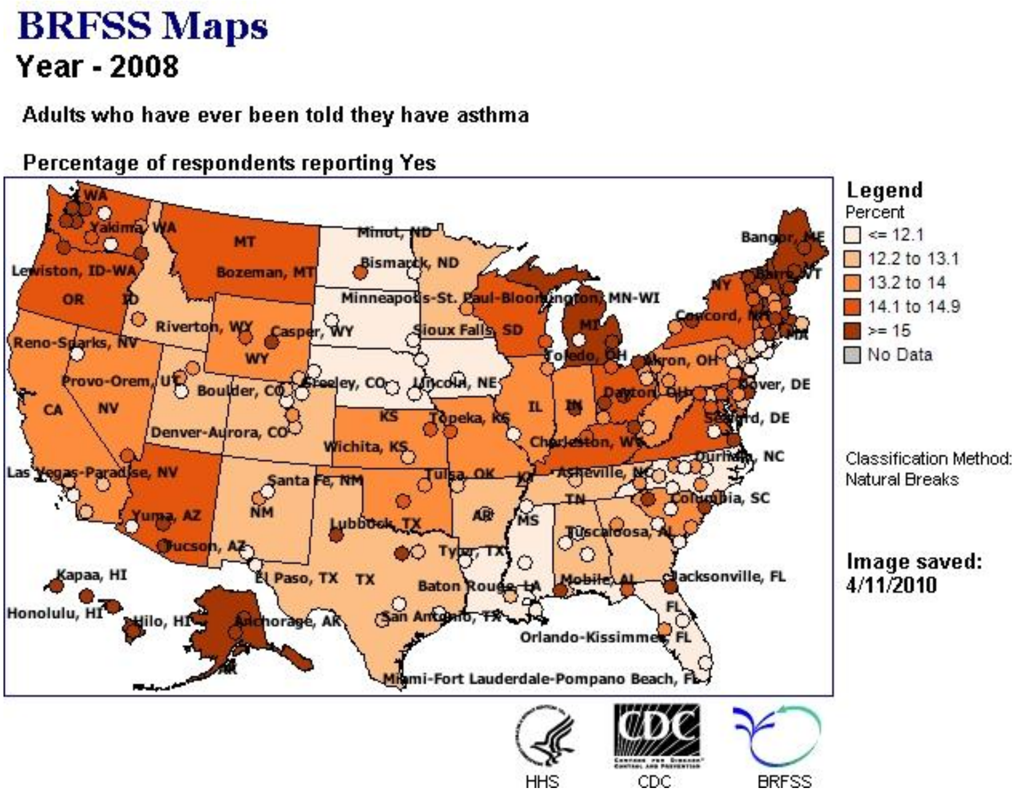


*Per 10,000 population. Race-, sex-, and age-adjusted to the 1970 U.S. population.

Manninao et al., 1998

Figure 6 is a slide from the CDC's Behavioral Risk Factor Surveillance System (BRFSS, 2010) geographic information system (GIS) mapping service which also indicates that the Northeast region experiences a substantially elevated incidence of asthma. Climate changes that result in increases in pollen counts are likely to result in an increased incidence of allergic disease symptoms and asthma (Ziska et al., 2008).

Figure 6: Adults who have ever been told they have asthma.



A prospective cohort study of 14 patients in an outpatient pulmonary rehabilitation program in the Hartford, Connecticut area looked at the association between weather and pulmonary function in these patients. Mann et al. (1993) found that in summer, higher temperatures were positively associated with increased morning dyspnea or difficulty breathing. The greatest predictor of increased morning dyspnea was a combination of increased lower respiratory tract infection (LRI), higher mean temperature and a rise in barometric pressure. In New England in the summer, rising barometric pressure is often associated with hot, hazy, humid weather (Zielinski and Keim, 2003). It is likely that increasing summer temperatures will be associated with increasing asthma related morbidity.

Indirect Effect Diseases of Interest

Bacteria

Indirect effect emerging and resurging diseases of interest in New England can be divided into those that affect animals and those that affect plants. Many animal diseases infect both humans and mammals with insects as the vectors. Beneficial insects like bees are also affected by diseases (van Engelsdorp, 2009). A few diseases have been shown to infect both animals and plants. *Vibrio cholerae*, which causes cholera in humans, has been found in biofilms on plants (Types of Bacteria, 2010; Watnick et al., 1999). Enteric bacteria such as *Salmonella* and *E. coli* can also colonize inside the internal structure of plants meaning that the infectious bacteria cannot be removed by surface cleaning efforts. Higher temperatures are also associated with higher colonization rates of these types of bacteria (Tyler and Triplett, 2008), increasing the risk of significant plant borne human infections as temperatures rise.

Arboviruses

An increase in Arboviruses (arthropod-borne viruses) is likely to have the largest disease impact in New England due to increasing temperatures. The West Nile Virus outbreak that started in New York in 1999 represented the largest arboviral encephalitis outbreak ever in North America (Weaver and Reisen, 2010). With today's air travel, any infectious disease in the world is only 24-hours away. Since insects often act as vectors and transmit diseases to other animals, including humans, an indirect result of rising temperatures and rain fall is the likelihood that mosquito ranges will be expanded, making new regions vulnerable to mosquito borne diseases such as Dengue Fever, Malaria, and Ross River Encephalitis (Kurane, 2009). Other mosquito borne diseases of concern include West Nile Virus, Saint Louis Encephalitis and Eastern Equine

Encephalitis (EEE) (Gilchrist, 2009). EEE is a deadly disease to both humans and horses, though there is an EEE vaccine for horses.

