RETROSPECTIVE REVIEW OF WILD WATERFOWL DISEASES IN KANSAS

by

THOMAS ALLEN BECKER

B.S., Kansas State University, 2015

A THESIS

submitted in partial fulfillment of the requirements for the degree

MASTER OF SCIENCE

Department of Horticulture and Natural Resources
College of Agriculture

KANSAS STATE UNIVERSITY
Manhattan, Kansas

2016

Approved by:

Major Professor
Dr. David A. Haukos

Copyright

Abstract

There is a wide variety of diseases that affect wild migratory birds. Occurrence, causes, and impacts of disease outbreaks in wild bird populations are rarely studied beyond documentation of large epizootic events. The relationships between the wildlife-livestock-human interface is rapidly blurring together. Global interests in avian diseases increased around 1990 as a result of the prevalence of zoonosis and potential threat to domestic livestock. A central disease reporting protocol does not exist in many states, which has led to a lack of available historical knowledge of disease occurrence that could be used to predict and manage future outbreaks. Due to changes of abundance and distribution of the migrant populations of Ross's goose (*Chen rossii*) and Snow goose (*C. caerulescens*), geese are increasing their stopover duration in Kansas potentially increasing risk of disease outbreaks. We compiled historic records of wild waterfowl disease events in Kansas from 1967-2014 and related the frequency of events with indices of light geese abundance from 1970-2014. We found 32 reports spanning 16 counties consisting of the diseases avian cholera, avian botulism, aspergillosis, renal coccidiosis, west Nile, aflatoxicosis, and mycotoxicosis. Using a retrospective survey, we found there was a significant relationship between population densities of light geese in Kansas during the Mid-Winter Waterfowl Inventory and occurrence of avian cholera. Efforts to increase the understanding of relationships between disease outbreaks and host species will improve management of future disease outbreaks. Understanding factors known to facilitate wild waterfowl disease events (e.g., environmental, species, and individual), may assist in disease identification and determine a disease management course of action. This course of action is predetermined in a disease management plan. Disease management plans should be developed at the state and station level; incorporating planning, response, disease control, and surveillance and monitoring schemes to build upon the centralized disease database and to promote future disease understanding.

Table of Contents

List of Figures

List of Tablesk

Acknowledgments

I am blessed to have all these people to thank during thesis writing process. Peg Shaw-McBee for your guidance into graduate school. To my advisor, David Haukos, thank you for your guidance in my research, the much needed editing and refocusing. Thank you, Adam Ahlers, for supportive advice and statistical knowledge throughout this writing. I greatly appreciate, Ted Cable, for serving on my committee, and Tim Todd for your statistical assistance. Thank you Shane Hesting for the information and ideas for this project. I would also like to thank all the Kansas Department of Wildlife, Parks, and Tourism personnel who assisted me in the records collection process, and all the graduate students in the Haukos lab. Thank you to my wife, Valerie, for supporting me through this adventure, and to my son, James, who always greeted me joyfully when I arrived at home in the evenings. This research was funded by Kansas Department of Wildlife, Parks, and Tourism through funding provided by U.S. Fish and Wildlife Service Avian Health and Disease Program, I appreciate the assistance from T. Roffe and L. Jones in facilitation of the funding.

Chapter 1 - A Review of Avian Disease

Introduction

Avian diseases are not usually a high priority for wildlife managers during development of wildlife management plans; however, diseases do have the potential to affect the health and well-being of wildlife, livestock, and humans on local to global scales. Of particular concern are aggressive epizootic events that reach isolated wildlife populations, are present at critical migratory stopover habitats (Blanchong et al. 2006), or interrupt the world food supply by infecting millions of livestock resulting in euthanasia of those affected to prevent further disease spread (USDA 2015). Even more pressing are zoonotic events that cause the illness and death of humans. By studying, recording, and analyzing avian disease events, a holistic understanding of where, when, how, and why these events occur can be developed.

Avian diseases threaten more than just the health of wildlife and humans, they also impact the food supply and economy, affecting the lives of billions of people worldwide (USDA 2015). For example, 50,400,000 birds were affected by the Highly Pathogenic Avian Influenza (HPAI) outbreak in 2015, which decimated 211 commercial flocks within the United States (USDA 2016). This outbreak not only decimated millions of poultry but potentially affected exports of poultry products and an increase in food prices for U.S. consumers (USDA 2015).

History

Disease, by definition, is the impairment to health of an organism (including functional health; Friend et al. 2001), not just illness or death (Wobeser 1997). The primary focus of my review will be on diseases that result in wildlife mortality. Disease awareness has been around since the early 1500s, arising during the germ theory era (Friend et al. 2006). During the late 1800s, people began advancing their understanding of diseases when germ theory was validated.

In 1933, the first wildlife disease laboratory was established at Michigan State University in East Lansing, and "Control of Disease" was a chapter in Aldo Leopold's "Game Management" (Leopold 1933). The Wildlife Disease Association was formed in 1951. In 1975, the U.S. Fish and Wildlife Service (USFWS) established the National Wildlife Health Center, Madison, Wisconsin, with transfer to the U.S. Geological Survey in the mid-1990s. However, it was not until the 1990s that the world community came together to try and understand the wildlife disease quandary (Boadella et al. 2011).

In 1991, the struggle to monitor the health and management of free-ranging mammals started with a symposium in Nancy, France, which was followed by the first conference of the European section of the Wildlife Disease Association in 1994. Until 1994, wildlife diseases were not of great concern to the global community, but due to the increasing concern of infectious and zoonotic diseases spreading throughout Europe, Africa, and Asia, the call for such awareness could no longer go unheard. These calls resulted when diseases (e.g., avian influenza, swine fever) were witnessed transitioning borders, typically spreading throughout several countries and continents, altering the human-livestock-wildlife interface (Boadella et al. 2011).

Anthropogenic change (e.g., landscape fragmentation, supplemental feeding of wildlife) is altering the human-livestock-wildlife interface and wildlife reservoirs are becoming more common (Boadella et al. 2011, Altizer et al. 2011). These disease reservoirs are a global concern because they do not recognize political jurisdictions as some species migration span continental boundaries. For example, migratory birds can carry diseases vast distances along migratory routes while appearing healthy (Friend and Franson 1999, Simpson 2002, Altizer et al. 2011). Some migratory species that serve as reservoirs (e.g., snow goose [*Chen caerulescens*]) are

changing their migratory patterns due to anthropogenic landscape and global climate change (Patz et al. 2008, Altizer et al. 2011, Van Hemert et al. 2014).

Increasing global temperatures (Van Hemert et al. 2014) are posing new problems relative to disease spread and transmission among migratory bird breeding colonies (Friend et al. 2001) (e.g., snow goose and northern pintail [*Anas acuta]*). These problems include interactions among previously isolated populations and species as distributions are expanding throughout their summer ranges (Friend et al. 2001, Harvell et al. 2009). Increasing seasonal temperatures are also shifting the wintering range for some species, such as the snow and Ross's geese (*C. rossii*). Historically, the wintering range for snow and Ross's geese in the Central Flyway was associated with the Gulf Coast of Mexico and Texas where they used costal marshes surrounded by agricultural fields, as well as the playa region of the Southern High Plains (Robertson and Slack 1995). Currently, the Central Flyway Mid-Wintering Survey (2015) indicated that the largest proportion of the snow geese population in the flyway is wintering farther north in Kansas (Figure 1.1)

Perspective

Global

Understanding wildlife diseases from a global prospective gives an awareness to the extent of the potential effects. A global perspective of such effects may cause great concern when dealing with the potential spread of disease via migratory birds. Many waterfowl migrate great distances covering several continents potentially contributing to the spread of disease (Fischer et al. 2015). The potential reach of these diseases warrants surveillance efforts to detect latent threats to wildlife, livestock, and humans. Such worldwide surveillance efforts already exist for much of the agriculture industry and through human health organizations (e.g., World

Health Organization, World Animal Health). Monitoring of wildlife diseases was once limited to outbreak response and record keeping by wildlife management units, county, state, or country entities; however, now global interests are incorporating large-scale surveillance schemes; allowing those involved to quickly detect and attempt to counter infectious and zoonotic disease in wildlife populations (Boadella et al. 2011).

Persistent surveillance and global monitoring of wildlife die-offs could potentially aid in forecasting disease outbreaks and track trends. As the global community's understanding of wildlife diseases improves though surveillance, there are some complexities to overcome (Henke et al. 2007). For instance, monitoring and accurately identifying disease at the field level is especially difficult, as similar signs and symptoms are common among, requiring field necropsies as a form of generalized diagnostics (Friend and Franson 1999, Soos and Wobeser 2006). Thus, positive identification can only be determined by wildlife disease diagnostic laboratories; however, few wildlife disease labs exist throughout the world.

Regional

In North America, there are four delineated administrative flyways for management of migratory waterfowl: Atlantic, Mississippi, Central, and Pacific. All of these flyways have breeding populations that mix within the northern latitudes (Friend and Franson 1999, Fischer et al. 2015). Movements of infected individuals across flyways likely leads to disease transmission (Hill et al. 2016). For example, it is believed that the rapid spread of West Nile virus (1999-2002) was due to migratory routes of songbirds, as well as host and vector relationships (Campbell et al. 2002, Blanchong et al. 2016).

There are agencies dedicated to monitoring the spread of wildlife diseases throughout North America, including the U.S. Geological Survey's National Wildlife Health Center, and the

National Animal Health Surveillance System (NAHSS) developed by the U.S. Department of Agriculture. The NAHSS monitors for diseases that may affect agriculture stakeholders. These agencies efforts are helpful, but they do not capture every disease event, which is why there should be continued surveillance scheme at a local level.

Local

Depictions of the regional distribution of the Central Flyway show the abundance of migrating waterfowl bottlenecking through the Rainwater Basin in south-central Nebraska, then longitudinally expands through Kansas, Oklahoma and Texas. Many migratory waterfowl are found throughout Kansas; however, there are a few main wetland areas where migratory waterfowl concentrate. These areas include Cheyenne Bottoms Wildlife Area in Barton County and Quivira National Wildlife Refuge in Stafford County. The concern with the abundance of waterfowl at Cheyenne Bottoms and Quivira National Wildlife Refuge is duration of high concentrations, because higher densities of migrating waterfowl lead to the increased risk of disease outbreaks (Smith et al. 1990, Friend 2001, Altizer et al. 2011). The concentration of waterfowl in a single wildlife area location during migration can range from a few thousand to hundreds of thousands of migratory birds.

It is believed that as the waterfowl abundance increases, the probability of disease transmission increases. This is due to the gregarious nature during spring and fall migration for some species such as snow geese and northern pintail (Robertson and Slack 1995, Altizer et al. 2011). As disease establishes in a local waterfowl population, the continual spread will endure until the transmission chain is broken (Henke et al. 2007, Altizer et al. 2011). Managing a spreading disease is difficult due to the large gap of knowledge in the complexity of disease, host-vector interactions, and environmental relationships.

Diseases

Wildlife diseases can be categorized into five major groups: bacterial, viral, fungal, parasitic, and toxin (biologic and chemical) (Arnall and Keymer 1975, Wobeser 1997, Friend and Franson 1999). Within each of these categorizes are a multitude of diseases, but there are six avian diseases of primary concern (Table 1.1). Although most efforts focus on diseases that end in the mortality of the animals involved; some diseases pose transmission risk to other wildlife species, as well as to livestock or even humans. Wildlife diseases persist in many fashions and present infection through four basic routes and exposures (Table 1.2).

There is still much uncertainty of the complex relationships between diseases and transmission. For example, it is thought that a bird-maggot cycle is a major cause of avian botulism (*Clostridium botulinum*) transmission (Wobeser 1997, Friend and Franson 1999); however, many other factors may also contribute to these epizootic events (Locke and Friend 1989, Wobeser 1997). The understanding of general concepts for routes and exposures assist in the recovery and handling of diseased animals when conducting disease management, mitigation, and clean up (Altizer et al 2011). This is especially important when managing diseases that cause high mortality risk among migratory birds as well as possible transmission to agricultural livestock.

Because of the possibility of mass mortality among migratory birds at stopover sites, there are a few avian diseases that warrant increased concern. Historically, avian botulism was the primary avian disease causing mortality in large numbers; however, in 1975 the Nebraska Rainwater Basin had a report of avian cholera (*Pasteurella multocida*) and subsequently, outbreaks of this disease became an annual occurrence until the 2000s (Smith et al. 1990, Blanchong et al 2006, Samuel et al. 2007, Webb et al. 2010). Both avian botulism and avian

cholera are reported frequently across the United States in the all four flyways and all seasons (Friend and Franson 1999). There are other diseases that are of major concern due to their epizootic nature (e.g., duck plague, avian pox, Newcastle disease, West Nile, and trichomoniasis); however, they do not always result in mortality but rather an impairment to the individual's health.

