4-12-2013

2012 Epizootic Hemorrhagic Disease in White-tailed Deer in Michigan: History, Consequences, and Predictions

Melissa Ann Schrauben
Grand Valley State University

2012 Epizootic Hemorrhagic Disease in White-tailed Deer in Michigan

History, Consequences, and Predictions

Melissa Ann Schrauben

4/12/2013

Honors Senior Project

Table of Contents

Part 1: Abstract……..……...…...3

Part 2: Introduction……..……...……...3

Part 3: Epizootic Hemorrhagic Disease……...……...4

3.1 Definition……...…..…..4

3.2 Symptoms……..……6

3.3 Historic Range…….…..8

3.4 Transmission……..9

3.5 Climatic Factors……..…….11

Part 4: 2012 Outbreak……...……..12

4.1 Reported Cases…….……12

4.2 Climatic Influence……16

4.3 Effects to Deer Population…….……..16

Part 5: Control Methods and Predictions…….…….17

5.1 Control Methods……..……18

5.2 Future Outbreak Predictions……..……..18

Part 6: Conclusion…….…..…….21

Part 7: Bibliography……..……..22

List of Figures

Figure1: Map of Epizootic Hemorrhagic Disease Occurrences……..13

Figure 2: Total Deer Count by Week…….…….……..14

Figure 3: Percent Lowland vs. Confirmed Cases…….……15

Figure 4: Percent Lowland by County…….……..…20

Abstract

The summer and fall of 2012 had a reported white-tailed deer death toll of 14,898 as a result of Epizootic Hemorrhagic Disease in Michigan. These cases include those which were confirmed by the Michigan Department of Natural Resources and those that were suspected. This makes the 2012 outbreak 13 times worse than the previous worst outbreak in Michigan which occurred in 2010. Observations of the number of deaths and spatial range of deaths due to EHD have shown an increasing trend over the past few years. Factors that influence EHD outbreaks include weather and climate, deer population size, and land type. Evidence suggests that there is a relationship between percent of lowland by township and number of confirmed EHD death. The most recent outbreak caused detrimental effects to some local deer populations, while regional deer populations were left relatively unharmed. There are no practical methods of controlling the spread of the disease once it has become established in a population. Predicting the severity and timing of an outbreak is difficult, but current lack of feasible control methods makes disease prevention the most logical option. The objective of this study was to investigate the specific factors intensified the 2012 outbreak of EHD in Michigan. In addition, this study aimed to determine the most practical means predicting time, location, and severity of future outbreaks.

Introduction

Epizootic Hemorrhagic Disease is not a new phenomenon to North America, or to Michigan specifically. There have been reported cases of EHD in Michigan in the past; however the worst recorded outbreak of the disease in Michigan occurred during the summer and fall of 2012. The recent outbreak resulted in a death toll of white-tailed deer of more than 13 times

greater than the previously reported worst outbreak. The nearly 15,000 deaths caused by EHD in 2012 were only the reported and investigated cases that were attributed to EHD. It is likely that this number is on the low end of actual deaths caused by the outbreak, as not every case could be studied and investigated. In addition, there were likely a large number of deaths that were never discovered. The facts about the outbreak raise questions as to its development and progression. What caused the 2012 outbreak of EHD to be the most severe outbreak the state of Michigan has ever seen? What are the identifiable signs of the disease and what is the normal mortality for white-tailed deer with the disease? What factors played a role in the intensification of its spread and the death rates? What role did weather and regional factors play in the recent outbreak? What are the impacts to regional and local deer populations, as well as implications for hunting? Are there possible factors that could help prevent or predict another severe outbreak? This study aims to address these questions.

Epizootic Hemorrhagic Disease

Definition

There are various forms of hemorrhagic diseases that have been known to infect ruminants around the world. Two of these diseases which can have a large toll on deer populations are Epizootic Hemorrhagic Disease and the Bluetongue Virus. Both are RNA viruses within the *Reoviridae* family, occurring mostly within white-tailed deer (*Odocoileus virginianus)* (Biek, 2007). These two diseases have virtually the same symptoms (QMDA, 2012). As a result, they can be misclassified with each other unless they are confirmed in the laboratory. A broad term for these diseases would be Hemorrhagic Diseases. Thomas M. Cooley, wildlife biologist

and pathologist for the Michigan Department of Natural Resources, stated that the severe outbreak in Michigan in 2012 was confirmed as EHD, with Bluetongue virus not being present.

"Epizootic Hemorrhagic Disease (EHD) is an acute, infectious, often fatal viral disease of some wild rudiments" (Epizootic Hemorrhagic Disease, 2013). There are three prevalent subtypes of EHD and five subtypes of Bluetongue Virus in North America (EHD Fact Sheet, 2012), which are all very similar in symptoms and pathology. The disease has been seen worldwide in a multitude of different strains. Most cases of EHD around the world involving white-tailed deer can be attributed to serotype EHDV-2 (Biek, 2007). Thomas Cooley noted, however, the two worst outbreak of EHD in Michigan had EHDV-6 being the predominant serotype. Although EHD is infectious, it is not contagious (Savini et al, 2011). Therefore, hosts cannot pass the disease to each other or other animals through contact. EHD is classified as an Orbivirus (Schmitt, 2012). The disease cannot survive outside either the host animal or the biting fly carrier (EHD Fact Sheet, 2012). Savini et al. note that the spread of EHD is restricted to the specific vectors of the carrier, meaning that it is spread only through specific biting flies. The disease can infect a range of both wild and domestic ruminants including deer, elk, cattle, sheep, and goats. Pigs cannot get the disease, and it is unknown about the susceptibility of dogs (Savini et al, 2011). Humans are not at risk of contracting the disease from either receiving a bite from the vector or eating infected venison (EHD Fact Sheet, 2012). Savini et al. also state that EHD shows high mortality in white-tailed deer, therefore they are the species that is most severely affected by the disease: "The estimated number of affected animals and populations in risk areas suggests an infection rate of 29% and mortality rate of 20% in white-tailed deer" (Savini et al, 2011). Unexplained deer mortality in the late summer or early fall in an area can be a sign of an EHD outbreak (EHD Fact Sheet, 2012).