Recent Northeastern America warming trends have been due more to increases in temperature minima which results in earlier springs and later falls (Bradshaw and Holzapfel, 2001) than temperature extremes. Bradshaw and Holzaphfel (2001) have documented rapid genetic shifts in the *Wyeomyia smithii* pitcher plant mosquito which they attribute to climate changes. Animals and plants use photoperiodic light cues to help them determine when they should grow and reproduce and when they should hibernate or go dormant. The critical photoperiodic responses for arthropods increase evenly with higher latitudes and elevations (Bradshaw and Holzapfel, 2001). Bradshaw and Holzapfel have for 30 years studied photoperiodic changes in *W. smithii*, which ranges from the Gulf of Mexico to Northern Canada. The photoperiodic response is a highly inherited trait (Bradshaw and Holzapfel, 2001). *W. smithii* samples collected in 1972 were compared with those collected in 1999. The results indicate that the *W. smithii* mosquitoes have genetically adapted to lengthening growing seasons trends by shifting to shorter photoperiods in as little as five years. These changes have been greater in northern areas than southern (Bradshaw and Holzapfel, 2001).

West Nile Virus

West Nile Virus (WNV) is a *Flavivirus* and was first identified in 1937 in Uganda (Petersen and Roehrig, 2001). The Centers for Disease Control and Prevention (CDC) reports that symptoms range from none or very mild flu-like illness to death due to paralysis (poliomyelitis), swelling of the brain (encephalitis) or inflammation of membranes surrounding the brain or spinal cord (meningitis). In 1999 WNV made its first known appearance in the USA in the New York City area with 62 known human cases and 7 deaths (Beasley et al., 2004).

Since then there have been an estimated 200,000 illnesses, 20,000 cases and 770 deaths; making WNV the largest vector-borne disease in this continent (Kilpatrick et al., 2006).

WNV is a zoonotic disease found mostly in birds and transmitted primarily by the bite of an infected *Culex pipiens* mosquito. Kilpatrick et al. (2006) showed that the *Culex pipiens* mosquito shifted its primary feeding pattern that summer and fall from its preferred host the American Robin, *Turdus migratorius* and other birds, to humans. Molaei and Andreadis (2006) looked at feeding patterns of *Culiseta melanura* and *Aedes vexans* mosquitoes in Northeast, USA as secondary vectors of WNV. *Ae. vexans* fed most often on white-tailed deer and *Cs. melanura* fed mostly on birds, primarily robins. Miller et al. (2005) showed that white-tailed deer can be infected by WNV. Other studies have shown WNV infections in reptiles and amphibians as well as birds and mammals, substantially expanding the potential host reservoirs and mosquito vectors (Miller et al., 2005). Mosquito populations are positively correlated to warmer, wetter weather conditions (Soverow et al., 2009), making it likely that WNV will increasingly pose a risk to New England.

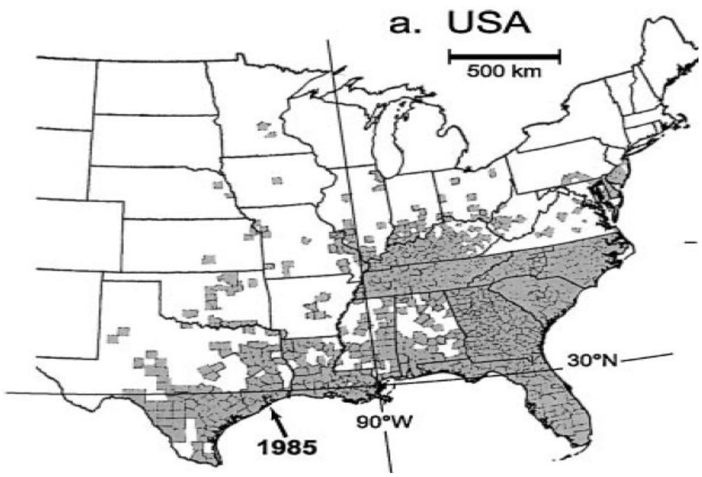
Yellow Fever, Dengue Fever and Malaria

Urbanization, growing susceptible populations and a wetter and warmer climate create the likelihood that one of these reemerging diseases will impact New England in the future due to the spread throughout the region of the mosquito vectors of these diseases. Both floods and drought create mosquito breeding opportunities in mud and still pools. While excessive heat could kill mosquitoes, heat also speeds up the reproduction of pathogens in mosquitoes (Epstein, 2000) and increases the biting habits of the female mosquito (Epstein, 2005). The expansion of the *Aedes aegypti*, *Anopheles* and *Aedes albopictus* mosquitoes in the USA has been well documented. The *Anopheles* mosquito is widely spread throughout the USA, including most of

New England (CDC, 2010). Hutchinson et al. (2008) documents 62 species of mosquitoes in Pennsylvania, including all these disease carrying mosquitoes.