Bacterial

Bacterial diseases are the most common cause of mortality in wild birds (Friend and Franson 1999, Soos and Wobeser 2006). Bacteria in the genus *Clostridium* are responsible for greater mortality in wild birds than any other disease agent (Friend and Franson 1999); however, avian cholera has become one of the most notable bacterial disease in recent years (Samuel et al. 2007). A few other notable bacterial diseases are Tuberculosis (*Mycobacterium avium*), Salmonellosis (*Salmonella* spp.), and Chlamydiosis (*Chlamydia psittaci*). These diseases are not of primary concern because of their infrequency in wild birds, although concerning due to their nature to affect livestock and other wild mammals.

Viral

New viruses are frequently being discovered and continually evolving (Arnall and Keymer 1975, Friend and Franson 1999). Viral diseases, such as duck plague, Newcastle, and avian influenza, are not recognized as major contributors to avian (wild) disease in North America (Friend and Franson 1999). Although viral outbreaks were recorded in the early 20th century, the mechanistic understanding of their occurrence and transmission was not clear until after World War II (Friend and Franson 1999).

Major viral infections include avian influenza, which has caused panic at a global scale due to the potential zoonotic threat. As mentioned, this endemic has caused nations to halt trade

between affected countries and major efforts have been undertaken to eliminate the possibility of further spread. Past viral infections were recorded in detail due to the large numbers in mortality; for example, duck plague first appeared in the United States in 1973 and resulted in the mortality of roughly 40,000 mallards (*Anas platyrhynchos*) and numerous deaths of other waterfowl species in South Dakota (Friend and Franson 1999). Unfortunately, some viral outbreaks are difficult to determine due to their manifestation and symptom similarities (Wobeser 1997); however, regardless of the similarities, many virus outbreaks in the poultry industry result is in the elimination of all birds and sterilization of facilities where infection is found. Many serious viral diseases (e.g., Newcastle disease, avian influenza, and duck plague) require immediate attention and involvement from disease control specialists (Friend and Franson 1999). Other viral diseases call for intervention, but the severity of a mitigation response should be lessened to prevent spread. Most of the lesser viral diseases result in gross lesions and prevention should be the focus.

Fungal

Fungi are unique because they are commonly found within the normal environment (Arnall and Keymer 1975, Friend and Franson 1999). Individuals that are infected by fungi are usually immunosuppressed and concurrently suppressed by other stressors for prolong periods of time (Arnall and Keymer 1975, Friend and Franson 1999). Fungal infections are opportunistic and present themselves uniquely by different manifestations; these are direct invasion of tissues by fungal cells, allergic reactions as a result of hypersensitivity by the host, and mycotoxicosis (results from ingestion of toxic fungal metabolites [Wobeser 1997, Friend and Franson 1999, Altizer et al. 2011]). The primary fungal infection affecting migratory wild birds is aspergillosis (*Aspergillus fumigatus* [most common]). Another fungal disease is candidiasis, but due to its

infrequent reporting, this disease does not need management at this time (Friend and Franson 1999).

Parasitic

The parasitic category is a very diverse group of organisms ranging from ticks, fleas, and mites, to members of nematodes, cestodes, trematodes, acanthocephalans, and protozoans (Friend and Franson 1999). These types of parasites have a close relationship between themselves and avian species (host) as to which they parasitize nourishment. Life cycles among these groups are vastly different from one another; where some parasite life cycles are directly related with the host species, and other parasite life cycles involve one or more (complex) intermediate hosts (Friend and Franson 1999). In some cases, parasites require host mortality to continue their life cycle (Arnall and Keymer 1975); however, most parasites identified in avian species prevail with no clinical disease (Friend and Franson 1999).

Toxins

There are two main subcategories of toxins: biological and chemical. Biological is the main focus of this paper because it pertains to the development of toxins produced by cells of other living organisms. This could include venomous groups such as spiders and reptiles. For the purpose of this paper I focused on the biological toxins produced by *Clostridium botulinum* bacteria (here after, botulism) and mycotoxins produced by fungi. Botulism has been known to cause death of hundreds of thousands of avifauna during a single event (Wobeser 1997, Friend and Franson 1999). These deaths are the result of toxins produced within the replicating bacteria (Soos and Wobeser 2006). There are seven known toxin types from botulism (a – g), all which affect different species differently. Botulism outbreaks are commonly associated with many

degraded environmental conditions, such as saline waters and low oxygen content (Wobeser 1997, Friend and Franson 1999).

Fungal toxins are associated with moldy organic matter, which are typically not found palatable by wildlife (Beer 1988, Wobeser 1997, Friend and Franson 1999). During stressed environmental conditions, wildlife may consequentially feed from food sources that contain the mold and succumb to the toxicity (Beer 1988, Wobeser 1997, Friend and Franson 1999). Identification of the toxic source is needed to prevent further infections. With most toxins, eliminating the point source of the outbreak is key to prevent further infection; this is also true when investigating chemical toxins as well.

Many chemical toxins become environmental contaminates due to their application of use (Friend and Franson 1999). The task of diagnosing exact toxic cause of morality is difficult due to an array of potential wildlife that may be exposed. This may be a result of pesticides, metals, petroleum, and other synthetic compounds (Wobeser 1997, Friend and Franson 1999).

Discussion

Despite efforts by many wildlife disease organizations, the reporting of avian disease events is usually not a high priority for many state wildlife managers (Henke et al. 2007, Boadella et al. 2011); however, when diseases do occur it can be at a great cost (Friend and Franson 1999, Heneke et al. 2007). There have been millions of dollars spent on the clean-up and prevention of further contamination from disease events (e.g., avian influenza [USDA 2016]). Costs are not only associated with contamination prevention and elimination, but with economic damages worldwide (USDA 2016).

Economic damages come from the loss trade from affected countries; not only inflicting financial burdens on the industry, but trickling down costs to all consumers. This creates a

reaction of fear when disease events occur and potential effects are unknown. It is believed that the world will be seeing an increase in large scale disease events occurring due to the current state of climate change (Patz et al. 2008, Altizer et al. 2011, Van Hemert et al. 2014).

With the shifts in global climate, the perspective of diseases has to be beyond the local and regional scale; however, by viewing events on the global scale, many disease monitoring agencies have lost sight of the local scale events that can be precursors to larger (regional scale) events. I suggest a disease surveillance and monitoring program should be established at the state level to ensure that disease events do not go unreported. Much is still unknown about diseases, and the complexity that each disease presents needs to be further understood. Through the advancement of knowledge, the understanding and mitigation of avian diseases could increase.

Literature Cited

Altizer, S., R. Bartel, and B. A. Han. 2011. Animal migration and infectious disease risk. science 331:296-302.

Arnall, L., and I. F. Keymer. 1975. Bird diseases. An introduction to clinical diagnosis and treatment of diseases in birds other than poultry. Bailliere Tindall, London, England.

Beer, J. V. 1988. Diseases of gamebirds and wildfowl. The Game Conservancy, Fondingbridge, Hampshire, England.

Blanchong, J., M. Samuel, and G. Mack. 2006. Multi-species patterns of avian cholera mortality in Nebraska's rainwater basin. Journal of Wildlife Diseases 42:81-91.

Blanchong, J. A., S. J. Robinson, M. D. Samuel, and J. T. Foster. 2016. Application of genetics and genomics to wildlife epidemiology. Journal of Wildlife Management 80:593–608.

Boadella, M., C. Gortazar, P. Acevedo, T. Carta, M. P. Martín-Hernando, J. Fuente, and J Vicente. 2011. Six recommendations for improving monitoring of diseases shared with wildlife: examples regarding mycobacterial infections in Spain. European Journal of Wildlife Research 57:697-706.

Campbell, G. L., A. A. Marfin, R. S. Lanciotti, and D. J. Gubler. 2002. West Nile virus. The Lancet Infectious Diseases 2:519-529.

Fischer, J., F. Broerman, S. Kovach, B. Scotton, M. Bertram, K. Sowl, R. MacDonald, T. Moran, M. Petrula, and R. Suydam. 2015. Northern Pintail, Anas acuta. <http://alaska.usgs.gov/science/biology/avian_influenza/species/species.php?code=nopi>. Accessed 27 Jan 2016.

Friend, M., and J. C. Franson. 1999. Field manual of wildlife diseases. General field procedures and diseases of birds. Biological Resources Division Information and Technology Report 1999-01, US Department of the Interior and US Geological Survey, Washington, D.C., USA.

Friend, M., R. G. McLean, and F. J. Dein. 2001. Disease emergence in birds: challenges for the twenty-first century. Auk 118:290-303.

Friend, M. 2006. Disease emergence and resurgence: the wildlife-human connection. Circular 1285. US Geological Survey, Reston, V.A., USA.

Harvell, D., S. Altizer, I. M. Cattadori, L. Harrington, and E. Weil. 2009. Climate change and wildlife diseases: when does the host matter the most? Ecology 90:912-920.

Henke, S. E., A. M. Fedynich, and T. A. Campbell. 2007. Wildlife disease management: an insurmountable challenge? U.S. Department of Agriculture National Wildlife Research Center Staff Publication 684. Fort Collins, CO, USA

Hill, N. J., E. J. Ma, B. W. Meixell, M. S. Lindberg, W. M. Boyce, and J. A. Runstadler. 2016. Transmission of influenza reflects seasonality of wild birds across the annual cycle. Ecology Letters 19:915-925.

Kruse, K. 2015. Central flyway mid-winter survey results. U.S. Fish and Wildlife Service. Lakewood, CO, USA.

Locke, L. N., and M. Friend. 1989. Avian botulism: geographic expansion of a historic disease. U.S. Fish and Wildlife Service, Fish and Wildlife Leaflet 13.2.4. Washington, D.C., USA.

Patz, J. A., S. H. Olson, C. K. Uejio, and H. K. Gibbs. 2008. Disease emergence from global climate and land use change. Medical Clinics of North America 92:1493-1491.

Robertson, D. G., and R. D. Slack. 1995. Landscape change and its effects on the wintering range of a lesser snow goose Chen caerulescens caerulescens population: a review. Biological Conservation 71:179-185.

Samuel, M. D., R. G. Botzler, and G. A. Wobeser. 2007. Avian cholera. Pages 239-269 *in* N. J. Thomas, D. B. Hunter and C. T. Atkinson, editors. Infectious diseases of wild birds. Blackwell Publishing Professional, Ames, Iowa, USA.

Simpson, V. R. 2002. Wild animals as reservoirs of infectious diseases in the UK. The Veterinary Journal 163:128-146.

Smith, B. J., K. F. Higgins, and W. L. Tucker. 1990. Precipitation, waterfowl densities and mycotoxins: their potential effect on avian cholera epizootics in the Nebraska Rainwater Basin Area. Transactions of the North American Wildlife and Natural Resources Conference 55:269–282.

Soos, C., and G. Wobeser. 2006. Identification of primary substrate in the initiation of avian botulism outbreaks. Journal of Wildlife Management 70:43-53.

United States Department of Agriculture [USDA]. 2015. USDA questions and answers: avian influenza outbreaks in the United States. Office of Communications, Washington, D.C., USA.

United States Department of Agriculture [USDA]. 2016. Animal and Plant Health Inspection Service biosecurity for birds. HPAI 2014/15 confirmed detections. <https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-disease-information/avian-influenza-disease/sa_detections_by_states/hpai-2014-2015-confirmed-detections>. Accessed 30 July 2016.

Van Hemert, C. R., J. M. Pearce, and C. M. Handel. 2014. Wildlife health in a rapidly changing north: focus on avian disease. Frontiers in Ecology and the Environment 12:548-556.

Wobeser, G. A. 1997. Diseases of Wild Waterfowl. Second edition. Plenum Press, New York, New York, USA.

Table 1.1 List of common avian diseases of concern (Friend and Franson 1999)

Disease	Type	Species Commonly affected	Season*	Comments
Avian cholera *(Pasteurella multocida)*	Bacteria	Geese Ducks Swans Coots	Fall through Spring	• Assumed first occurrence was mid to late 1800s • Carriers have been found in Snow Goose (*Chen caerulescens*) breeding colonies • Primary source of infection: Environmental contamination from diseased birds • Multiple strains • Affects wildlife and domestic animals • Primarily affects geese and ducks due to their gregarious nature • Annual occurrence in some parts of North America • Outbreaks coincide with migration
Avian botulism *(Clostridium botulinum)* *Type C* *Type E*	Bacteria	Geese Ducks Swans Shorebirds Gulls Pheasants	July through September	• Optimal bacterial growth in laboratory 25° C to 40° C, protein source, and anaerobic environment • Toxin produced by bacterium • Seven toxin types • Type C, affects wild birds, cattle, horses, poultry and low risk to human health • Type E, affects shorebirds with high risk to human health • 12 major outbreaks since 1900; resulting in the mortality of around eight million waterfowl

Disease	Type	Species Commonly affected	Season*	Comments
Renal coccidiosis *(Eimeria sp.)*	Parasite	All birds	Breeding and Winter	• Cause of protozoal parasite • Affects renal tissues • Several species of coccidian • Distribution by oocysts in feces • Control of disease in wild population is not feasible
Aspergillosis *(Aspergillus sp.)* *(A. fumigatus)*	Fungal	All birds	Late Fall through Winter	• Acute respiratory illness which is not contagious, but have resulted in large die-offs • One of the first noted avian disease (1813) • All birds are susceptible even mammals • Caused by moldy feed sources, moldy straw (e.g. straw used in nesting boxes), and other decaying organic matter in environments that promote fungal growth (e.g. agricultural waste)
Mycotoxicosis	Fungal/ Toxin	All birds	Fall through Winter (commonly associated with migration)	• Fungal infections that are not contagious • Difficult to diagnose • Causes the disease called Aflatoxin Poisoning • Fungi become more toxic once ingested and metabolized
Avian influenza	Virus	Waterfowl and shorebirds	Year round	• Transmission through fecal-oral routes • Possible 144 combinations of strains

* - Season indicates when disease is most prevalent. Most diseases are represented throughout the calendar year.