Most outbreaks occur in the late summer and early fall, and generally stop abruptly when the first frosts begin (Epizootic Hemorrhagic Disease, 2013). The distribution of EHD is limited to the distribution of the *Culicoides* (biting fly/midge) vector (Savini et al, 2011). The extent of an outbreak can be linked to the population of the fly in the area. Although much is known about the disease, there are some aspects about the pathology and epidemiology that are still not well understood. For example, Trainer argues that with the mortality rates being so high in white-tailed deer, the host range being very small, and the inability of the disease to spread through direct contact, it could be reasoned that the disease would be a short lived phenomenon. However, it has been around for many years in multiple locations worldwide, suggesting that there may be more factors and mechanisms responsible for the long term survival of the disease than originally thought (Trainer, 1964).

Symptoms

The symptoms of both EHD and Bluetongue Virus are almost indistinguishable from each other. Both have symptoms that are very characteristic of the disease, and most of them are fairly recognizable. White-tailed deer usually begin to show signs of the disease about seven days after exposure (Epizootic Hemorrhagic Disease, 2013). However, the onset of the disease can be from three to ten days after the infection (Hoff and Hoff, 1976). Initially deer will lose their appetite and their fear of man (Epizootic Hemorrhagic Disease, 2013). Another early sign of the disease is the development of a fever (Hoff and Hoff, 1976). This prompts the deer to migrate toward bodies of water in an attempt to cool off. Many deer are found dead in or around water sources for this reason. Finding dead deer near water sources is one of the most characteristic signs of the disease. The infected animal will also experience weakness and increased salivation, as well as increased pulse and respiration rate. "Bluetongue" gets its name

from the blue appearance of the tongue due to lack of oxygen and hemorrhage (Epizootic Hemorrhagic Disease, 2013). Infected deer also have the tendency to avoid sunlight and experience an increased sensitivity to sunlight (Hoff and Hoff, 1976).

As inferred by its name, one of the most iconic signs of the disease is excessive hemorrhaging. These hemorrhages vary in size and location, with no organs being exempt from possible hemorrhaging. Similarly, a wide range of tissues are susceptible to hemorrhages, with the most common organs affected being the heart, liver, spleen, kidney, lungs, and intestinal tracts (Epizootic Hemorrhagic Disease, 2013). Blood is found in body cavities of dead deer from hemorrhaging (Schmitt, 2012). Infected individuals may also display swollen heads, neck, eyelids, or tongues (EHD Fact Sheet, 2012). Edema and increased pericardial fluid are also commonly found in EHD cases as well (Epizootic Hemorrhagic Disease, 2013).

As the disease progresses, the infected deer may begin to discharge a blood-like substance from its mouth and nose (Schmitt, 2012). Animals with the disease may become lame as well, as lesions take control of the host's legs. These internal and external lesions are often considered an indicator of the disease. The different types of lesions associated with the progression of the disease have led to the classification of three forms of hemorrhagic disease: peracute, acute, and chronic (EHD Fact Sheet, 2012). Savini et al. note that deer with peracute forms of EHD usually die the quickest, often times without showing noticeable symptoms. Deer with chronic forms of EHD are usually ill for several weeks but will generally recover (Savini et al, 2011). The chronic form has lesions forming characteristically on the animal's hooves, and can even "disintegrate" the hooves themselves (EHD Fact Sheet, 2012). It should be noted, however, that lesions are not always present on an individual who has contracted the ailment. For

those cases in which the disease takes an extended toll, ulcers may develop on the hard palate, tongue, and dental pad (Hoff and Hoff, 1976).

Within eight to thirty-six hours of showing symptoms, deer proceed to die (Epizootic Hemorrhagic Disease, 2013). Death comes in the form of severe shock (Trainer, 1964). It should be noted that not every symptom occurs in every case of EHD, and due to the fact that the hosts die so quickly after showing symptoms, it is often difficult to witness most symptoms. The wide range of symptoms involved may also be a reason that cases in the past may have been misclassified as a different disease. The most indicative signs of the disease can often only be witnessed after death. This includes the location of the bodies near water and the hemorrhaging.

Although the mortality of white-tailed deer once infected is high, some deer do survive. These individuals then have a natural resistance to the disease, although they will often carry signs that they had once had the disease. Savini et al. note that deer surviving the disease develop long-lasting neutralizing antibodies that can aid in herd immunity. If an individual does recover from EHD, they are often left lame from implications from legions on the legs and hooves (Savini et al, 2011).

Historic Range

The earliest known cases of EHD in North America were documented in 1955 and 1968 (EHD Fact Sheet, 2012). However, reviewing literature, it can be seen that die-offs of white tailed deer in similar fashions have been occurring in North America as early at 1890 (Trainer, 1964). Historically, EHD has not been distributed uniformly across the Unites States: there have been noted differences between location, frequency, and severity of EHD cases and deaths. In the northern regions of the United States, EHD has occurred infrequently in larger outbreaks,

resulting in a high mortality in those areas. In the south, however, the disease follows a more frequent pattern with less severity: not always resulting in death (Gaydos, 2002). In fact, outbreaks occur almost every year in the southeast, but the number of deaths is usually much smaller (EHD Fact Sheet, 2012).