March 25, 2008 at the Biannual State Public Health Vector Control Conference in Fort Collins, Colorado, Chester G. Moore presented slides showing that New England already had a small number of *Aedes aegypti and Aedes albopictus* mosquitoes reported, and widespread distribution of the *Ochlerotatus japonicas* mosquito. Figure 7 by Lounibos (2002) shows the distribution of the *Ae. Albopictus* in the USA and its movement up the coast towards New England.

Figure 7: Recent distributions of *Ae. Albopictus* in the USA



Lounibos, 2002

Malaria is a parasitic infection carried by the *Anopheles* mosquito. Small outbreaks of locally transmitted malaria have occurred in five states, including New York and the city of Toronto, Canada (Epstein, 2001). Until 1950 malaria was endemic in the USA. Malaria and dengue fever outbreaks have been seen in past decades in un-tropical countries such as Holland and at higher elevations and latitudes than is commonly assumed (Reiter, 1998). There are about 1,500 cases of malaria in the USA every year, mostly among travelers. From 1957 to 2009 there were 63 outbreaks of locally transmitted mosquito-borne malaria in the USA. A single sporozoite *Plasmodium falciparum* parasite acquired through the bite of an infected female

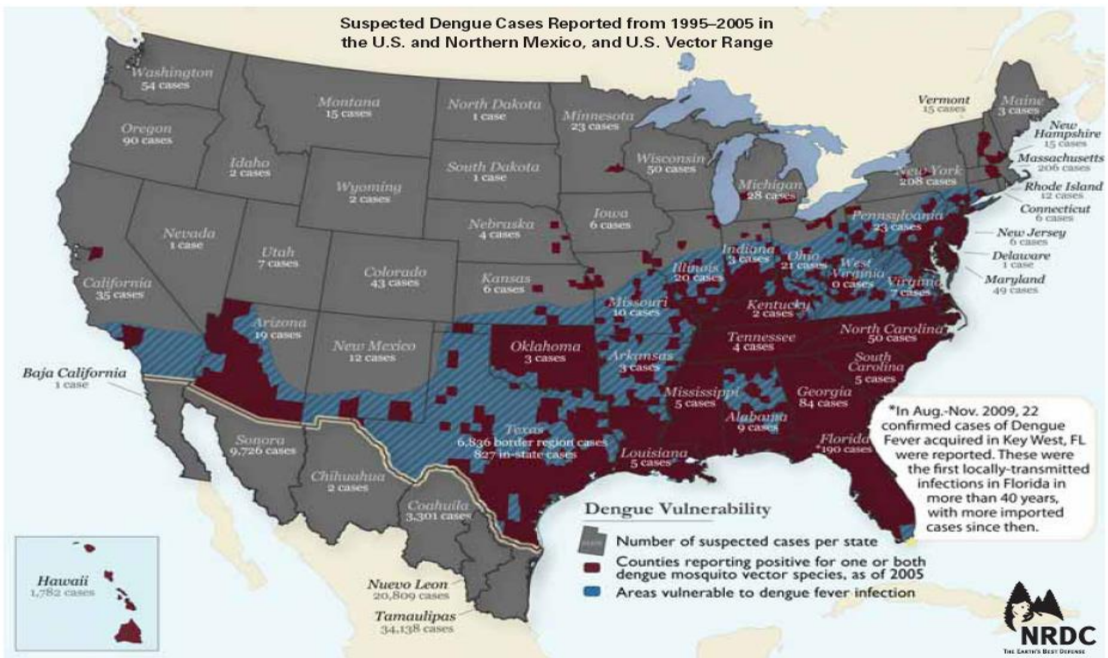
Anopheles mosquito can cause malaria. The parasite invades the liver and within six days grows a daughter cell that erupts into the blood stream, spreading the disease. Some of these parasite cells can lie dormant in the liver for years, reoccurring every two to four years (CDC FactSheets, 2010). There is no vaccine against malaria.

Yellow Fever is transmitted from human to human by the *Aedes aegypti* mosquito. The yellow fever outbreak in Philadelphia in 1793 killed more than 4,000 people and severely disrupted city commerce when one-third of the population fled in panic (Eisenberg, 2007). Since 1987 there has been a reemergence of yellow fever worldwide with 18,735 reported yellow fever cases and 4,522 deaths (Robertson et al., 1996). Yellow Fever can cause severe infection, leading to liver failure and jaundice or a yellowing of the skin and the whites of the eyes. This jaundice is what gave this disease its name. Severe yellow fever infections can be fatal if not treated promptly (CDC FactSheets, 2010).

Although there is a yellow fever vaccine, it carries substantial risk for older people especially those over 60 year old (Massad et al., 2005). The main non-urban reservoir for yellow fever is monkeys, making this disease hard to eradicate. The last epidemic in the USA was in New Orleans in 1905 (CDC, 2010). There is little immunity in the current population and vaccination rates are very low as vaccine is only given to travelers in outbreak areas.