Note: This is not a comprehensive list of avian diseases.

Table 1.2 General examples of routes and exposures to disease for wildlife (Arnall and Keymer 1975, Friend and Franson 1999).

Routes and exposures	Examples
Ingestion	• Food, Water
Inhalation	• Fungal Spores, Sprays, Aerosols
Injection	• Bites (Animal or Insect), Sting, Puncture
Absorption	• Indirect and Direct Contact

Note: This table is not all inclusive to routes and exposures with their examples.

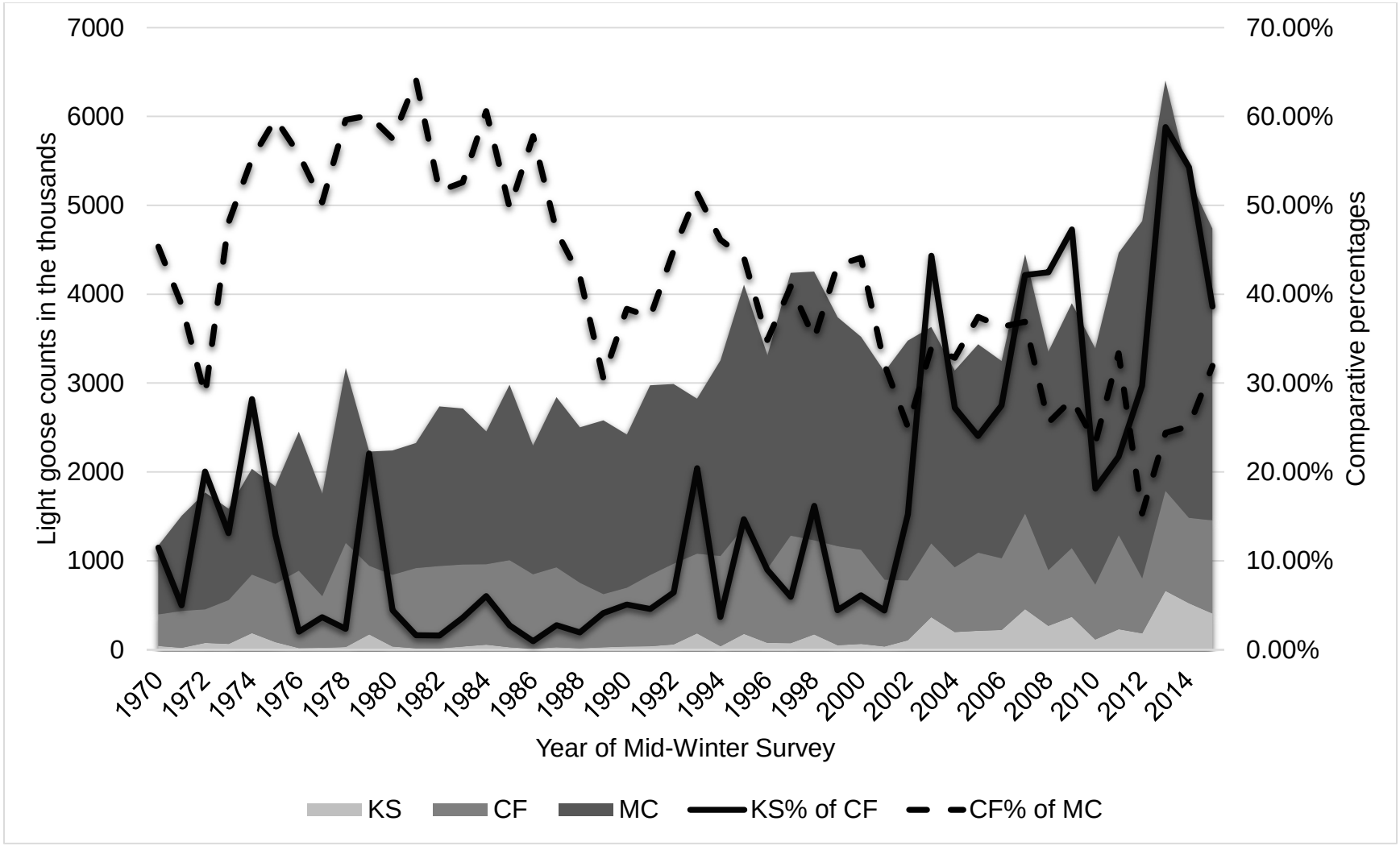

Figure 1-1 Annual light goose counts from the Central Flyway Mid-Winter Survey (1970-2015). This graph shows the visual comparison of counts (conducted in January) in Kansas (KS), Central Flyway (CF), and Mid-Continent (MC) wintering populations. The shaded area represents the populations in the thousands. The dashed line represents the percentage of Kansas mid-winter light goose population relative to Central Flyway population. The solid line represents the percentage of Central Flyway population relative to Mid-Continent population (Kruse 2015).

Chapter 2 - Occurrence of Avian Diseases in Kansas

Introduction

There is an array of diseases that can potentially affect wildlife populations. Specific diseases that affect wild migratory birds include avian cholera, avian botulism, duck plague, aspergillosis, west Nile, Newcastle disease, and avian influenza (Chapter 1). Diseases are usually categorized as bacterial, viral, fungal, parasitic, or toxic (Arnall and Keymer 1975, Friend and Franson 1999). Some diseases are known to cause detrimental effects local waterfowl populations (e.g., duck plague, avian cholera), in some instances causing die-offs equating to tens of thousands at one location (Friend and Franson 1999)

Avian cholera and avian botulism are bacterial diseases that frequently affect migratory wild birds and have the potential for large outbreaks (Blanchong et al. 2006, Samuel et al. 2007, Wobeser 2012). The causal agents of avian cholera and avian botulism are *Pasteurella multocida* and *Clostridium botulinum*, respectively. Avian cholera is an acute disease that can cause mortality in 6-8 hrs, but 24-48 hrs is a more common period of mortality (Friend and Franson 1999, Samuel et al. 2007, Wobeser 2012). Waterfowl most frequently affected by avian cholera are snow goose (*Chen caerulescens*) and northern pintail (*Anas acuta*). In the Central Flyway, avian cholera outbreaks typically occur in the Rainwater Basin of Nebraska and waterfowl wintering areas in Texas (Blanchong et al. 2006, Samuel et al. 2007, Webb et al. 2010). Avian botulism is a paralytic disease caused by a toxin (Types C and E) produced by the bacteria *Clostridium botulinum* (Friend and Franson 1999, Wobeser 2012). This disease is often fatal for many species of waterfowl; however, it is thought that dabbling duck species are most at risk (Friend and Franson 1999). This disease can persist for several months and is commonly

associated with warm seasonal temperatures and fluctuating water levels (Soos and Wobeser 2006, Wobeser 2012).

Duck plague, west Nile, and Newcastle are viral diseases. Duck plague, caused by the herpes virus, is an acute and fatal disease. Although not as common as other diseases, duck plague is highly contagious and infects ducks, geese, and swans (Friend and Franson 1999, Wobeser 2012). West Nile is a virus spread primarily by mosquitos, documented in >111 species of birds (Campbell et al. 2002), and believed to have spread throughout the United States along migratory songbird routes. Within a three-year period, this aggressive disease spread from New York, where it was first reported in North America, to the west coast (Komar 2003). This is a disease of great concern due to its ability to infect mammals and humans in addition to birds. Newcastle disease typically affects colonial nesting water-birds; most commonly, double-crested cormorant (*Phalacrocorax auritus*) breeding colonies (Friend and Franson 1999). This disease can result in high rates of mortality during outbreaks. Aspergillosis is a respiratory fungal disease most commonly caused by *Aspergillus fumigatus*. Migratory species foraging on waste grains appear to be particularly susceptible, especially during late winter and early spring when individuals are immunosuppressed, affected by eternal stressors, and environmental conditions are optimal for fungal growth (Arnall and Keymer 1975, Friend and Franson 1999). This disease infects mammals in addition to avian species.

A wide variety of avian species have been affected by diseases within wetland and wildlife areas in Kansas, USA. Disease outbreaks occur frequently in Kansas, often resulting in the mortality of hundreds to thousands of birds. For example, approximately 15,000 birds died as a result of an unidentified disease event in 1967 at Cheyenne Bottoms Wildlife Refuge, Barton County (Cheyenne Bottoms Wildlife Refuge, unpublished report). Events such as this are usually

overshadowed by other mortality events of larger size and annual frequency occurring elsewhere within the Central Flyway (e.g., Nebraska, Texas). Kansas' infrequent reported disease history has resulted in the lack of information and disease event recording.

Located in the Central Flyway, Kansas has become a vital layover stop for migratory waterfowl and more recently a possible wintering area for some species (i.e., snow goose [*Chen caerulescens*], Ross's goose [*C. rossii*]) due to climate and landscape changes (Robertson and Slack 1995). With increasing concerns for threatened and endangered migratory species (e.g., whooping crane [*Grus americana*]), the health of wetland ecosystems (Friend 2002), and threats to livestock and human health, surveillance and monitoring for diseases are typically at the forefront of discussion of wildlife managers (Boadella et al. 2011). These issues should move past discussion and onto paper by means of a disease management plan at the state- and station-level and a centralized disease reporting database maintained by the state. Because much remains unknown about migratory bird diseases, surveillance and monitoring schemes should be established to determine factors that result in disease outbreaks. My objectives were to (1) compile all known records of avian disease outbreaks for Kansas wildlife and wetland management areas, (2) assess the recovered documents and combine them into a centralized database, and (3) assess frequency of disease occurrence, species affected by diseases, and temporal trends of disease reports. I analyzed historical data collected throughout Kansas, and documented disease outbreaks with species affected, environmental indices at the time of outbreak, and population abundance at risk from 1967 to 2014.

Methods

Narrative reports from Kansas Department of Wildlife, Parks, and Tourism's (KDWPT) individual wildlife area offices and U.S. Geological Survey's National Wildlife Health Center

database were the primary sources utilized for this retrospective survey. I supplemented these records with a search of records in published literature and other government agency reports.

Study area

Kansas is situated within the Central Flyway and encompasses roughly 213,100 km^2 in the central United States. The state includes many state and federal parks, reservoirs, wildlife areas and fishing lakes primarily developed for recreational activities. The primary contiguous wetland system in Kansas is the Cheyenne Bottoms Wildlife Area, which is a 16,591-ha land sink located in Barton County, Kansas. As the largest inland freshwater marsh in the United States, it was designated as a RAMSAR (intergovernmental environmental agreement developed by the Ramsar convention; Ramsar, Iran [Pavri and Aber 2004]) wetland of international importance in 1988 (Aber et al. 2006). Located approximately 29 km from Cheyenne Bottoms was Quivira National Wildlife Refuge; this unique salt marsh and sand prairie encompassed 8,958 -ha. The majority of data were retrieved from these areas; however, a complete search for records was conducted throughout Kansas.

Data collection

I created a spreadsheet that included names, phone numbers, and email addresses of area wildlife managers and biologists from KDWPT, Kansas area U.S. Fish and Wildlife Service (USFWS) stations, and U.S. Geological Survey National Wildlife Health Center. I used the spreadsheet to systematically contact and note results of findings per conversations with wildlife personnel. Throughout summer 2014, direct contact was made with 44 individuals concerning availability of records for avian disease outbreaks in their respective areas. An additional 85 wildlife personnel (including the preceding 44) were contacted by a general email requesting any information on avian disease outbreaks in Kansas. The most effective method of data retrieval

was phone calls, as this allowed explanation of research being conducted and questions to be answered. Personnel were called multiple times because biologists and area managers were frequently conducting field work, with limited availability for conversation.

Initial efforts were to obtain electronic versions of information files; however, most field notes and other data on disease outbreaks were only available as paper documents typically in storage. Physical retrieval at Cheyenne Bottoms Wildlife Management Area was necessary due to extensive storage of paper files. This location provided the most information because of time spent thoroughly searching files. Other wildlife area offices in Kansas did not view the necessity of a site visit to retrieve data. These offices either emailed or mailed photocopied records. Disease information collected by the USFWS field stations in Kansas was kept and maintained by the National Wildlife Health Center in Madison, Wisconsin. An extensive search on disease events in Kansas was made with the assistance of the Health Center.