Schmitt details the history of the disease in Michigan. Prior to 2006, there have only been two reports of EHD in white-tailed deer. In both 1955 and 1974, there were roughly 100 deer involved with the disease in the state. In 2006, the estimated number of deer which died from EHD was between 50 and 75. Since then, there have been reports of EHD in Michigan in 2008, 2009, 2010, and 2011, with the largest die-off recorded being about 1,125 deer in 2010 (Schmitt, 2012). In 2012, there were just under 15,000 deaths reported, which is approximately 13 times higher than 2010 and 50 times higher than the 300 cases involved in 2011. There seems to be an increasing trend in the number of EHD cases in Michigan, especially within the last 10 to 20 years. Part of this trend may be attributed to the fact that the disease is becoming easier to identify, and therefore fewer cases are being misclassified. However, there are likely other factors that have contributed to this increasing trend.

Transmission

EHD is most often transferred through the biting fly (or midge) *Culicoides*. *Culicoides* is a small biting fly which comes from the family *Ceratopogonidae* (Savini et al, 2011). One of the most common carriers for the disease in North America is *Culicoides variipennis,* however there are others that transmit the disease as well (Epizootic Hemorrhagic Disease, 2013). The midge prefers damp areas, though not under water (Rutledge-Connelly, 2005). Therefore, low areas are better suited habit for the fly then highland areas. Lenneman states that these wet areas are

sought after as a place for reproduction. Females lay their eggs in wet, muddy areas where they later hatch and develop into larvae (Lenneman, 2001). The larvae are not specifically aquatic or terrestrial, but occupy the zone between (Kettle, 1962). Eggs hatch in between two and ten days after being laid (Rutledge-Connelly, 2005). The small size of the fly aids in the spread and infection of the disease, as they are difficult to see, track, and keep off of the hosts (Sanders et al., 2011).

The life cycle of the fly can range anywhere from half a year to three years (Lenneman, 2001). However, several generations can be produced in a year (Cranshaw et al., 2012). Adult flies rarely travel far from their breeding area and are therefore usually concentrated within a smaller area, which may be why outbreaks of EHD usually correspond with watersheds (Cranshaw et al., 2012). *Culicoides* are sensitive to desiccation and have low flight speeds (Sanders et al., 2011). Sellers notes that there are certain weather patterns that promote the lifecycle of the fly. Temperatures ranging from 13°C to 35°C are optimal for development of larva and will therefore result in the highest number of adults. In addition, increased humidity is also a good factor for their life cycle (Sellers, 1980).

Wilhelm and Trainer note that generally, epizootic diseases such as EHD can only occur when there is a large host population that is susceptible and the conditions are correct for the fast spread of the disease. They also note that if the population is partially immune, the extent of an outbreak is greatly diminished (Wilhelm and Trainer, 1966). In addition, land use can have an effect on the abundance of *Culicoides* in an area (Sanders et al., 2011). An EHD fact sheet states that due to the fact that the disease is spread through insects, EHD usually becomes prominent in late summer and early fall. Specifically, mid-August through October are the most critical months for the disease, which is when the number of biting flies is usually the highest. In

addition, it is also expected that the disease spreads faster though larger populations of deer as opposed to sparse populations. The first frost of the fall usually kills off a majority of the biting flies, stopping an outbreak (EHD Fact Sheet, 2012). Similarly, the severity of the winter can affect the population of flies the following summer. Severe winters may kill off eggs that are in the soils.

Climatic Factors

Tabachnick states that climate is a significant driver in influencing vector borne disease epidemiology. These disease cycles are very sensitive to changes in climate (Tabachnick, 2009). The spread of insect-borne animal diseases is influenced by several factors, including weather, moisture, and wind (Sellers, 1980). Temperature and rainfall patterns can have a direct effect on vector borne diseases. Aspects such as temperature, precipitation, humidity, and wind can affect different aspects of the vector's life cycle such as survival, behavior and population (Tabachnick, 2009). Drought and heat contribute to quality conditions for the biting flies that carry the disease (QMDA, 2012). The biting fly relies on damp muddy areas for reproduction. Climatic factors control the existence of these suitable habitats. Variables such as climatic or geographic factors can influence the number of carrier insects in an area, which greatly influences the speed and intensity of an outbreak (Savini et al, 2011).

Things are further complicated by the fact that even climatic factors have contrasting effects of vectors. Tabachnick illustrates this point; for general vectors of disease, increasing temperatures can result in higher and lower transmission potentials. Higher temperatures result in decreased vector generation time, increased vector growth rate, increased transmission period, and decreased pathogen incubation period. These all result in increased transmission potential.

Increased water results in increased larval habitats, increased population sizes, and increased host populations over time. These also result in higher potential for transmission. However, higher temperatures can also result in decreased vector longevity and decreased life expectancy, which lower the potential for transmission (Tabachnick, 2009). That being said, it is generally agreed that higher temperatures and higher amounts of water result in a higher potential for transmission.

2012 Outbreak

Reported Cases

The EHD outbreak of 2012 yielded a death toll of suspected or confirmed cases reported by the Michigan Department of Natural Resources of 14,898 deaths. The previously reported worst outbreak which occurred in 2010 resulted in slightly over 1,100 deaths by comparison. The cases in 2012 were concentrated in Lower Peninsula counties in mid-Michigan, particularly in Ionia, Kent, Calhoun, and Barry counties. Almost all counties in the Lower Peninsula had at least one reported case of EHD in the 2012 outbreak, however many of the northern counties had very few. Not only were the number of cases in 2012 dramatically higher than in 2011, but so was the spatial range of the disease. In 2011, the 300 cases were restricted to the southwest counties of Michigan, specifically St. Joseph and Cass counties. The range of counties in the 2012 outbreak was much larger. Figure 1 shows the extent of the concentrated suspected and confirmed cases in 2012.