In the northern hemisphere the *Aedes aegypti* mosquito is the principal mosquito vector of dengue viruses. Dengue is endemic in Northern Mexico (CDC, 2010) creating a reservoir of infection that could travel into the USA. As was seen in a 2001 outbreak in Hawaii *Aedes albopictus* (CDC, 2010) can also transmit Dengue fever and is widely distributed in the eastern USA (Knowlton, et al., 2009). Dengue Fever is caused by four closely related dengue viruses; DENV 1, DENV 2, DENV 3, or DENV 4 (CDC, 2010). Disease symptoms range from mild to severe with the greatest risks to those who develop dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS), (CDC, 2010). Figure 8 by Knowlton, et al., (2009) shows both the reported cases of Dengue Fever and the current ranges of its mosquito vectors. Although New England only has sporadic cases and mosquito vectors, this is may change rapidly if the climate continues to get warmer and wetter.

Figure 8: Suspected Dengue Cases and U.S. Vector Range



NOTE: Dengue vulnerability in the United States. Among the social and environmental factors that increase community vulnerability to dengue and other infectious diseases are poor municipal infrastructure and frequent storm damage to homes. Red areas of the map show U.S. counties that have reported the presence of one or both of the mosquito species (*Aedes aegypti* and *Aede albopictus*) that can potentially transmit dengue fever; blue regions highlight the area encompassing most of the positive counties. Numbers of suspected cases of dengue infection reported fro 1995–2005, inclusive, are shown below each state name.[44] Reported counts of suspected dengue fever cases are also included for the six Mexican states that border the United States.[45]

Knowlton, et al., 2009

Tick borne Diseases

Tick borne diseases are also likely to expand in range and intensity in New England as tick prevalence is strongly correlated to warm temperatures (Rogers et al., 2007). Lyme, babesiosis, and ehrlichiosis already cause significant morbidity and mortality in southern New England. Of the ten tick borne diseases recognized in the USA, three of them were identified in the last forty years (Gratz, 1999). Lyme disease is of particular interest in New England where a dramatic increase in the number of Lyme disease infections has been documented. This is likely due to the increasing range of the tiny *Ixodes* deer tick (Kurane, 2009), warmer temperatures, increased populations at risk and an expansion of the mice and deer reservoirs.

Lyme Disease

Lyme is caused by the bacterium *Borrelia burgdorferi*, occurs worldwide and is called a resurging disease (Gratz, 1999) because it is rapidly spreading probably due to increased reservoir hosts, population densities as well as increased tick vectors. The national distribution for Lyme during the period 1994 -1999 and the potential areas for future growth in Lyme disease are shown in Figure 9 (Ashley and Meentemeyer, 2004), making Lyme an increasing risk in most New England counties.

Figure 9: Increasing Lyme Cases and Suitable Habitat in the USA, 1999

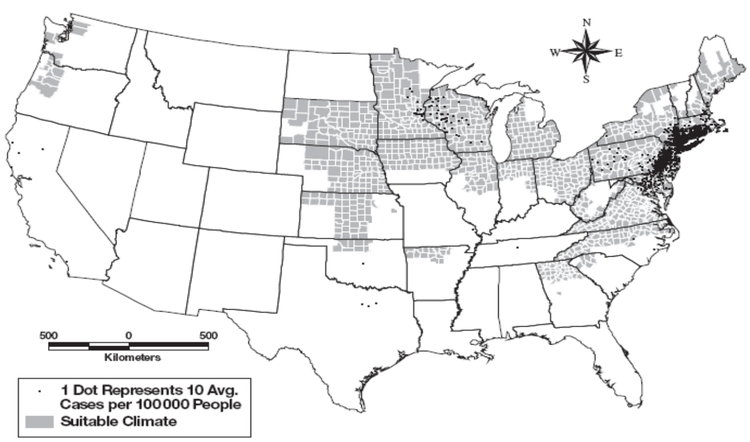


Fig. 12. Potential Climatic Risk Map based on a 'climatic envelope' model. This model is constrained by the average (1994 to 1999) AMJ total precipitation, AMJ average temperature, and AMJ total soil moisture surplus values of the middle 90% of counties with >10 average cases per 100000 people. Counties shaded in grey are within the suitable climate range specified by the model

Ashley and Meentenmeyer, 2004

Temperature, moisture and humidity play important roles in prolonging the period of time that ticks are active and able to infect hosts. The presents of dense, moist forest litter is important for the survival of the tick during hot, dry weather. Lyme has a two-year life cycle that includes the egg stage and three blood meal stages (Penn State, 2010). In late spring the eggs hatch into larvae that are not infected. The larvae take their first blood meal from mice and other small birds and animals where the ticks acquire the Lyme spirochetes from the host reservoir. The larvae are inactive over the winter. In the spring they molt into the nymph stage and can pass on the infection to new host. In the fall nymphs molt into adults that feed and mate on large animals like deer, then fall off and lay their eggs, completing the cycle. Nymphs and adults are the vectors for Lyme. It is critical that the larvae feed after an infected nymph has passed on disease to the host.

There is a direct correlation between temperature changes and Lyme transfer rates. Warmer temperatures are associated with an increase in the window of opportunity where infection transfers can occur (Gatewood et al., 2009), increasing the risks of Lyme disease in New England as temperatures rise.