I conducted a literature review to find available data on avian disease events in Kansas. I used Google Scholar, Web of Science core collection, Web of Science, and other databases provided by Kansas State University Libraries. Records were noted and compared to findings from the statewide search. Other records recovered for analysis were weather and waterfowl population surveys. I obtained weather data associated with disease outbreaks from the Kansas State University Weather Data Library; these data included daily high and low temperatures as well as daily precipitation from site-specific weather stations located near the disease outbreak.

Population abundance indices of light geese (snow goose and Ross's goose) were obtained from the annual (January) Mid-Winter Inventory counts that were provided by USFWS Flyway representatives. I analyzed data from 1970 to 2015 to be consistent among the recovered records of disease events in Kansas. The Central Flyway data included counts for the mid-

continent population from North Dakota, South Dakota, Nebraska, Kansas, Oklahoma, and Texas. I did not include the Western Central Flyway Population which included Colorado, New Mexico, western Oklahoma, western Texas, western Nebraska, Wyoming, and Montana, because it does not include the study area for which I was reviewing. These indices proved a snapshot of the light goose population during the winter assuming there was no migration occurring at the time.

I used linear regression models to evaluate relationships between (1) weather indices (average high and low temperature and monthly precipitation) and disease outbreak type and (2) population abundance of light geese and disease frequency. Mid-Winter Inventory counts of light geese were logarithmic transformed prior to analysis. All analyses were performed in SAS v.9.3 (PROC GLM; SAS Institute, Cary, North Carolina, USA) and Program R.

Results

First event reported

During the fall of 1968, Cheyenne Bottoms Waterfowl Management area in Great Bend, Kansas, lost approximately 12,000 waterfowl to avian botulism, with approximately 63,000 potentially affected. This confirmed event was proceeded (1967) by an unconfirmed disease outbreak (believed to be caused by avian cholera) where 10,000 to 15,000 ducks and numerous shorebirds were reported dead. Specimens from the 1967 outbreak were submitted to the Denver Wildlife Research Center (which tested for pesticides) and Kansas State University College of Veterinary Medicine whose findings were inconclusive. So when there was a confirmed outbreak of avian botulism the following year, it was brought to the attention of a wildlife disease specialist from the Northern Prairie Wildlife Research Center in Jamestown, North Dakota. This

was Kansas' first attempt to, in great detail, record the loss of thousands of waterfowl and shorebirds due to disease.

Information recorded during the 1968 avian botulism event noted many circumstances that were believed to have lead up to the outbreak. This began with a water-level drawdown in the spring of 1968 in preparation of aerial seeding of the wetlands. Re-flooding started August 1, with the first losses of waterfowl noted on September 20, 1968. Concentrations of ducks were found around the exposed islands where hunting blinds were placed. Notable losses were to puddle ducks, American coot (*Fulica americana*), and shorebirds; however, no notable losses were caused to several hundred white pelicans (*Pelecanus erythrorhychos*) using the wetlands. Observed number of waterfowl using the wetlands was in excess of 75,000 with roughly 2,000 estimated deaths (~3% mortality).

It is likely to be the drawdown created the environmental conditions that favored a botulism outbreak. The drawdown caused a prolonged common carp (*Cyprinus carpio*) die-off, which in turn supported a massive blow fly (*Calliphoridae* spp.) larva population. During the re-flooding, many of the maggots, which are a source of the toxins for waterfowl (Arnall and Keymer 1975, Soos and Wobeser 2006), were believed to have washed into the pools where the outbreak occurred. It was concluded that anaerobic conditions (re-flooding), a protein source (the carp die-off), and warm temperatures were causal agents of this particular outbreak (Arnall and Keymer 1975, Locke and Friend 1989, Soos and Wobeser 2006).

1967-2014

All reported disease events from 1967 through 2014 (n = 32) were recorded and documented (Figure 2.1; Appendix A). Records of seven unique diseases were reported in 17 locations throughout Kansas (Figure 2.2). Of these, 37.5% were a result of avian cholera (n =

12), 31.25% were avian botulism (n = 10), and remaining 31.25% (n =10) were a mix of five identified diseases and one unknown (Figure 2.3). The most frequent disease outbreaks occurred during March, September, and December (Figure 2.4).

Species distribution of mortalities involved with avian cholera were 81% snow goose (n = 7,310), 5% greater white-fronted goose (n = 445, *Anser albifrons*), 7% mallard (n = 628, *Anas platyrhynchos*), and the remaining 7% was comprised of 17 other species. The majority of species succumbing to avian botulism were dabbling ducks with 21% green-winged teal (n = 4,678, *A. crecca*), 14% blue-winged teal (n = 3,027, *A. discors*), 14% northern pintail (n = 3,013, *A. acuta*), 10% American wigeon (n = 2,290, *A. americana*), and the remaining 41% were comprised of 42 different additional species including unidentified shorebirds and waterfowl. Due to the nature of botulism and environmental conditions favoring rapid decay, it is difficult to positively identify some species (Blanchong et al. 2006).

Botulism, is associated with warm temperatures (Arnall and Keymer 1975, Locke and Friend 1989, Friend and Franson 1999); the average temperature within 30 days of confirmed botulism outbreaks was between 25º C to 40º C, which is an optimal bacterial growth range (Friend and Franson 1999, Soos and Wobeser 2006). The minimum average monthly high for a botulism recorded event was 23.672º C and maximum of 36.161º C with a mean of 31.043º C. There were 10 reported events for avian botulism ranging from June to October with greatest frequency in September. There was no statistical relationship between the reported botulism events per year and the mean temperature during the months most susceptible of disease outbreaks ($F_{(1, 45)} = 1.02$, $P = 0.32$). The effects of other environmental conditions (e.g., water level fluctuations, precipitation, etc. [Locke and Friend 1989, Friend and Franson 1999]) were not assessed due to the lack of reported disease information within the records.

There were 12 reported events for avian cholera ranging from November to March with December being the mode, and one event in May. The relationship between avian cholera and minimum average temperatures (minimum = -10.35°C, maximum = 6.29°C, mean = -3.48°C) in Kansas was not significant ($F_{(1, 46)} = 0.06$, $P = 0.81$). Events appear to be associated with fall and spring migration; however, this information was not included in these data and comparisons were not attempted. Other analyses (i.e., water quality) were not conducted due to lack of evidence in the disease reports.

Light goose counts

The frequency and occurrence of disease outbreaks involving migratory birds within Kansas has increased since 2000. The Mid-Wintering Inventory of light goose counts in Kansas provided a range of 8,111 (1986) to 661,928 (2013) with a mean of 129,916 (1970-2014) (Figure 2.5). Ten disease events were reported to affect light goose populations in Kansas. Of these events, eight (80%) occurred after 1998, corresponding to increased populations of light geese. The mortality counts for these eight events ranged from 6 to 4,039. Because it may not have been possible to detect all carcasses due to decay and submersion, these reports likely underestimate the extent of mortality associated with these events (Blanchong et al. 2006, Soos and Wobeser 2006). All but two of these events were the result of avian cholera; the remaining two were due to outbreaks of aspergillosis. There was a positive relationship between the Mid-Winter count of light geese and frequency of disease outbreak ($F_{(1, 43)} = 4.45$, $P = 0.04$) during the 45 years (1970 – 2015) of data. Many of these years there were no reported disease events, however, there is an increasing frequency of disease occurrence during the past decade (Figure 2.6).

Discussion

My study revealed both inconsistent and nonexistent reporting of avian disease outbreaks in Kansas, USA. Efforts to record disease events was not a consistent priority for wildlife personnel. This may be due to the increased effort required to survey a disease outbreak (e.g., extra personnel, hours, and a delay of daily duties [Friend and Franson 1999, Soos and Wobeser 2006]). With a few cases, retrospective recording of disease outbreaks resulted in rough estimations in observed mortality of avian species; however, there were cases of disease outbreak reports that were well documented, with extensive records kept on the wildlife area. These well-documented cases will be helpful in developing future collaborative disease management plans for Kansas Department of Wildlife, Parks, and Tourism. Future development of disease reporting at the state level requires a greater emphasis on disease surveillance and documentation.

Future surveillance efforts should provide a better spatial and temporal understanding of disease outbreaks (Chan et al. 2010). The data I could recover spanned over four decades with few gaps in time of no reported occurrence. Friend and Franson (1999) suggested that the lack of disease reports may not be due to the lack of events, but rather the lack of reporting, such as what I believe may have occurred in Kansas.

Recorded avian cholera outbreaks in Kansas show seasonality ranging from November to March, with December being the mode; coinciding well with fall and spring waterfowl migrations (Wobeser 1997, Friend and Franson 1999, Samuel et al 2007). Avian cholera cannot persist for long periods in a wetland system; rather it is associated with a couple primary species (e.g., northern pintail and snow goose; [Samuel et al. 2005, Shadduck et al. 2006]). The majority of recovered carcasses from cholera die-offs were snow geese (estimated recovered 7,310) which are believed to be the primary carriers of *P. multocida* (Friend and Franson 1999, Blanchong et

al. 2006, Shadduck et al. 2006, Samuel et al. 2007). The risk of disease outbreaks is believed to be associated with species abundance at epizootic locations, but past findings were inconclusive (Blanchong et al. 2006); however, my results show that as light goose abundance increased throughout the state reported disease events involving light geese increased.

With the shifts in global climate, I anticipate an increase in the probability of disease outbreaks in Kansas, because of the increased residence time by migratory birds residing in the state for longer periods during winter and migration. The Mid-Winter Inventory data (Kruse 2015) indicates a 213% increase in wintering light geese in Kansas during the past 20 years (when comparing the five year averages of 1995-2000 and 2010-2014), concurrent with these population increases are increases in reported disease outbreaks within the last 15 years. This observation suggests that as light goose population densities increase, the probability of a disease outbreak (primarily avian cholera) increases as well (Smith and Higgins 1990, Webb et al. 2010). This is due to physiological stressors (e.g., extreme temperature change, weather conditions, resource availability) caused by the increasing abundance of geese in confined locations during migration (Smith and Higgins 1990, Arzel et al. 2006, Webb et al. 2010). These stressors may lead to more immunosuppressed individuals where disease risk is increased (Wobeser 1997, Arzel et al. 2006). These data are limited and reporting of disease outbreaks is essential for the future of disease forecasting and understanding. Avian cholera is currently becoming the disease dominating avian mortality in North America (Friend and Franson 1999, Samuel et al. 2007). Avian botulism follows avian cholera as the most common avian disease causing mortality (Rocke and Samuel 1999, Soos and Wobeser 2006).

Prior to an avian botulism outbreak, there are many complex ecological relationships that are believed to occur prior to an epizootic event. Botulism bacterial growth is likely the result of

poor wetland health. These factors that contribute to poor wetland health can be influenced by water fluctuations, pH, salinity, seasonal temperatures ranging from 25º C to 40º C, and other environmental conditions (Wobeser 1997, Friend and Franson 1999, Rocke and Samuel 1999, Soos and Wobeser 2006). Warmer temperatures during June through October may relate to increased frequencies of annual botulism outbreak events. Although higher temperatures are not the mechanistic cause of botulism outbreaks, they provide an easily measured index for other factors that are known to affect disease outbreaks (Rocke and Samuel 1999, Soos and Wobeser 2006). Rocke and Samuel (1999) found that water temperatures influenced *C. botulinum* growth by amplifying pH of the water, which in turn increased risk and broadened the range of suitable conditions for botulism. Water temperatures also influences the effects of invertebrate involvement (e.g., maggots; Rocke and Samuel 1999, Soos and Wobeser 2006). It is likely with the shifting global climate, Kansas' risk of avian cholera outbreaks will increase as seasonal temperatures continue to rise, and drought and precipitation events become more severe (Easterling et al. 2000, Patz et al. 2008, Dai 2013).

The narratives of botulism events from Cheyenne Bottoms indicate there was prior drawdowns of water in the pools affected by disease or precipitation totals ≥15 cm. Those few instances where rain events were ≥ 15 cm occurred during the month or month prior to an outbreak at Cheyenne Bottoms in 1993, 1998, and 2000. The largest amount of rain totals for the month prior to the epizootic event occurred during 1993 (32.66 cm). My limited dataset prevented a formal analysis of this relationship. Future research should attempt to relate water fluctuations, water and ambient air temperature, and other possible characteristics that promote *C. botulinum* growth in wetland ecosystems.

Due to nature of disease and decomposition, detectability of carcasses during any disease event may have been an issue when estimating mortality (Soos and Wobeser 2006). There are a number of biases to overcome when estimating mortality due to disease loss; such as carcasses loss to scavengers, imperfect detection by searchers, variable ecological site characteristics (e.g., vegetation height, type and density, and water depth and clarity), species characteristics (e.g., size and colorization), mortality occurring outside primary search area, and time (Homan et al. 2001, Soos and Wobeser 2006, Huso 2010). These biases in addition to carcass decomposition may cause underestimation in estimated losses and may not reflect the impact of disease on the affected population.