Not only was the location of cases in 2012 quite diverse, but the timing of occurrences was broad as well. Cases were reported as early as the beginning of July and as late as

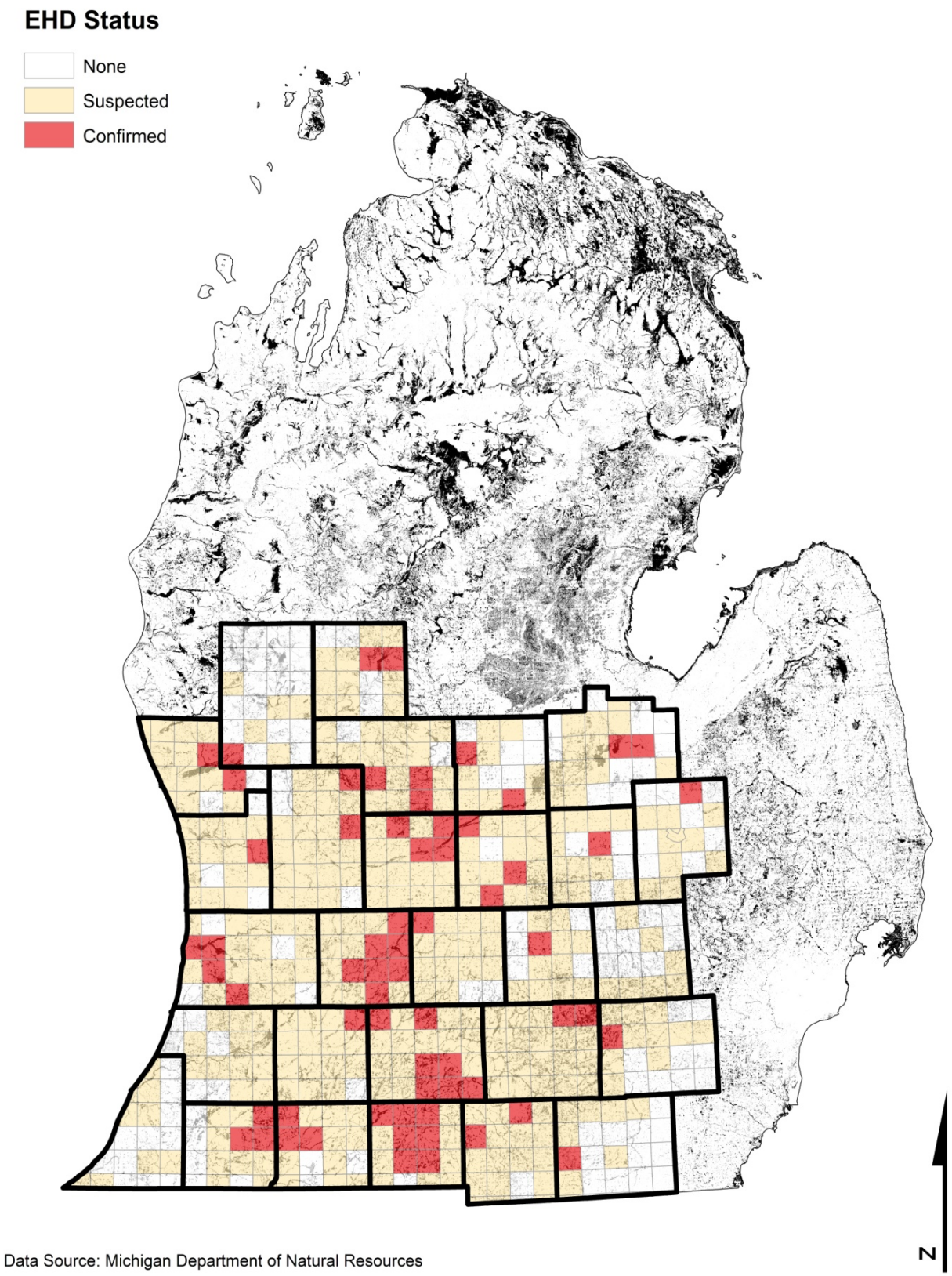

Figure 1: Map of Epizootic Hemorrhagic Disease occurrences in Michigan by township in 2012. EHD status ranges from none (no cases reported), suspected (cases likely due to EHD), and confirmed (cases confirmed to be due to EHD). Only counties that had the most concentrated cases were included. Black polygons within the Michigan boundary represent lands classified as lowland or wetland.

December. By looking at Figure 2, it can be seen that there was a sharp increase in total deaths between early September and mid-October. The Michigan Department of Natural Resources had weekly updated totals of confirmed and suspected cases of deer deaths up until late November They then released the total number for the season in early January of 2013. Although the weekly data is missing for December, Figure 2 shows the sharp increasing trend in September and October. If that trend would have continued, the total deaths as of January 2013 would have been much higher. It can be reasoned, then, that the rate of deaths caused by EHD was highest at the end of September and early October.