Babesiosis

Babesiosis is a tickborne zoonotic, intraerythrocytic protozoa found in red blood cells. The parasites break down red blood cells, causing anemia and leading to organ failure and death, especially in people with compromised immune systems (Gray et al., 2010). Symptoms include fever, chills, weight loss, and fatigue. There are two main groups of this protozoan genus *Babesia*, *Babesia microti*-like and *Babesia* sensu stricto with fewer effective treatments for *Babesisa* microti-like (Gray et al., 2010). Risk factors are the rising deer and tick populations, infection from blood transfusions and the expanding number of immuno-compromised individuals. In 1999 there were 50 known cases of babesiosis infection by blood transfusion and the available post donation screening tests are unreliable and expensive (Ngo and Civen, 2009). Educating medical providers in identifying this disease along with prevention of initial infections are the two most important strategies for preventing babesiosis.

Other Diseases of Interest

Other disease such as plague and hantavirus could also become a significant concern (Epstein, 2000). Climate changes may also reduce the number of insect predators like bats, birds and ladybugs. Bat White Nose Syndrome (WNS) which is killing bats in New England could be associated with climate changes (Blehert, et al., 2009). WNS has also been identified in France, though so far the bats there have remained healthy (Puechmaille et al., 2010). Whatever the cause, declining bat populations in New England will likely result in an increase in total insect vectors like mosquitoes.

Only two outbreaks of Tuleremia have ever been recorded on Martha's Vineyard, the first in 1978 and the second in 2000 (Feldman et al., 2001). A tick borne disease called Powassan virus has been found in Canada and the United States (Gilchrist, 2009). Asian tiger mosquito could become more prevalent and are carriers of Chikungunya virus (CHIKV) and Rift Valley Fever (Gilchrist, 2009). An outbreak of CHIKV transmitted by the *Aedes Albopictus* mosquito occurred in Italy in 2007, infecting hundreds of people (Weaver and Reisen, 2010).

New England could experience outbreaks of plague, typhus carried by lice, leishmaniasis, a parasitic disease carried by the sandfly, trypanosomiasis or African Sleeping Sickness carried by parasitic protozoan, Rocky Mountain Spotted Fever, relapsing fever carried by lice or ticks, Chagas (American trypanosomiasis) disease carried by insects (Click Lambert et al., 2008). These so called tropical diseases are increasingly being documented in the USA, especially in low income, minority populations (Hortez, 2009). Figure 10 shows a comparison of neglected tropical disease (NTD) prevalence in selected minority populations in the USA. While the rates in the USA are substantially lower than in developing countries, significant percentages of minority populations in the USA could be affected (Hortez, 2009).

Figure 10: Comparison of NTD Prevalence and Incidence Rates between Selected Minority Populations in the United Sates and in Selected Developing Countries

Neglected Tropical Disease	Underrepresented Minority US Population	Prevalence	Developing Country	Prevalence	Reference
Trichomoniasis	African Americans	13%–29%	Nigeria	38%	[4–7]
Toxocariasis	African Americans	21%	Nigeria	30%	[4,8,9]
HIV/AIDS	African Americans	2%	Nigeria	3%	[10,11]
Cysticercosis	Hispanic Americans	2%	Mexico	44%	[4,12,13]
Dengue	Hispanic Americans	2%	Mexico	7%	[4,14]
Invasive haemophilus disease	Native Americans	20.5 cases per 100,000 (age<5)	Mozambique	125 cases per 100,000 (age<5)	[20,21]

doi:10.1371/journal.pntd.0000405.t001

.
Hortez, 2009

Discussion

Some species and areas may benefit from climate change. Some colder areas may become more temperate and have a longer growing season. Some waters that used to be covered in ice may become navigable such as is expected to occur in Canada along the Northwest Passage. Some arid areas may become wetter as others become more arid as is occurring now in parts of Australia. Climate changes may benefit some, but it is likely that most forecasted disruptions due to climate change will increase morbidity and mortality in New England, far outweighing any theoretical benefits resulting from climate change. Public Health authorities need to increase surveillance activities to identify emerging threats to individuals and at risk populations. Emergency planning efforts must include strategies for dealing with public health threats due to increasing temperatures and rain fall, especially during and after weather extremes. Effective proactive interventions and programs need to be developed to address the rising incidence of vector borne diseases in New England and the potential for an outbreak of a novel or emerging disease brought to this area by travelers. Local, State and Federal public health authorities should prepare coordinate risk communication strategies and messages for public health emergencies due to increasing climate related diseases. Research should continue to identify risks and mitigation strategies for increasing diseases due to climate change.

References

Ashley, S.T., Meentemeyer, V., 2004. Climatic analysis of Lyme disease in the United States. Clim. Res. 27, 177-187.

Basu, R., Samet, J.M., 2002. Relation between elevated ambient temperature and mortality: A review of the epidemiologic evidence. Epidemiol. Rev. 24, 190-202.