In attempt to relate environmental variables (monthly average high and low temperature, and monthly precipitation) to disease outbreaks, I discovered that there was insufficient evidence from this study to suggest such relationships. Efforts to develop predictive models for disease occurrence were unsuccessful, primarily due to the complex ecology of each disease and lack of disease reporting data in Kansas. To better understand the disease dynamics occurring in Kansas wetlands, I recommend developing station and state disease management plans. These disease management plans should encompass a state-wide monitoring and surveillance scheme for a more holistic approach to disease investigations. Through the development of a plan such actions would be emplace to track and monitor disease events, as well as continue the database and assist investigators in future efforts.

Literature Cited

Aber, J. S., S. W. Aber, F. Pavri, E. Volkova, and R. L. Penner. 2006. Small-format aerial photography for assessing change in wetland vegetation, Cheyenne Bottoms, Kansas. Transactions of the Kansas Academy of Science 109:47-57.

Arnall, L., and I. F. Keymer. 1975. Bird diseases. An introduction to clinical diagnosis and treatment of diseases in birds other than poultry. Bailliere Tindall, London, England.

Arzel C., J. Elmberg, and M. Guillemain. 2006. Ecology of spring-migrating Anatidae: a review. Journal of Ornithology. 147:167-184.

Blanchong, J., M. Samuel, and G. Mack. 2006. Multi-species patterns of avian cholera mortality in Nebraska's rainwater basin. Journal of Wildlife Diseases 42:81-91.

Boadella, M., C. Gortazar, P. Acevedo, T. Carta, M. P. Martín-Hernando, J. Fuente, and J Vicente. 2011. Six recommendations for improving monitoring of diseases shared with wildlife: examples regarding mycobacterial infections in Spain. European Journal of Wildlife Research 57:697-706.

Campbell, G.L., A. A. Marfin, R. S. Lanciotti, and D. J. Gubler. 2002. West Nile virus. Lancet Infectious Diseases 2:519-529.

Chan, E. H., T. F. Brewer, L. C. Madoff, M. P. Pollack, A. L. Sonricker, M. Keller, C. C. Freifeld, M. Blench, A. Mawudeku, and J. S. Brownstein. 2010. Global capacity for emerging infectious disease detection. Proceedings of the National Academy of Sciences of the United States of America 107:21701-21706.

Dai, A. 2013. Increasing drought under global warming in observations and models. Nature Climate Change 3:52-58.

Easterling, D.R., G.A. Meehl, C. Parmesan, S.A. Changnon, T.R. Karl, and L.O. Mearns. 2000. Climate extremes: observations, modeling, and impacts. Science 289:2068-2074.

Friend, M., and J. C. Franson. 1999. Field manual of wildlife diseases. General field procedures and diseases of birds. Biological Resources Division Information and Technology Report 1999-01, US Department of the Interior and US Geological Survey, Washington, D.C., USA.

Homan, H., G. Linz, and B. Peer. 2001. Dogs increase recovery of passerine carcasses in dense vegetation. Wildlife Society Bulletin 29:292-296.

Huso, M. M. 2011. An estimator of wildlife fatality from observed carcasses. Environmetrics 22:318-329.

Komar, N. 2003. West Nile virus: epidemiology and ecology in North America. Advances in Virus Research 61:185-234.

Kruse, K. 2015. Central flyway mid-winter survey results. U.S. Fish and Wildlife Service. Lakewood, CO, USA.

Leopold, A. 1987. Game management. University of Wisconsin Press.

Locke, L. N., and M. Friend. 1989. Avian botulism: geographic expansion of a historic disease. U.S. Fish and Wildlife Service, Fish and Wildlife Leaflet 13.2.4. Washington, D.C., USA.

Patz, J. A., S. H. Olson, C. K. Uejio, and H. K. Gibbs. 2008. Disease emergence from global climate and land use change. Medical Clinics of North America 92:1493-1491.

Pavri, F., and J. S. Aber. 2004. Characterizing wetland landscapes: A spatiotemporal analysis of remotely sensed data at Cheyenne Bottoms, Kansas. Physical Geography 25:86-104.

R Core Team. 2016. R: A language and environment for statistical computing. R Foundation for Statistical Computing. Vienna, Austria. URL https://www.R-project.org/.

Robertson, D. G., and R. D. Slack. 1995. Landscape change and its effects on the wintering range of a lesser snow goose Chen caerulescens caerulescens population: a review. Biological Conservation 71:179-185.

Samuel, M. D., R. G. Botzler, and G. A. Wobeser. 2007. Avian cholera. Pages 239-269 *in* N. J. Thomas, D. B. Hunter and C. T. Atkinson, editors. Infectious diseases of wild birds. Blackwell Publishing Professional, Ames, Iowa, USA.

Shadduck, D., M. Samuel, D. Goldberg, D. Shadduck, and L. Creekmore. 2006. Wetland environmental conditions associated with the risk of avian cholera outbreaks and the abundance of Pasteurella multocida. Journal of Wildlife Management 70:54-60.

Smith, B. J., K. F. Higgins, and W. L. Tucker. 1990. Precipitation, waterfowl densities and mycotoxins: their potential effect on avian cholera epizootics in the Nebraska Rainwater Basin Area. Transactions of the North American Wildlife and Natural Resources Conference 55:269–282.

Soos, C., and G. Wobeser. 2006. Identification of primary substrate in the initiation of avian botulism outbreaks. Journal of Wildlife Management 70:43-53.

Webb, E. B., L. M. Smith, M. P. Vrtiska, and T. G. Lagrange. 2010. Effects of local landscape variables on wetland bird habitat use during migration through the rainwater basin. Journal of Wildlife Management 74:109-119.

Wobeser, G. A. 1997. Diseases of wild waterfowl. Plenum Press, New York, New York, USA.

Wobeser, G. A. 2012. Diseases of wild waterfowl. Springer Science & Business Media.

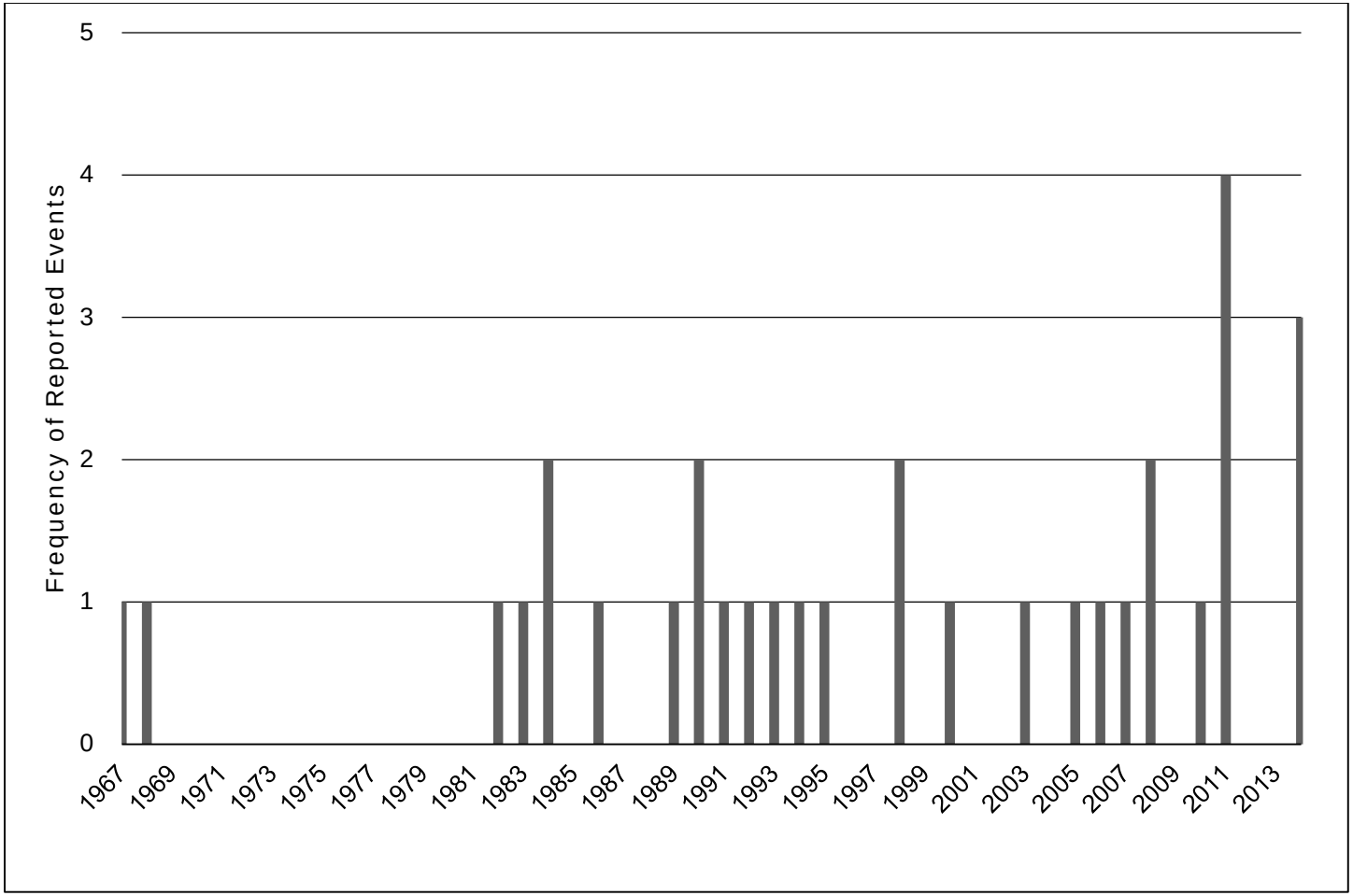

Figure 2-1 Temporal distribution of all reported avian disease outbreaks in Kansas (*n* = 32) from 1967-2014

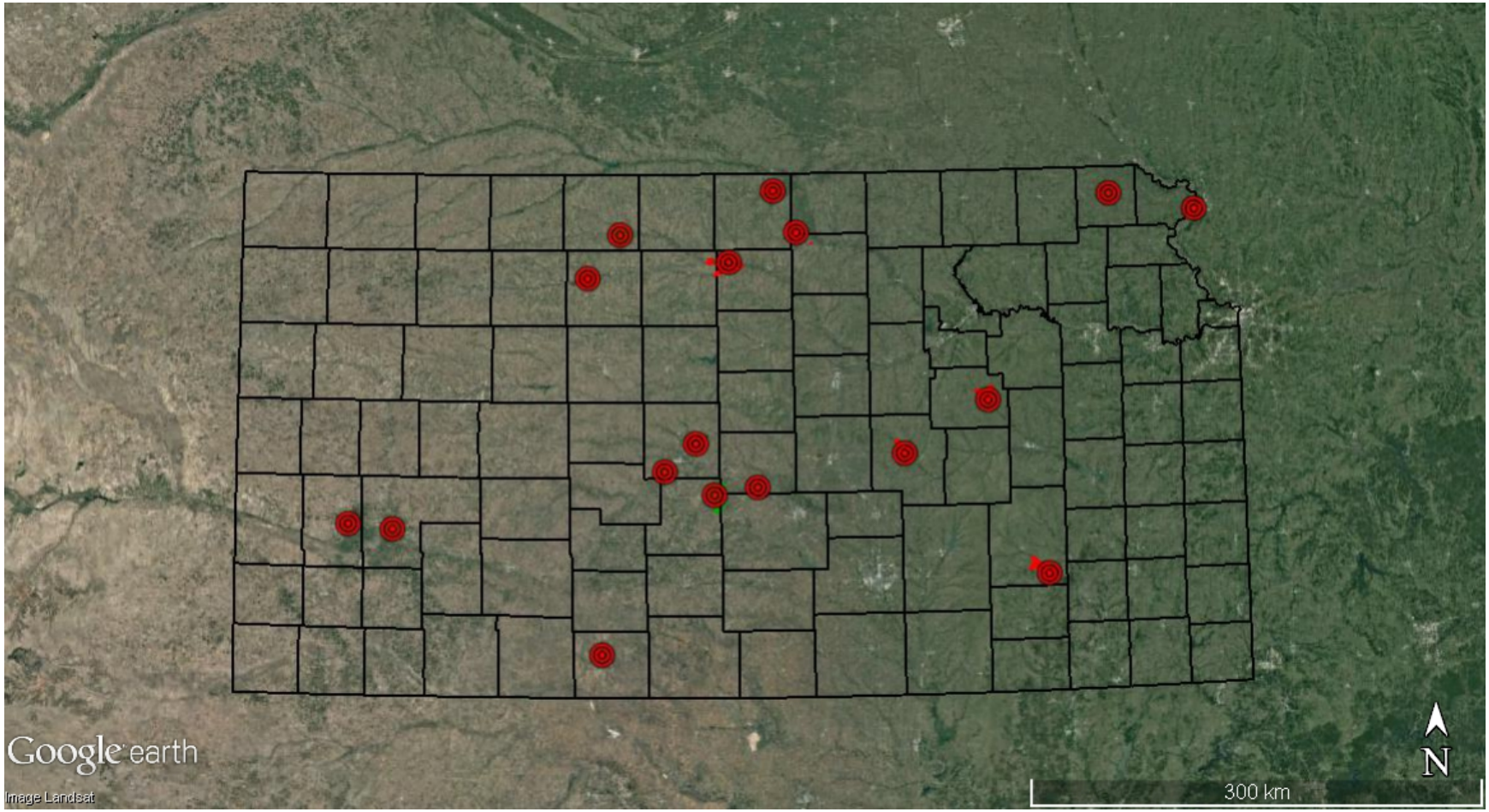

Figure 2-2 Spatial distribution of reported avian disease outbreaks in Kansas during 1967-2014.