However, the surveillance system for reporting and confirming deaths due to EHD was not systematic. The reported deaths may not represent an accurate time of death. There was lag time between when the deer were discovered, when they were decided to be tested, and when a tester was able to check the deer. In addition, there were likely surges in the death toll after press releases about the disease were made public, as it encouraged people to search for dead deer when they were not previously. In addition, when farmers started harvesting crops, it was likely that more deer were found. Therefore, the trends observed from the provided data may not be

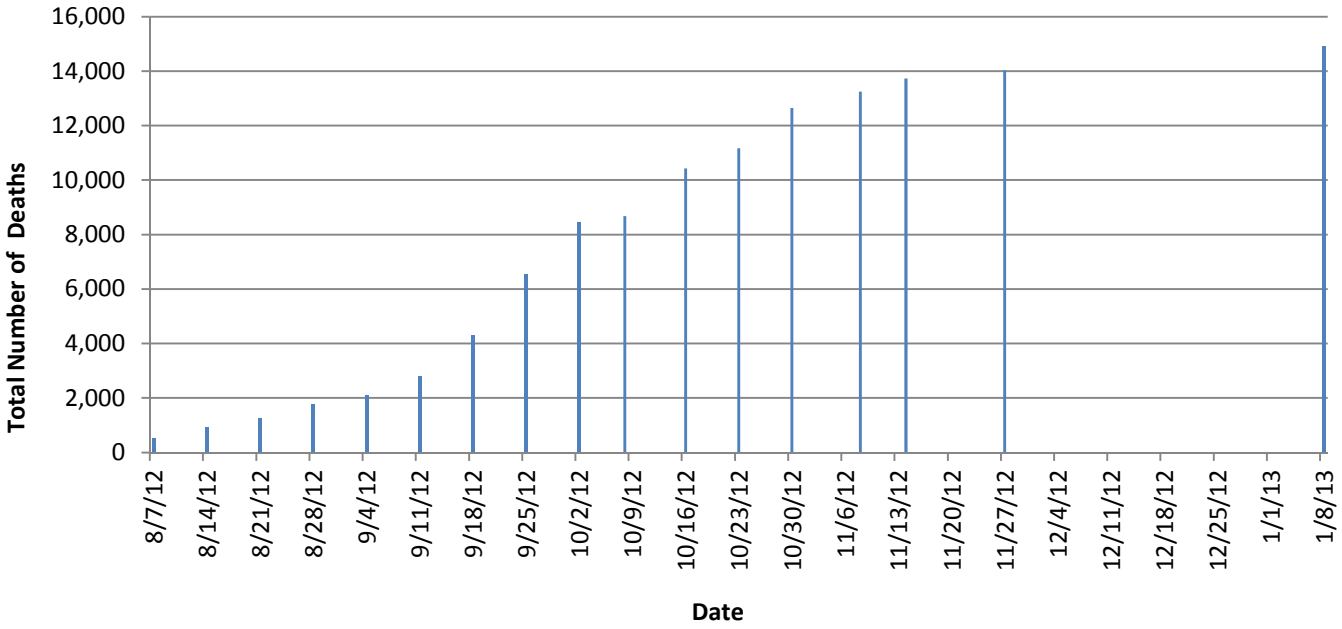

Figure 2: Weekly total number of white-tailed deer deaths confirmed or suspected due to Epizootic Hemorrhagic Disease in Lower Peninsula, Michigan, 2012. The total count as of January 8th, 2013 was 14,898 deaths.

completely indicative of the exact timing of death. However, Figure 2 still displays that by late October and early November, a majority of the cases were already reported. This could be because frosts had, by that point, come through and wiped out the midge flies, ending the outbreak. Another possibility is that with the coming of winter, fewer people were looking for dead deer on their property.

It was also discovered that there was a relationship between land type and the location of confirmed cases of EHD. Michigan townships with confirmed cases of EHD deer deaths in 2012 had statistically higher percentages of lowland, as indicated by non-overlapping 95% confidence intervals. This can be seen in Figure 3. The townships that were considered for this analysis were those townships in counties that had the highest concentration of suspected or confirmed EHD cases. These counties were Allegan, Barry, Berrien, Branch, Calhoun, Cass, Clinton, Eaton, Genessee, Gratiot, Hillsdale, Ingham, Ionia, Jackson, Kalamazoo, Kent, Lenawee, Livingston, Mecosta, Montcalm, Muskegon, Newaygo, Ottawa, Saginaw, St. Joseph, Shiawassee, Van Buren, and Washtenaw. These counties had a combined total of 14,754 deaths out of the total

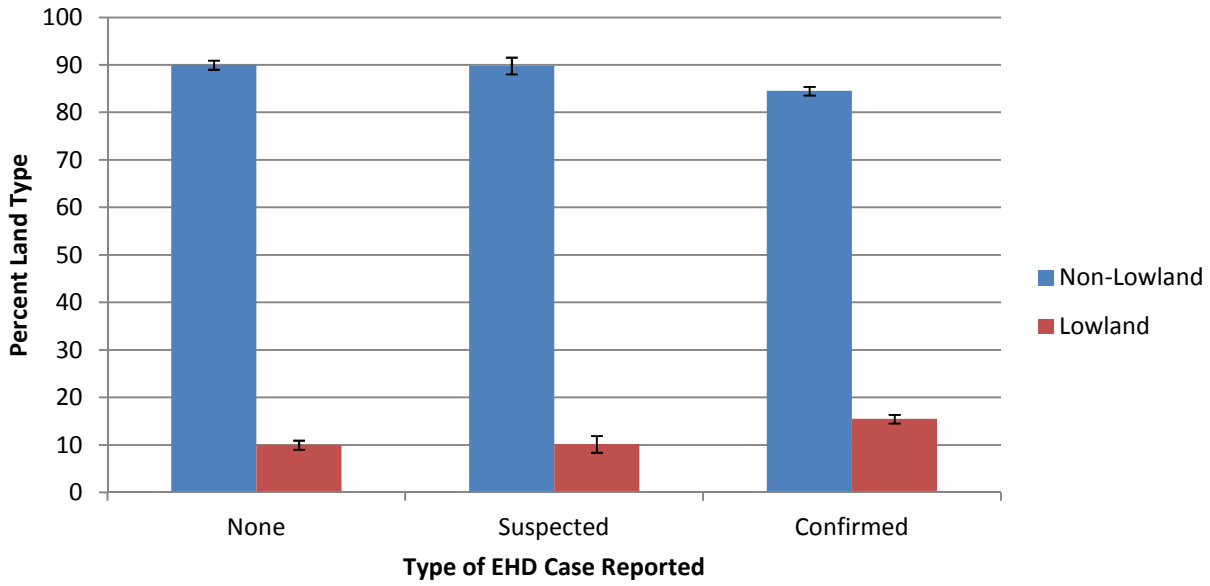

Figure 3: Average percent of lowland and non-lowland land types by townships with zero (none), suspected, and confirmed cases of Epizootic Hemorrhagic Disease caused deaths in white-tailed deer. Average percent of lowland with confirmed cases was shown to be significantly higher than that of suspected or no cases. It was assumed that temperature trends were the same throughout the investigated counties.