Beasley, D.W., Davis, C.T., Whiteman, M., Granwehr, B., Kinney, R.M., Barrett, A.D., 2004. Molecular determinants of virulence of West Nile virus in North America. Arch. Virol. 35-41.

Blehert, D.S., Hicks, A.C., Behr, M., Meteyer, C.U., Berlowski-Zier, B.M., Buckles, E.L., Coleman, J.T.H., Darling, S.R., Gargas, A., Niver, R., Okoniewski, J.C., Rudd, R.J., Stone, W.B., 2009. Bat white-nose syndrome: An emerging fungal pathogen? Sci. 323, 227.

Bradshaw, W.E., Holzapfel, C.M., 2001. Genetic shift in photoperiodic response correlated with global warming. Proc. Natl. Acad. Sci. U. S. A. 98, 14509-14511.

BRFSS, Behavioral Risk Factor Surveillance System, CDC, http://apps.nccd.cdc.gov/gisbrfss/select_question.aspx, accessed 4/11/10

CDC, Extreme Heat: A Prevention Guide to Promote Your Personal Health and Safety, http://www.bt.cdc.gov/disasters/extremeheat/heat_guide.asp, accessed 4/11/10

CDC FactSheets, http://www.cdc.gov/ncidod/dvbid/yellowfever/YF_FactSheet.html, accessed 4/18/10.

CDC Fastats, http://www.cdc.gov/nchs/fastats/asthma.htm, CDC, accessed 4/11/10.

Census, Quick Facts, http://quickfacts.census.gov/qfd/states/09/0952000.html, accessed 4/10/10.

Chung, J.-Y., Honda, Y., Hong, Y.-C., Pan, X.-C., Guo, Y.-L., Kim, H., 2009. Ambient temperature and mortality: An international study in four capital cities of East Asia. Sci. Total Environ. 408, 390-396.

Click Lambert, R., Kolivras, K.N., Resler, L.M., Brewster, C.C., Paulson, S.L., 2008. The potential for emergence of Chagas disease in the United States. Geospat. Health. 2, 227-239.

Eisenberg, L., 2007. Furor therapeuticus: Benjamin Rush and the Philadelphia yellow fever epidemic of 1793. Am. J. Psychiatry. 164, 552-555.

Epstein, P.R., 2000. Is global warming harmful to health? Sci. Am. 283, 50-57.

Epstein, P.R., 2001. Climate change and emerging infectious diseases. Microbes Infect. 3, 747-754.

Epstein, P.R., 2002. Climate change and infectious disease: Stormy weather ahead? Epidemiol. 13, 373-375.

Epstein, P.R., 2005. Climate Change and Human Health. N. Engl. J. Med. 353, 1433-1436.

Feldman K. A., Russell E., Lathrop, S.L., Matyas, B. T., McGuill, M., Schriefer, M. E., Stiles-Enos, D., Dennis, D. T., Petersen, L. R., Hayes, E. B., 2001. An Outbreak of Primary Pneumonic Tularemia on Martha's Vineyard. N. Engl. J. Med. 345, 1601-1606.

Ford, T. E., Colwell, R. R., Rose, J. B., Morse, S. S., Rogers, D. J., Yates, T. L., 2009. Using Satellite Images of Environmental Changes to Predict Infectious Disease Outbreaks. Emerg. Infect. Dis. 15, 9.

Frenz, D.A., 2001. Interpreting atmospheric pollen counts for use in clinical allergy: Allergic symptomology. Ann. Allergy Asthma Immunol. 86, 150-158.

Garcia-Herrera, R., Díaz, J., Trigo, R.M., Luterbacher, J., Fischer, E.M., 2010. A review of the European summer heat wave of 2003. Environ. Sci. Technol. 40, 267-306.

Gardner, L.R., 2009. Assessing the effect of climate change on mean annual runoff. J. Hydrol. 379, 351-359.

Gatewood, A.G., Liebman, K.A., Gwenaël Vourc'h, Bunikis, J., Hamer, S.A., Cortinas, R., Melton, F., Cislo, P., Kitron, U., Tsao, J., Barbour, A.G., Fish, D., Diuk-Wasser, M.A., 2009. Climate and tick seasonality are predictors of *Borrelia burgdorferi* genotype distribution. Appl. Environ. Microbiol. 75, 2476-2483.

Gilchrist, M.J., 2009. Emerging Infectious Diseases in New England from the Puritans to the Present. Clin. Mic. News. 31, 71-78.

Gilmour, M.I., Jaakkola, M.S., London, S.J., Nel, A.E., Rogers, C.A., 2006. How exposure to environmental tobacco smoke, outdoor air pollutants, and increased pollen burdens influences the incidence of asthma. Environ. Health Perspect. 114, 627-633.

Global Change Research, 2010. Our Changing Planet. The U.S. Global Change Research Program Fiscal Year 2010.

Gratz, N.G., 1999. Emerging and resurging vector-borne diseases. Annu. Rev. Entomol. 44, 51-75.

Gray, J., Zintl, A., Hildebrandt, A., Hunfeld, K.-P., Weiss, L., 2010. Zoonotic babesiosis: Overview of the disease and novel aspects of pathogen identity. Tic. Tick-borne Dis. 1, 3-10.