***Note*: Location of icons are approximate to disease location**

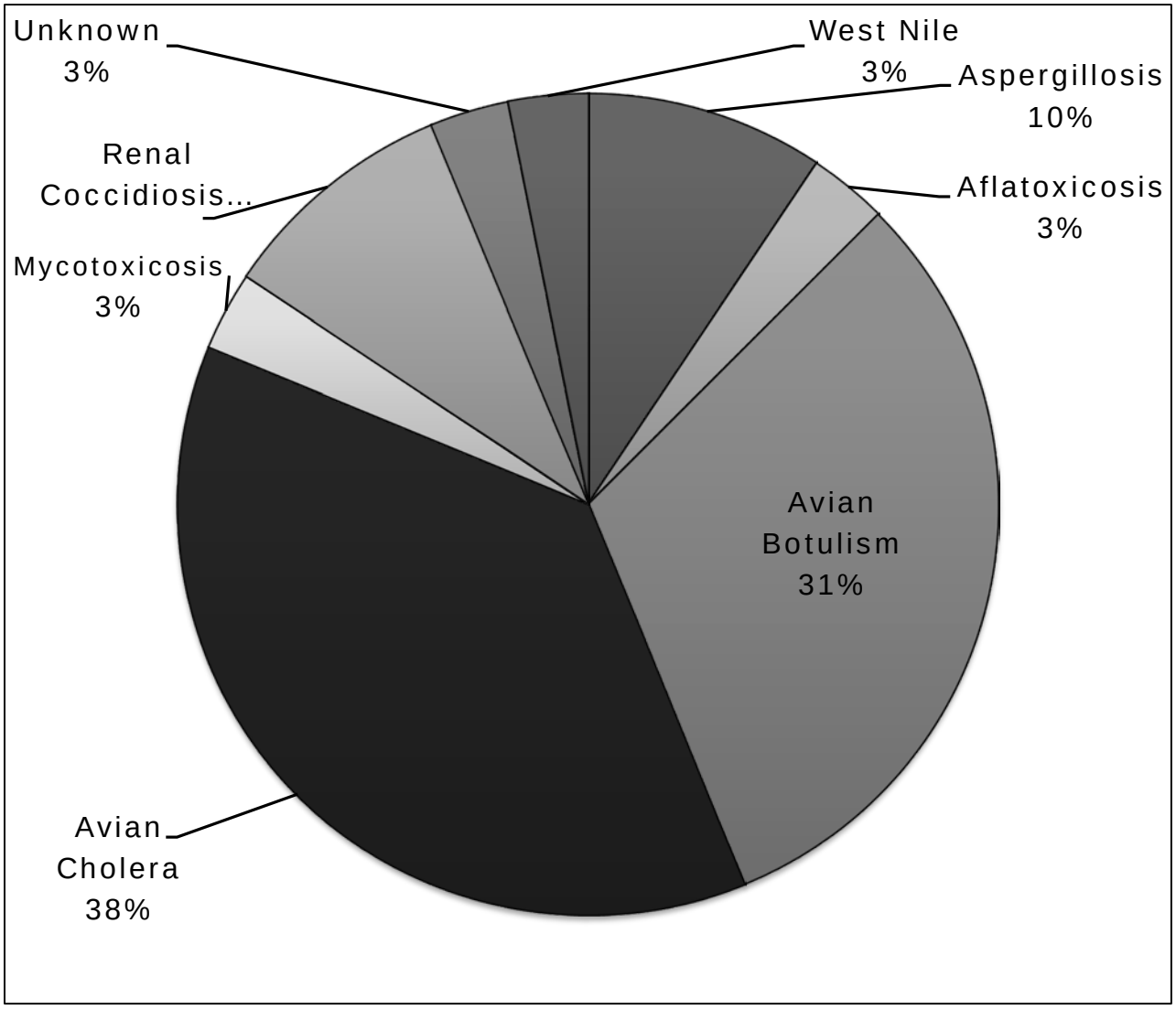

Figure 2-3 Relative distribution of avian disease types recorded during outbreaks in Kansas from 1967-2014

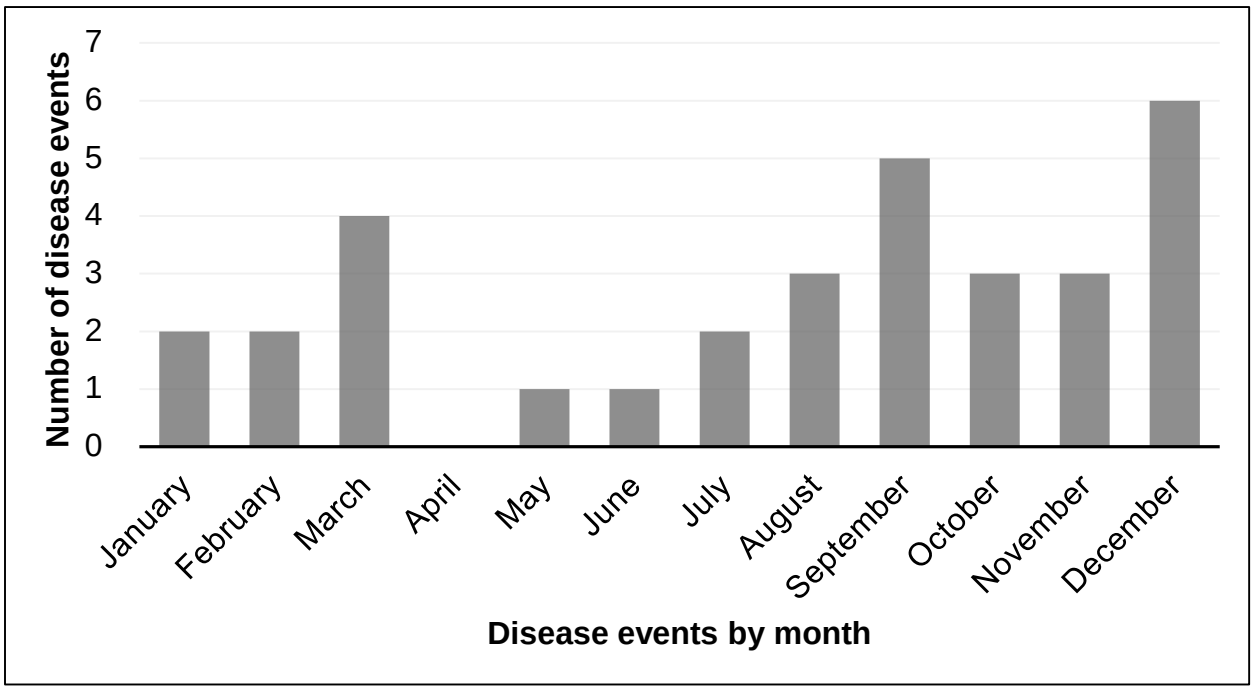

Figure 2-4 Monthly frequency of reported avian disease outbreaks in Kansas (*n* = 32) from 1967–2014.

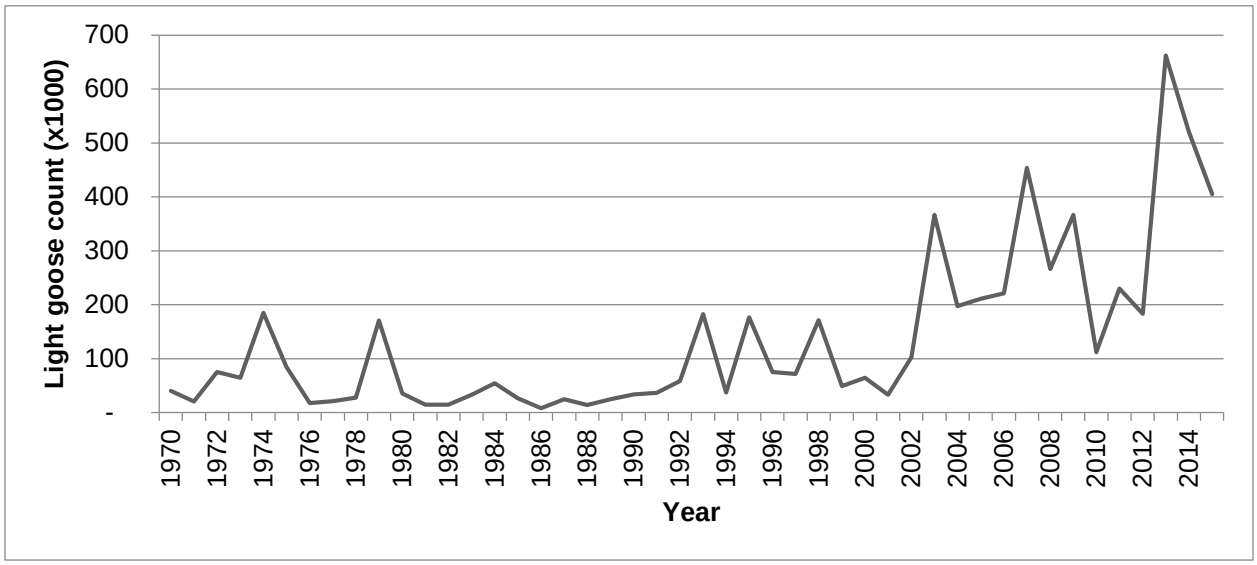

Figure 2-5 Combined abundance of snow goose (*Chen caerulescens*) and Ross's goose (*C. rossii*) as indexed by the annual January Mid-Winter Inventory in Kansas.

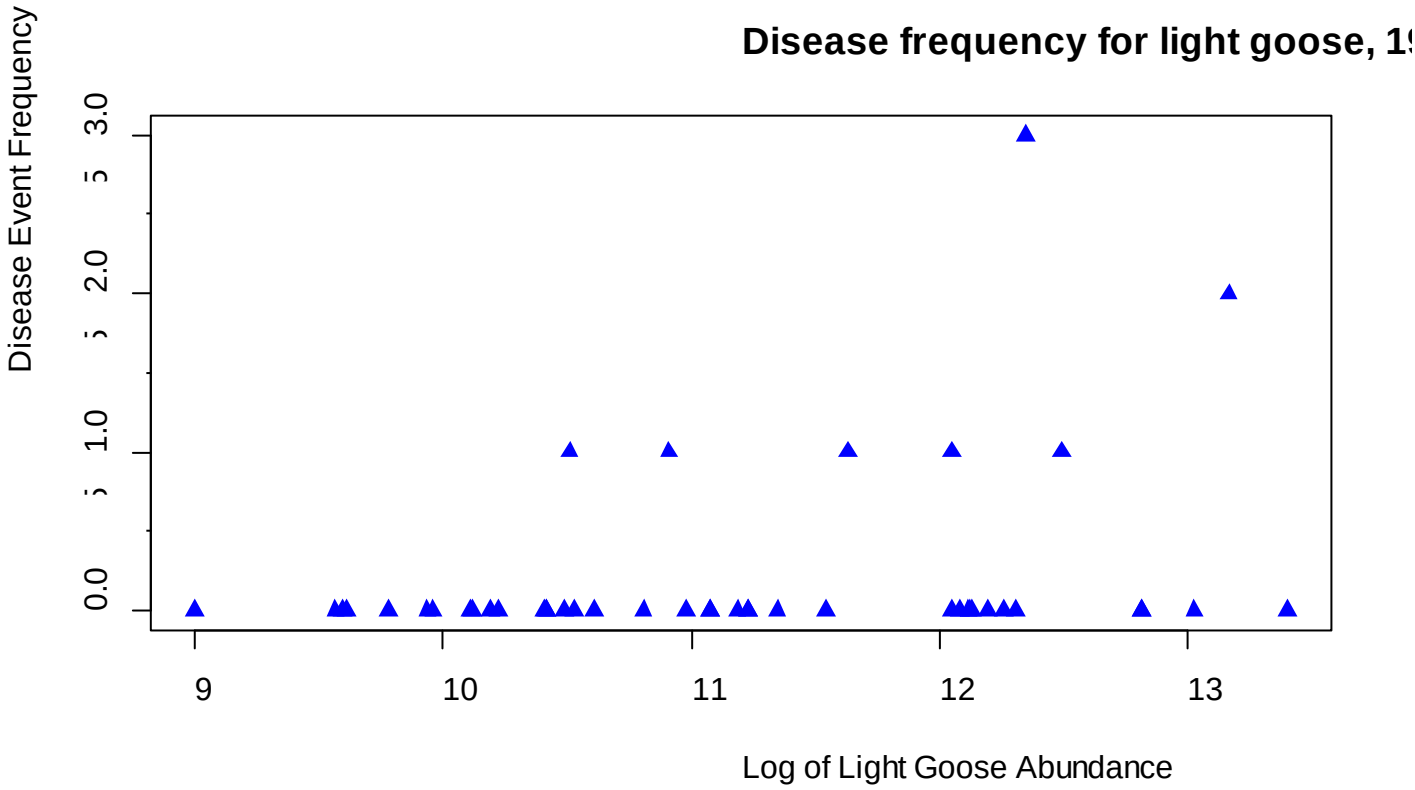

Figure 2-6 Frequency of reported disease events for waterfowl in Kansas relative to the log counts of light geese during the annual January Mid-Winter Inventory from 1970-2014.