14,895 deaths. Therefore, only those counties and their respective townships were considered. In addition, these results were based under the assumption that the temperature trend is the same throughout all of the counties. The direct effect of temperature trends on location of 2012 EHD cases was not included in this study.

Climatic Influence

Analyzing the weather patterns in 2012 and the climatic factors that influence EHD and similar diseases, it can be reasoned why the 2012 outbreak was as severe as it was. The previous winter reported record highs, with very little precipitation of any type. This was followed by early warming in spring, one hard frost, then a record high summer. The lack of precipitation over the course of the 2011 winter and the 2012 summer resulted in many bodies of water drying up, leaving behind muddy patches. A very mild winter failed to kill off the larvae stored in the soil. In addition, there were not many hard frosts that usually help kill off midge larvae. Record setting high temperatures throughout a majority of the summer aided in bodies of water drying up and leaving mud behind. This mud made for excellent breeding habitats for the midges. Similarly, warm and humid weather promotes the biting fly life cycle. The combination of a warm winter, lack of precipitation, and hot summer led to the creation of ideal living conditions for the midge carrier. With prime conditions for the carrier and an over population of deer in the area, the outbreak was set to reach high mortality.

Effects to Deer Population

The high mortality rates caused by EHD outbreaks, including the 2012 epidemic, can have a great impact on the population of deer in a given area (Epizootic Hemorrhagic Disease, 2013). Local populations see the most effects of an outbreak; however, historically there have

been no documented cases of EHD completely decimating a population (EHD Fact Sheet, 2012). Die-offs from EHD cause great alarm in the public however. Regionally, outbreaks have caused even less of an effect on populations, but even local mortality can be noticeable and alarming for management agencies regulating hunting licensing. For the counties in Michigan that were hit the hardest with the 2012 outbreaks, the Michigan Department of Natural Resources decreased the amount of doe tags each individual was allowed to purchase. This was an attempt to stop further depletion of local populations. For DMU 486 (the multi-county private land unit in the southern Lower Peninsula), the number of antlerless licenses a hunter could buy was decreased from 10 licenses to 5 licenses in late November 2012. For public land, it was deemed necessary for the number of antlerless doe permits per hunter to be reduced to 2 permits in particular DMUs in the following counties: Branch, Ionia, Kalamazoo, Kent, Lapeer, Sanilac, Shiawassee, Tuscola, and Van Burn (Parker, 2012).

Reducing permits and licenses can have consequences on other factors as well: less money taken in from hunting licenses means less money going toward wildlife management. In addition, displaced hunters may begin to hunt in other counties, which would impact the expected harvest and population of deer in those areas. However, neither of these consequences would likely result in drastic changes in funding or deer populations. There is little doubt that the local populations will be able to bounce back from the unexpected die-offs they experienced in 2012 (Epizootic Hemorrhagic Disease, 2013). However, future EHD outbreaks can continue to damage local populations with tangible consequences. If future outbreaks continue to get worse, local and region populations may suffer more long term effects.

Control Methods and Predictions

Control methods

Once the viral form of EHD becomes established in an area, it is very difficult to control the spread of the disease (Savini et al, 2011). Trying to control the disease through vaccination is a possible solution, however it is rare and not practically feasible. The use of pre-emptive vaccinations could be possible for white-tailed deer in captivity, however is not practical for wild populations (Hoff and Hoff, 1976). Another way to try to control the disease is to monitor and control the vector of the biting flies. However, the success and actual implementation of such plans have not been studied (Savini et al, 2011). One factor that can aid in controlling the number of the biting flies is manipulating the specific requirements for the larvae. The larvae need wet soils to survive. If this factor could be controlled or altered, then perhaps the population of biting flies could also be controlled. However, this would be a difficult task over large areas. If this option were to be attempted, the necessary steps would include occasionally flooding and/or completely drying of ponds that are breeding sites. In addition, eliminating stagnant water can help reduce the fly's habitat (Cranshaw et al., 2012).

Usually, the most that can be done to avoid a severe outbreak is to minimize the risk. The risk of an outbreak is lower when deer herds do not exceed the natural carrying capacity of the habitat. Therefore, the best and most practicable way to limit the impacts of the disease is to manage a deer population is by properly managed sport hunting, increasing and decreasing the number of permits when as necessary (EHD Fact Sheet, 2012).

Future Outbreak Predictions

When control methods are not viable options, it must be considered what warning signs there are before an outbreak occurs. Despite the correlation between climate and vectors,

attempting to predict the spread of vector borne diseases by climate is difficult. The cycle between the vector and climate is a complex one (Tabachnick, 2009). In addition, trying to predict future climatic factors is itself very difficult. However, weather patterns can affect the midge fly population and should be considered. Mild winters can fail to kill insect populations stored in the soil. Warm dry summers can help create ideal habitats for the biting midges. In addition, if the population of white-tailed deer in Michigan continues to be as large as it has been recently, there is an increased chance of severe outbreaks in the near future. Monitoring the deer population gives information about the number of deer in certain areas, which can be used to predict highly susceptible areas.