Hotez, P.J., 2009. One world health: Neglected tropical diseases in a flat world. PLoS. Negl. Trop, Dis. 3, e405.

Houle, G., 2007. Spring-flowering herbaceous plant species of the deciduous forests of eastern Canada and 20th century climate warming. Can. J. For, Res. 37, 505-512.

Hutchinson, M.L., Darsie, R.F., Spichiger, S.-E., Jones, G.E., Naguski, E.A., 2008. Annotated checklist of the mosquitoes of Pennsylvania including new state records. J. Am. Mosq. Control Assoc. 24, 1-5.

Jackson, S.T., Betancourt, J.L., Booth, R.K., Gray, S.T., 2009. Ecology and the ratchet of events: Climate variability, niche dimensions, and species distributions. Proc. Natl. Acad. Sci. U. S. A. 106, 19685-19692.

Kilpatrick, A.M., Kramer, L.D., Jones, M.J., Marra, P.P., Daszak, P., 2006. West Nile virus epidemics in North America are driven by shifts in mosquito feeding behavior. PLoS. Biol. 4, e82.

Korhola, A., Ruppel, M., Seppä, H., Väliranta, M., Virtanen, T., Weckström, J., 2010. The importance of northern peatland expansion to the late-Holocene rise of atmospheric methane. Quat. Sci. Rev. 29, 611-617.

Knowlton, K., Solomon, G., Rotkin-Ellman, M., 2009. Mosquito-Borne Dengue Fever Threat Spreading in the Americas. Natural Resources Defense Council.

Kurane, I. 2009. The emerging and forecasted effect of climate change on human health. J. Health Sci. 55(6), 865-869.

Kürschner, W.M., Kvaček, Z., Dilcher, D.L., 2008. The impact of Miocene atmospheric carbon dioxide fluctuations on climate and the evolution of terrestrial ecosystems. Proc. Natl. Acad. Sci. U. S. A. 105, 449-453.

Ladeau, S.L., Clark, J.S., 2006. Pollen production by *Pinus taeda* growing in elevated atmospheric CO_2. Funct. Ecol. 20, 541-547.

Ledneva, A., Miller-Rushing, A.J., Primack, R.B., Imbres, C., 2004. Climate change as reflected in a naturalist's diary, Middleborough, Massachusetts. Wilson Bull. 116, 224-231.

Lounibos, L.P., 2002. Invasions by insect vectors of human disease. Annu. Rev. Entomol. 47, 233-266.

Luder, T., Benz, W., Stocker, T.F., 2003. A model for long-term climatic effects of impacts. J. Geophys. Res. 108, 10-1.

MacDonald, G.M., 2010. Global warming and the Arctic: A new world beyond the reach of the Grinnellian niche? J. Exp. Biol. 213, 855-861.

Mann, M., Patel, K., Reardon, J.Z., Goldstein, M., Godar, T.J., ZuWallack, R.L., 1993. The influence of spring and summer New England meteorologic conditions on the respiratory status of patients with chronic lung disease. Chest., 103,1369-1374.

Mannino, D. M., Homa, D. M., Pertowski,, C. A., Ashizawa, A., Nixon, L. L., Johnson, C. A., Ball, L. B., Jack, E., Kang, D. S., 1998. Surveillance for Asthma – United States, 1960 – 1995. MMWR Morb. Mortal. Wkly. Rep. April 24, 1998 / 47(SS-1);1-28.

Massad, E., Bezerra Coutinho, F.A., Burattini, M.N., Lopez, L.F., Struchiner, C.J., 2005. Yellow fever vaccination: How much is enough? Vacc. 23, 3908-3914.

Miller, D.L., Radi, Z.A., Baldwin, C., Ingram, D., 2005. Fatal West Nile virus infection in a white-tailed deer (*Odocoileus virginianus*). J. Wildl. Dis. 41, 246-249.

Miller-Rushing, A.J., Inouye, D.W., Primack, R.B., 2008. How well do first flowering dates measure plant responses to climate change? The effects of population size and sampling frequency. J. Ecol. 96, 1289-1296.

Molaei, G., Andreadis, T.G., 2006. Identification of avian- and mammalian-derived bloodmeals in *Aedes vexans* and *Culiseta melanura* (*Diptera: Culicidae*) and its implication for West Nile virus transmission in Connecticut, U.S.A. J. Med. Entomol. 43, 1088-1093.

Molnar, P., England, P., Martinod, J., 1993. Mantle dynamics, uplift of the Tibetan Plateau, and the Indian monsoon. Rev. Geophys. 31, 357-396.

Moorman, J. E., Rudd, R. A., Johnson, C. A., King, M., Minor, P., Bailey, C., Scalia, M. R., Akinbami, L. J., 2007. National Surveillance for Asthma --- United States, 1980—2004. MMWR Morb. Mortal. Wkly. Rep. October 19, 56(SS08);1-14;18-54.

NASA, http://climate.nasa.gov/keyIndicators/ , climate change indicators, accessed 2/25/10.

NERA, New England Regional Assessment , U.S. Global Change Research Project, http://www.necci.sr.unh.edu/necci-report/rockbookexec.pdf, accessed 4/10/10.