Chapter 3 - Management of Factors Affecting Waterfowl Disease Outbreaks

Many factors and influences contribute to an avian disease outbreak. Complex relationships among waterfowl species, habitat conditions, and environment relative to disease outbreaks are too numerous to depict with certainty (Wobeser 1997, Smith and Higgins 1990); however, there are a few indicators throughout the literature that represent contributing factors for a disease outbreak. These include, but are not limited to environmental factors (e.g., stressors, precipitation, temperature, water fluctuations, invertebrate populations), and species' health and demography (Smith and Higgins 1990, Friend and Franson 1999, Rocke and Samuel 1999). Although this is not a compendium of every factor that influences disease, it is a start to the understanding of these complex relationships.

An understanding of these complex relationships could be manifested by developing a disease monitoring and surveillance scheme. Most disease outbreaks are retrospectively studied after it occurred and investigators are left attempting to account for missing data (Wobeser 1997). Developing a database to compile the monitoring and surveillance data can assist in associating disease events with environmental factors and developing disease management plans.

The purpose of this chapter is to describe our basic understanding of what factors are involved in an avian disease event, strengthen the argument for continued monitoring and surveillance, and develop an outline for a disease management plan. The future disease management is based on decisions made today; this is why it is important to reiterate that there is still much unknown about avian diseases, whereby monitoring present environmental conditions today help determine conditions for disease outbreaks tomorrow. By using data from

standardized sampling regimes to inform management actions, regional and local authorities can develop contingency plans for when a disease event occurs.

Factors Known to Cause Avian Disease Outbreaks

Environmental

Knowledge of environmental factors that contribute to disease events is essential for understanding when, where, and how outbreak events occur. Resource limitations, environmental variability, and wetland health may enhance species-specific disease risk (Friend and Franson 1999, Arzel et al. 2006). The interaction between extreme weather events and reduced food availability may increase the risk of a disease outbreak among avian species, because individual birds have a reduced ability to fight infection due to malnutrition (Wobeser 1997, Arzel et al. 2006).

Cold temperatures may cause waterfowl to congregate in larger and denser flocks for thermal regulation (Arnall and Keymer 1975) and ice formation causes waterfowl to congregate in small patches of open water possibly increasing infection risk (Samuel et al. 2007). Avian botulism is associated with high temperatures as well as water-level changes due to drought and precipitation events (Smith and Higgins 1990, Wobeser 1997, Friend and Franson 1999, Rocke and Samuel 1999). Degraded water quality appears to be a major contributor to disease events (Smith and Higgins 1990, Samuel et al. 2007).

The primary diseases affecting migrating and wintering waterfowl are avian cholera and botulism. There was 32 reported disease events from 1967 to 2014 in Kansas, with avian cholera (n = 12) and avian botulism (n = 10) representing 68.75% of diseases from seven disease types

reported (Chapter 2). Poor wetland health has been attributed to botulism (Rocke and Samuel 1999) and cholera outbreaks (Smith and Higgins 1990).

Degradation of wetlands reduces water quality, which, in turn, promotes bacterial growth for avian botulism and avian cholera. Rocke and Samuel (1999) concluded that environmental factors promoting botulism may include shallow, stagnant water with low dissolved oxygen; along with extreme water fluctuations within the wetland system. Rocke and Samuel (1999) reported pH (between 7.5 and 9.0) and water temperatures (> 20°C) magnify the outbreak risk, so monitoring water pH and temperatures may be beneficial to indicate an increasing botulism outbreak risk. Findings from Rocke and Samuel (1999) correspond well with documented records of summer and early fall outbreaks of botulism across North America (Friend and Franson 1999), as well as data from Kansas (Chapter 2), where botulism outbreaks occurred from June to October, with the mode being September. During this period, seasonal temperatures are usually within the favorable botulism growth range (25° C to 40° C [Friend and Franson 1999]). Determining the temporal patterns of disease outbreaks will assist managers in determining when to focus monitoring efforts. Wetland health is important in disease outbreaks and should be monitored; for example, when the wetland system is not able to support the fluctuation of migrating individuals, it in turn, reduces the health of individuals and associated population resulting in the increase risk of disease outbreak (Smith and Higgins 1990).

Environmental factors are difficult to determine during reaction stages of a newly developed disease agent, as well as known disease agents. With still much unknown about the roles environmental factors play in disease development, keeping records of environmental conditions prior, during, and after a disease outbreak would assist future investigators in developing insight to try and predict disease outbreaks. These data will benefit the development

of disease control measures for future outbreaks, and identify unique conditions during the time of the outbreak (Wobeser 1997, Friend and Franson 1999).

Species

Species affected by diseases may appear to be indiscriminant when large epizootic events occur; however, knowing what species are affected by disease is especially important during preliminary determination of a disease agent (Wobeser 1997, Friend and Franson 1999). Species specific disease outbreaks may be due to unique behaviors or ecological niches (Wobeser 1997, Altizer et al. 2011). For example, feeding habits (Wobeser 1997, Friend and Franson 1999), where filter-feeding and dabbling ducks are susceptible to toxins (e.g., botulism) found in wetland substrates and invertebrates (e.g., blow fly larva; Wobeser 1997), while other waterfowl with other feeding strategies in the same area are not (Wobeser 1997, Friend and Franson 1999). Phylogenetic differences in species also contribute to transmission and the resulting disease event. For example, avian cholera is known to be carried by snow geese (*Chen caerulescens*) populations and subsequently result in the majority of individuals recovered during cholera events (Friend and Franson 1999, Samuel et al. 2007). Members of the taxa family Anatidae are all susceptible to duck plague, but not all species respond to the disease equally; for example, the northern pintail (*Anas acuta*) is not as susceptible to the disease as blue-winged teal (*A. discors*) (Wobeser 1997). Determining primary species involved during the initial stages of a disease event is just the beginning puzzle piece to the bigger picture, which can include condition of individuals (Altizer et al. 2011).

Individuals

By individuals, I am referring to subsets within the population that are present during the time of an outbreak; this could be categorized as age (juvenile or adult) or sex cohorts within the

population susceptible to the outbreak. The health of individuals contributes greatly to the effects of the disease outbreaks (Altizer et al. 2011). Increasing abundance in a single confined area (i.e., frozen wetland with minimal amount of open water) can result in the mass outbreak of disease (Friend and Franson 1999, Altizer et al. 2011). Understanding the cohort of individuals most affected by a disease event should increase the ability of investigators to assess the effect of the event. For example, some disease agents are age or sex related, or affect females or males that are in situations where there is segregation among them (Friend and Franson 1999). Documenting the abundance, age and sex composition, and species involved during an outbreak will assist in determining future contributing factors of disease outbreaks.

Surveillance and Monitoring

With little known about wildlife diseases and their important roles within ecosystems and population dynamics of waterfowl (Beer 1988), monitoring and developing a surveillance protocol for avian disease events at local and state scales could inform predictive models of disease occurrence. I propose that there should be a state-level disease management plan as well as a station-level disease management plan in preparation of likely increasing disease events in Kansas (Chapter 2). The two plans should support one another to some degree for a proactive and reactive approach to a disease event. The state-level plan should focus on components that would be common among all responses to disease outbreaks, including disease identification, response equipment, personnel, disposal, and emergency funding. The station-level plan should reflect similar components; however, this plan should be tailored to meet the needs of the station and outline defined roles and responsibilities. All the while, during the development of these plans, an understanding that continually monitoring conditions during the response should be a high priority.

Monitoring

A fundamental reason for establishing a disease management plan would be to acquire standardized data investigative questions regarding the causal mechanisms of disease outbreaks, and potentially update monitoring protocols as additional information becomes available. Long-term standardized monitoring should be designed to assess conditions prior to and during a disease event, where environmental trends can be identified (Lindenmayer and Likens 2010). Long-term monitoring will be largely dependent on the funding and leadership to sustain the effort. Collaboration among state and federal wildlife professionals will be required to continue long-term monitoring, with agency specific responsibilities clearly established.

Disease surveillance plans should include known locations of past disease outbreaks, as well as opportunistic surveillance of locations with large numbers of waterfowl with no known history of disease. Opportunistic surveillance occurs, when wetland or wildlife biologists are conducting daily duties, but remain vigilant for observing potential disease die-offs. Surveillance frequency should be determined by wildlife personnel to be station specific; for example, inspections of prior disease areas in Kansas could be checked bi-weekly from September through May, but monthly June through September due to the infrequency of disease events during these months in recent years (Chapter 2). Transects should be planned for standardized surveillance, these transect specifics need to be explained in the state-level plan.

All mortality events of $n > 10$ individuals (determined by wildlife personnel) should be reported immediately to wetland or wildlife managers to initiate preliminary investigation of possible disease outbreak. Decomposition of carcasses may reduce detectability during any disease event, which may create issues while locating individuals (Soos and Wobeser 2006). Along with decomposition, there are numerous issues to overcome when locating carcasses, such

as visibility due to ecological site characteristics (e.g., vegetation height, type and density, and water depth and clarity) and species characteristics (e.g., size and colorization; (Homan et al. 2001, Soos and Wobeser 2006, Huso 2010). These issues in addition to carcass decomposition should be considered when developing plans to investigate suspected mortality events. Reports of disease outbreaks from the public or landowners should be investigated immediately. Strategies for including approaches to inform and educate the public on the necessity to report diseased and dead wildlife should be listed in the disease management plan.

Disease Management Plan

Due to the nature of disease events being unpredictable, disease management plans should be developed to be an adaptable guide, easily accessible to the parties involved, with clear responsibilities for all participants. Plans should also be updated frequently to assure contact personnel are current, and federal and state permits are quickly accessible. I developed a general outline and information that is recommended for inclusion in a plan (Appendix B).

Because disease can spread through wildlife populations rapidly, it is suggested that there be a disease management plan at the state and local level. These plans should be developed in coordination with disease specialists and wildlife biologists. The purpose of the state plan should be to develop general response procedures and outline the state's responsibilities during a disease event. Due to the uniqueness of disease outbreaks, plans should be used as guides rather than protocol, so that personnel are able to deviate actions if necessary.

Disease management plans should include but are not limited to the following: planning, initial response, disease control, surveillance, and analyses (Friend and Franson 1999). Planning is essential for the quick resolution of disease events and understanding of resources available to assist in the event of a disease outbreak. A response section should outline the roles and

responsibilities at state and station level for responding to a disease event, as well as reactive steps needed to manage disease mitigation. Disease mitigation proceeds disease control, which includes carcass collection and disposal, decontamination, and habitat manipulation (e.g., water level manipulation, burning, etc.). Surveillance of the infected area following a disease event should ensure that the event is officially concluded. Once the event is officially concluded and all resources have been returned, analysis of response to the event would improve future efforts.

Planning

An introduction page should outline the parameters of the region or area for which the disease management plan is developed, physical descriptions of the region or area, general information on past areas inflicted with disease, and overview of responsibilities. Within the planning section should an overview of the state and the potential "hotspots" for disease outbreak, for example, one location should be Cheyenne Bottoms Wildlife Area, Barton County, Kansas, due to its multiple disease outbreaks in the last 45 years (Chapter 2). The station level should have an overview of their area of responsibility, with the addition of the surrounding private land. The surrounding land is included because the request for assistance could come from a private landowner.

Included in planning is identification of special needs to effectively combat disease outbreaks. Special needs include sources of special resources, recording biological information, and preparing contingency plans (Friend and Franson 1999). Identifying the needs of a disease event are essential for a quick and effective response. To identify special needs for an area, plan developers need to include maps, pre-planned transects for surveillance, pre-determined work areas for disease operations, burn permits, state and federal permits, species in need of care

consultation, logistics for personnel, carcass disposal areas, and media relations (Friend and Franson 1999).

The planning stage should also include a biological information section, which could be included in the introduction section. This may include species involved (migratory and non-migratory), movement patterns, and past history of disease (Friend and Franson 1999). Descriptions of species migration patterns could assist in the identification of the potential for disease outbreaks as well as determine the extent of response necessary for disease mitigation. Increased magnitude of a response may be necessary during fall or spring migration, or for threatened species (e.g., whooping crane [*Grus americana*]) (Friend and Franson 1999, Altizer et al. 2011). To alleviate potential confusion, contingency plans are recommended to determine what should be occurring.