In this study, there was evidence to suggest that there was a relationship between lowland areas and confirmed cases of EHD. Therefore, it can be reasoned that townships with more lowland areas are more likely to see a larger number of deaths during future outbreaks. This can be seen in Figure 4. When comparing Figure 1 (which shows the townships by EHD case status) with Figure 4, it can be seen that the townships with confirmed and suspected cases have similar patterns with the townships with higher percent lowland. In addition, by comparing the location of the 2011 and 2012 cases, it can be seen the disease had expanded in the east north east direction. By analyzing Figure 4, it can be seen that the counties of Livingston, Jackson, and Ingham have high percentages of lowland. In the 2012 outbreak, these counties did not see as large of confirmed or suspected cases as other counties. However, due to the high percentage of lowland in those townships, they should be watched closely during future outbreaks. In addition to these particular counties, Figure 4 shows many other townships which have high percentages of lowland areas, making them susceptible to high numbers of EHD cases and deaths during future outbreaks.

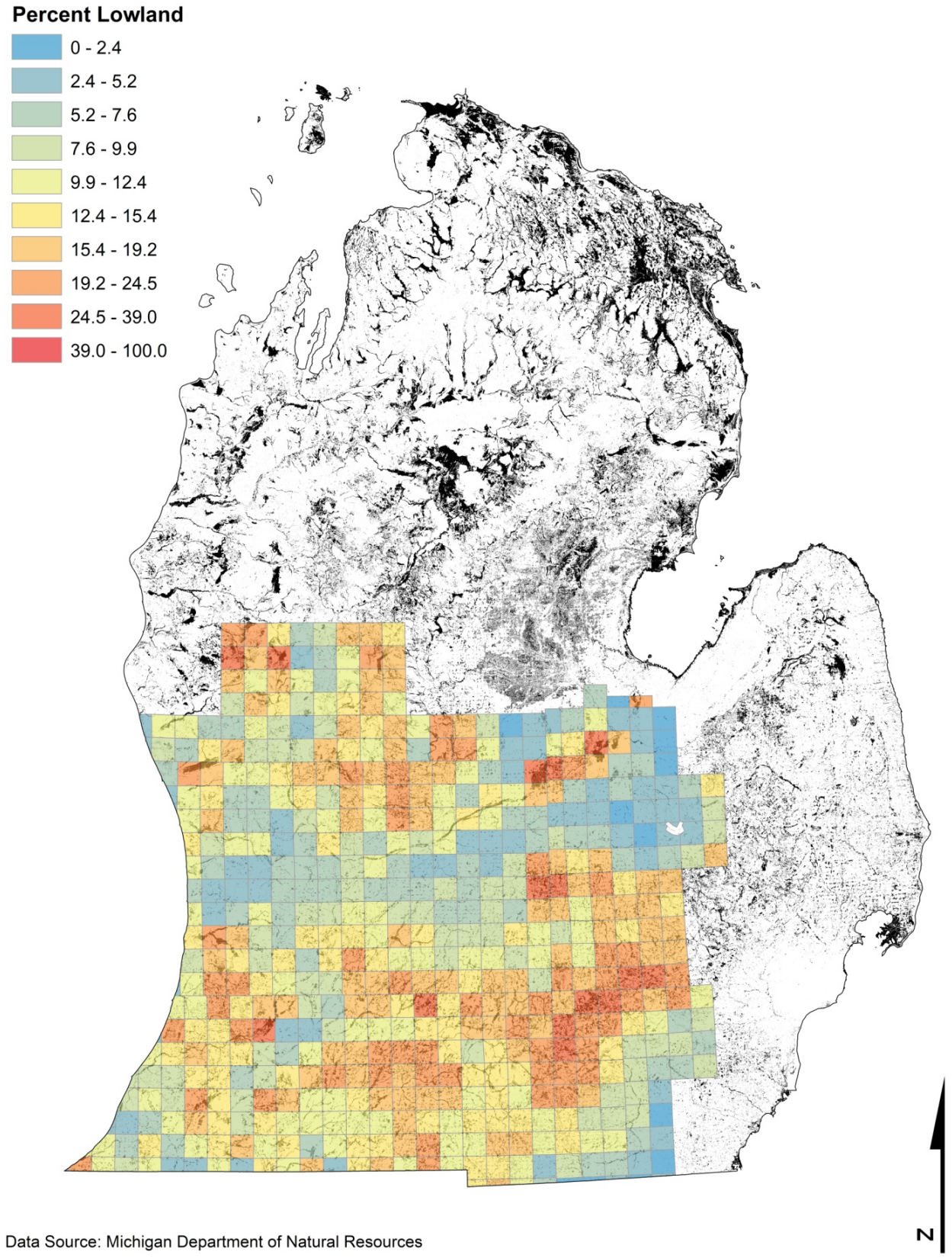

Figure 4: Percent of lowland areas by townships in Michigan. Townships with higher percentages of lowland are likely to have higher deer deaths due to an Epizootic Hemorrhagic Disease outbreak in the future. Only townships located in counties with concentrated cases were considered. Black polygons within the Michigan boundary represent lands classified as lowland or wetland. classifications in Michigan.

Predicting the time and location of any disease is difficult, as there are many factors that play a role in the transmission and severity of diseases, including insect-borne diseases. For EHD outbreaks in Michigan, there are certain factors that can indicate the likelihood, location, and severity of an outbreak. This study concludes that three of the most indicative factors of timing, location, and severity of EHD outbreaks in Michigan are deer population size, weather and climate patterns, and land type.