Ngo V., Civen R., 2009. Babesiosis acquired through blood transfusion, California. Emerg. Infect. Dis. May.

NOAA, Ocean Service, http://oceanservice.noaa.gov/facts/sealevel.html, accessed 2/26/10

Staudt, A., Glick, P., Mizejewski, D., Inkley, D., 2010. Extreme Allergies and Global Warming. National Wildlife Federation.

O'Neill, M.S., Zanobetti, A., Schwartz, J., 2003. Modifiers of the temperature and mortality association in seven US cities. Am. J. Epidemiol. 157, 1074-1082.

Penn State, Department of Agriculture, 2010, http://ento.psu.edu/extension/urban/lyme-disease/life-cycle-of-the-blacklegged-tick, accessed 2/27/10

Petersen, L. R., Roehrig, J. T., 2001. West Nile Virus: A Reemerging Global Pathogen. Emerg. Infect. Dis. 7, 4.

Puechmaille, S.J., Verdeyroux, P., Fuller, H., Ar Gouilh, M., Bekaert, M., Teeling, E.C., 2010. White-nose syndrome fungus (*geomyces destructans*) in Bat, France. Emerg. Infect. Dis.16, 290-293.

Rand, P.W., Lacombe, E.H., Dearborn, R., Cahill, B., Elias, S., Lubelczyk, C.B., Beckett, G.A., Smith Jr., R.P., 2007. Passive surveillance in Maine, an area emergent for tick-borne diseases. J Med. Entomol. 44, 1118-1129.

Reid, C.E., O'Neill, M.S., Gronlund, C.J., Brines, S.J., Brown, D.G., Diez-Roux, A.V., Schwartz, J., 2009. Mapping community determinants of heat vulnerability. Environ. Health Perspect. 117, 1730-1736.

Reiter, P., 1998. Global-warming and vector-borne disease in temperate regions and at high altitude. Lancet, 351, 839-840.

Robertson, S.E., Hull, B.P., Tomori, O., Bele, O., LeDuc, J.W., Esteves, K., 1996. Yellow fever: A decade of reemergence. JAMA. 276, 1157-1162.

Rodgers, S.E., Miller, N.J., Mather, T.N., 2007. Seasonal variation in nymphal blacklegged tick abundance in southern New England forests. J. Med. Entomol. 44, 898-900.

Soverow, J.E., Wellenius, G.A., Fisman, D.N., Mittleman, M.A., 2009. Infectious disease in a warming world: How weather influenced West Nile virus in the United States (2001-2005). Environ. Health. Perspect.117, 1049-1052.

Staudt, A., Glick, P., Mizejewski, D., Inkley, D., 2010. Extreme Allergies and Global Warming. National Wildlife Federation.

Stinson, K.A., Bazzaz, F.A., 2006. CO_2 enrichment reduces reproductive dominance in competing stands of *Ambrosia artemisiifolia* (common ragweed). Oecologia. 147, 155-163.

Trombulak, S.C., Wolfson, R., 2004. Twentieth-century climate change in New England and New York, USA. Geophys. Res. Lett.31, L19202 1-4.

Tyler, H.L., Triplett, E.W., 2008. Plants as a habitat for beneficial and/or human pathogenic bacteria. Annu. Rev. Phytopathol. 46, 53-73.

Types of Bacteria, http://www.typesofbacteria.co.uk/bacteria-infect-plants.html, accessed 2/26/10 van Engelsdorp, D., Evans, J.D., Saegerman, C., Mullin, C., Haubruge, E., Nguyen, B.K., Frazier, M.A., Frazier, J., Cox-Foster, D., Chen, Y., Underwood, R., Tarpy, D.R., Pettis, J.S., 2009. Colony collapse disorder: A descriptive study. PLoS One. 4, e6481.

Watnick, P. I., Fullner, K. J., Kolter, R. , 1999. A Role for the Mannose-Sensitive Hemagglutinin in Biofilm Formation by *Vibrio cholerae* El Tor. J. Bacteriol. 181, 11, 3606-3609.

Wayne, P., Foster, S., Connolly, J., Bazzaz, F., Epstein, P., 2002. Production of allergenic pollen by ragweed (*Ambrosia artemisiifolia* L.) is increased in CO_2-enriched atmospheres. Ann. Allergy Asthma Immunol. 88, 279-282.

Weaver, S.C., Reisen, W.K., 2010. Present and future arboviral threats. Antiviral Res.85, 328-345.

WWF, World Wildlife Federation, http://www.wwfblogs.org/climate/content/statement-wwf-ipcc-feb2010, accessed 2/25/10

Zielinski, G. A., Keim, B. D., 2003. New England Weather, New England Climate. University of New Hampshire Press, New Hampshire.

Ziska, L. H., Epstein, P. R., Rogers, C. A., 2008. Climate Change, Aerobiology, and Public Health in the Northeast United States. Mitig. Adapt. Strat. Glob. Change. 13:607–613

Ziska, L. H., Epstein, P. R., Schlesinger, W. H., 2009. Rising CO2, climate Change, and Public Health: Exploring the Links to Plant Biology. Environ. Health Perspect. 117, 155-158.