Conclusion

The summer and fall of 2012 saw the worst documented outbreak of EHD in white-tailed deer in Michigan's history. The death toll was 50 times greater than the previous year, and the trend of deaths from EHD over the last 10 to 20 years has been increasing. While regional populations were not greatly disrupted, some local populations suffered severely, leading to implications for hunters. The weather patterns of 2011 and 2012 most likely led to the increased death toll, as they created ideal conditions for the disease's vector, the *Culiodes* biting fly. Similarly, evidence suggests that areas with higher percentages of lowland were related to more confirmed deaths. In addition, the high mortality rate of the disease aided in the death toll. Monitoring the weather and deer populations as well as noting lowland areas is not a guaranteed way to predict future outbreaks, however that is a start. Controlling the disease is extremely difficult and not practical, so minimizing the risk is a more realistic option of control. If future outbreaks continue to occur and occur at higher severities in Michigan, it could result in long term detrimental consequences to deer population as well as hunting policies. More research is needed to fully understand the consequences of the disease and to develop control and prediction methods.

Bibliography

Biek, Roman. 2007. Evolutionary dynamics and spatial genetic structure of epizootic hemorrhagic disease virus in the eastern United States. *Infection, Genetics and Evolution*. 7(5): 651-655. http://www.sciencedirect.com.ezproxy.gvsu.edu/science/article/pii/S1567134807000640.

Cranshaw, W.S., F.B. Peairs, and B. Kondratieff. 2012. Biting flies. Colorado State University: Extension. http://www.ext.colostate.edu/pubs/insect/05582.html.

Epizootic Hemorrhagic Disease (EHD) in white-tailed deer. 2013. Michigan Department of Natural Resources.

http://www.michigan.gov/dnr/0,1607,7-153-10370_12150-26647-,00.html.

Epizootic Hemorrhagic Disease: fact sheet. 2012. Growing Deer.tv.

http://www.growingdeer.tv/view/wp-content/uploads/2012/07/Epizootic-Hemorrhagic-Disease-Fact-Sheet.pdf.

Gaydos, Joseph K., William R. Davidson, Francois Elvinger, Daniel G. Mead, Elizabeth W. Howerth, and David E. Stallnecht. 2002. Innate resistance to epizootic hemorrhagic disease in white-tailed deer. *Journal of Wildlife Diseases*. 38(4): 713-719. http://www.jwildlifedis.org/content/38/4/713.full.pdf+html.

Hoff, Gerald L. and Diane M. Hoff. 1976. Bluetongue and Epizootic Hemorrhagic Disease: A review of these diseases in non-domestic artiodactyls. *The Journal of Zoo Animal Medicine.* 7(2): 26-30. htty://www.jstor.org/stable/20094362.

Kettle, D. S. 1962. The bionomics and control of Culicoides and Leptoconops (Diptera, Ceratopogonidae = Heleidae. *Annual Review of Entomology*. 7: 401-418. http://www.annualreviews.org/doi/pdf/10.1146/annurev.en.07.010162.002153.

Lenneman, Nathan. 2001. Culicoides varipennis. Animal Diversity Web. http://animaldiversity.ummz.umich.edu/accounts/Culicoides_variipennis/.

Parker, Bill. 23 Nov. 2012. DNR restricts doe permits in EHD areas. Michigan Outdoor News, 1+. Print.

QDMA says watch for blue tongue. 2012. *Heartland Outdoors*. http://www.heartlandoutdoors.com/heartland/story/qdma_says_watch_for_blue_tongue/.

Rutledge-Connelly, Roxanne C. 2005. Featured creatures: entomology and nematology.

University of Florida. http://entnemdept.ufl.edu/creatures/aquatic/biting_midges.htm.

Sanders, Christopher J., Chris R. Shortall, Simon Gubbins, Laura Burgin, John Gloster, Richard Harrington, Don R. Reynolds, Phillip S. Mellor, and Simon Carpenter. 2011. Influence of season and meteorological parameters on flight activity of Culicoides biting midges. *Journal of Applied Ecology*. 48: 1355-1364. http://onlinelibrary.wiley.com.ezproxy.gvsu.edu/doi/10.1111/j.1365-2664.2011.02051.x/abstract.

Savini, G., A. Afonso, P. Mellor, I. Aradaib, H. Yadin, M. Sanaa, W. Wilson, F. Monaco, and M. Domingo. 2011. Epizootic heamorragic disease. *Research in Veterinary Science*. 91(1): 1-17. http://www.sciencedirect.com.ezproxy.gvsu.edu/science/article/pii/S0034528811001780.

Schmitt, Stephen M. 2012. Summary of selected Michigan wildlife health issues. Michigan Department of Natural Resources. http://www.michigan.gov/documents/emergingdiseases/2012-WildlifeHealthIssues-Michigan-Booklet_382349_7.pdf.

Sellers, R. F. 1980. Weather, host, and vector: Their interplay in the spread of insect-borne animal virus diseases. *The Journal of Hygiene*. 85(1): 65-102. http://www.jstor.org/stable/3862445.

Tabachnick, W. J. 2009. Challenges in predicting climate and environmental effects on vector borne disease episystems in a changing world. *The Journal of Experimental Biology*. 213: 946-954. http://jeb.biologists.org.ezproxy.gvsu.edu/content/213/6/946.full.pdf+html.

Trainer, Daniel O. 1964. Epizootic Hemorrhagic Disease of Deer. *The Journal of Wildlife Management*. 28(2): 377-381. http://www.jstor.org/stable/3798102.

Wilhelm, A. R. and D. O. Trainer. 1966. A serological study of epizootic hemorrhagic disease of deer. *The Journal of Wildlife Management*. 30(4): 777-780. http://www.jstor.org/stable/3798284.