Contingency plans (also known as an "emergency action plan") are pre-planned actions to be taken when certain factors (e.g., disease, species, or season) exist. For example, if there were to be a cholera outbreak at location x, then y steps need to take place and these may differ from botulism response. Contingency plans should outline a myriad of activities and how those activities should take place. Such activities should include carcass collection, carcass disposal, decontamination procedures (environmental, personnel, and equipment), communications, surveillance and observations, population and habitat manipulations, and wildlife sampling and monitoring.

The following are examples of what these activities look like and what needs to be represented in the plan. Carcass collection, which could involve the drawdown of water to collect carcasses from substrate or collection involving observed deceased species by boat or wading, needs to include personnel protective clothing and equipment. Carcass disposal is dependent on

recommended practices and equipment by the most current literature, some examples are burial, burning, and compost (Friend and Franson, 1999). Decontamination procedures must be outlined and followed to prevent the spread of disease to other wildlife and in some cases to humans. Decontamination can be as simple as using buckets and brushes with a chlorine/water mixture to be used on equipment, and soap and water for personnel; however, I recommend consulting a disease specialist for the best method available. Communication activities are limited to the resources and funding available to the local station, examples include mobile phone and two-way radio. Wildlife sampling and monitoring activities should direct how biologists will monitor area for diseased wildlife during and after disease events.

I suggest that the state also include an evolving set of questions to be answered from the data collected during the surveillance and monitoring, so the plan will mitigate the opportunity for unproductive monitoring (Lindenmayer and Likens 2010). Questions should ask, but are not inclusive to, what environmental events lead to the disease outbreak, such as water temperature, water salinity, air temperature, significant precipitation, or flood events. The questions should be evolving because much still unknown about disease outbreaks. Data collection and storage should be coordinated at a state and station level; the state should maintain a database that encompasses all of its wildlife areas and station-level may maintain their own database. Funding for monitoring should be evaluated and directed by the state level plan. The long-term monitoring program will be in vain if there is not response to the disease events to collect further data and cleanup efforts.

Initial Response

The response section of the disease management plan should outline the roles and responsibilities of the individuals involved at the state and station level, as well as detail

preplanned recommended resources. These roles and responsibilities should be outlined to direct an initiation of a response to a disease event. Detailing the initiation of a disease response should occur when the identification of a disease event is confirmed. This includes diagnosing sick or dead birds through a field necropsy, and how and where to submit carcasses to obtain diagnostics by a disease laboratory. Packaging and shipping of specimens needs to be conducted in a timely manner and with precision; this is to prevent decay, proper identification, cross contamination, leaky fluids within the packaging, and ensuring proper labeling of all packages (Friend and Franson, 1999). Friend (1999) recommended contacting the designated disease laboratory for packaging and shipping instructions. An attachment should be developed identifying qualified laboratories with contact information and capabilities (Friend and Franson 1999). Finally, the initial response portion of the plan should include a collected list of personnel to call when additional resources are needed, and a list of resources to seek advice from a disease specialist (suggested to list contact information as an attachment).

Following the initial response outline of roles and responsibilities, an explanation detailing what should occur once a disease event is confirmed. Such actions could include the station manager initiating this pre-established protocol to deny access to affected areas and limit personnel into those areas as well as work areas, this is to prevent the further spread of the disease. This plan should outline how specific disease control measures should be carried out. Contamination control procedures should prearrange the course of actions and identification of work areas for which operations should take place. Other aspects of the initial response should include a communications plan, and determine where additional resources may be quickly obtained; recommendation of where to acquire general equipment is suggested in Appendix B.

When developing a plan with disease potential for being zoonotic, the upmost care must be taken to ensure the safety and health of personnel.

Disease Control

Disease control actions should be determined by the type of disease identified, environmental factors, species involved, and other circumstances. Disease control is where the plan outlines a response for when resources have been assembled and operations have begun. This is where management of the disease is actively being applied and includes management of the local public.

Management of the public can be difficult due to the large area of some wetlands, which is why it was recommended in the initial response to close off disease-affected areas. Members of the public will be curious and want to know what is ongoing at the local wetland or wildlife area. Therefore, news media briefing sessions should occur with public relations personnel or at least with personnel that have a comprehensive knowledge of what is occurring. The ability to spread the news of what is occurring alleviates the public's fear and curiosity.

Management activities for disease control are to prevent the further disease spread and reduce risk to wildlife, livestock, and humans, these activities include population and habitat manipulations. Examples of population manipulation include hazing, scare devices, relocation, and other techniques to disperse individuals. Habitat manipulation include burning vegetation, increasing or decreasing the water level, and any other method to diminish use of the area by waterfowl (Webb et al. 2010).

Decontamination should take place on personnel and equipment to prevent risk of disease spread as well as transmission of zoonotic diseases. All operations for decontamination should be outlined within the disease management plan. A plan should be developed at the station-level to

determine where to locate decontamination supplies and equipment, such supplies should be readily available and accessible at a grocery or hardware store. Without proper decontamination the potential for further disease spread is increased (Wobeser 1997).

Finally, there is a need to dispose of deceased wildlife properly. These steps will be disease specific; however, predetermined locations for disposal methods should be established. Examples include the location of digging a pit to bury dead wildlife, developing an improvised burn pit, or establishing composting facilities. Alternatively, prepositioned mobile incinerators may be equipment outlined in the state disease management plan.

Surveillance

Investigations following suspension of the disease control operations are essential to ensure the event does not reoccur. Outlining these steps also allow for further information to be gathered. Surveillance of the infected area should continue for 10 to 30 days after operations have ceased. Investigations should outline the collection of data to answer questions previously recommended in the planning section. These investigations could assist in the understanding of causes, what sustained the disease, exposure patterns, and disease reservoirs (Friend and Franson 1999).

Analyses

The final section of the disease management plan should be a description of how analyses of collected data should proceed. This portion ensures that the state and station learn from the disease event and response. Every event should provide a learning experience to ensure success in future operations. This involves evaluating operations, success, problems, and what can be done differently, also known as an after-action review. This portion also can outline where all the

information and data should be stored for future analysis. Within the roles and responsibilities, there should be a person in charge of maintaining the database where these data are stored.

Conclusion

The three main parameters to explain the complex relationships effecting disease outbreaks are environmental, species, and individuals (Smith and Higgins 1990, Friend and Franson 1999, Rocke and Samuel 1999). These parameters are like ingredients to baking bread; however, the recipe is unknown and can have a range of variability. With the development of surveillance and monitoring, future research could bridge the gap of understanding and assist in the understanding of how, when, and why diseases occur. To assist in the data collection for developing such understanding, state- and station-level disease management plans are proposed to be proactive and reactive to disease events. At the conclusion of such events, a disease management plan allows for the data collection to be consolidated at a centralized location for collective analysis.

As the world climate continues to change, the risk of disease continues to increase (Pounds et al. 2006, Patz et al. 2008, Altizer et al. 2011, Van Hemert et al. 2014); states and wildlife areas need to be prepared to combat and eliminate the risk of disease outbreaks and transmission. The United States Department of Agriculture, Animal and Plant Health Inspection Service Biosecurity for bird's website noted that 50,400,000 birds were affected by the Highly Pathogenic Avian Influenza (HPAI) outbreak in 2015, which decimated 211 commercial flocks within the United States. This disease is feared to have been spread through migratory birds (Chapter 1). Many avian diseases are feared to be spread through the manifestation of poor wetland conditions (Friend and Franson 1999); however, factors contributing to these disease outbreaks are not well known. As proposed, monitoring and development of a disease

management plan at a state and station level could assist investigators in determining the effects and proposing new ideas to combat disease outbreaks.

In Chapter 2, I pointed out that the increasing population of snow geese within Kansas has shown an increase in contributing disease related outbreaks. These outbreaks result in the loss of thousands of waterfowl species migrating at the time of the outbreaks, as well as the high probability of further spread to other areas. The development of the disease management plan should alleviate delayed responses and actions taken by the station and state personnel. Outlining clearly defined roles and responsibilities, allow for a quick response and resolve to disease outbreaks that could potentially effect larger numbers of migrating birds, endangered species, and public health (Chapter 1).

Literature Cited

Altizer, S., R. Bartel, and B. A. Han. 2011. Animal migration and infectious disease risk. science 331:296-302.

Arnall, L., and I. F. Keymer. 1975. Bird diseases. An introduction to clinical diagnosis and treatment of diseases in birds other than poultry. Bailliere Tindall, London, England.

Arzel C., J. Elmberg, and M. Guillemain. 2006. Ecology of spring-migrating Anatidae: a review. Journal of Ornithology. 147:167-184.

Beer, J. V. 1988. Diseases of gamebirds and wildfowl. The Game Conservancy. Fondingbridge, Hampshire, England.

Friend, M., and J. C. Franson. 1999. Field manual of wildlife diseases. General field procedures and diseases of birds. Biological Resources Division Information and Technology Report 1999-01, US Department of the Interior and US Geological Survey, Washington, D.C., USA.

Lindenmayer, D. B., and G. E. Likens. 2010. The science and application of ecological monitoring. Biological Conservation 143:1317-1328.

Patz, J. A., S. H. Olson, C. K. Uejio, and H. K. Gibbs. 2008. Disease emergence from global climate and land use change. Medical Clinics of North America 92:1493-1491.

Pounds, J.A., et al. 2006. Widespread amphibian extinctions from epidemic disease driven by global warming. Nature 439:161-167.

Rocke, T. E., and M. D. Samuel. 1999. Water and sediment characteristics associated with avian botulism outbreaks in wetlands. Journal of Wildlife Management 63:1249-1260.

Samuel, M. D., R. G. Botzler, and G. A. Wobeser. 2007. Avian cholera. Pages 239-269 *in* N. J. Thomas, D. B. Hunter and C. T. Atkinson, editors. Infectious diseases of wild birds. Blackwell Publishing Professional, Ames, Iowa, USA.

Smith, B. J., K. F. Higgins, and W. L. Tucker. 1990. Precipitation, waterfowl densities and mycotoxins: their potential effect on avian cholera epizootics in the Nebraska Rainwater Basin Area. Transactions of the North American Wildlife and Natural Resources Conference 55:269–282.

Van Hemert, C. R., J. M. Pearce, and C. M. Handel. 2014. Wildlife health in a rapidly changing north: focus on avian disease. Frontiers in Ecology and the Environment 12:548-556.

Webb, E. B., L. M. Smith, M. P. Vrtiska, and T. G. Lagrange. 2010. Effects of local landscape variables on wetland bird habitat use during migration through the rainwater basin. Journal of Wildlife Management 74:109-119.

Wobeser, G. A. 1997. Diseases of Wild Waterfowl. Plenum Press, New York, New York, USA.

Appendix A - Reported Disease Outbreaks in Kansas

Approximate start date (mm/yyyy)	Location	Disease	Species	Estimated number affected
09/1967	Cheyenne Bottoms Wildlife Area	*	• *	15,000
09/1968	Cheyenne Bottoms Wildlife Area Pool 2	Avian Botulism	• Widgeon (*Anas americana*) • Blue-winged Teal (*Anas discors*) • Green-winged Teal (*Anas crecca*) • Northern Pintail (*Anas acuta*) • Gadwall (*Anas strepera*) • Shorebird	12,000
09/1982	Glen Elder Reservoir	Avian Botulism	• American White Pelican *(Pelecanus erythrorhynchos*)	65
05/1983	Webster Reservoir	Avian Cholera	• American Coot (*Fulica americana*)	*
10/1984	Council Grove Reservoir	Renal Coccidiosis	• Double-crested Cormorant (*Phalacrocorax auritus*)	100
12/1984	Brown County, Kansas	Avian Cholera	• Snow Goose (*Chen caerulescens*)	165
12/1986	Marion Reservoir	Renal Coccidiosis	• Double-crested Cormorant (*Phalacrocorax auritus*)	40
10/1989	Kirwin National Wildlife Refuge	Renal Coccidiosis	• Double-crested Cormorant (*Phalacrocorax auritus*)	200
02/1990	Fall River Wildlife Management Area	Avian Cholera	• Canada Goose (*Branta canadensis*)	10
10/1990	Cheyenne Bottoms Wildlife Area Pool 2	Avian Botulism	• Green-winged Teal (*Anas crecca*) • Northern Pintail (*Anas acuta*) • Northern Shoveler (*Anas clypeata*) • Blue-winged Teal (*Anas discors*) • Widgeon (*Anas americana*) • Mallard (*Anas platyrhynchos*) • Gadwall (*Anas strepera*) • Ruddy Duck (*Oxyura jamaicensis*) • Redhead (*Aythya americana*) • Lesser Scaup (*Aythya affinis*) • Canvasback (*Aythya valisineria*) • Ring-necked Duck (*Aythya collaris*) • Cinnamon Teal (*Anas cyanoptera*) • Coot (*Fulica americana*) • Pelican *(Pelecanus erythrorhynchos*) • Double-crested Cormorant (*Phalacrocorax auritus*) • Sandhill Crane (*Antigone canadensis*) • Snowy Egret (*Egretta thula*) • Little Blue Heron (*Egretta caerulea*) • White-faced Ibis (*Plegadis chihi*) • Blackbird • Gull • Sandpiper • Unidentified	4,237
11/1991	Elwood, Kansas	Aspergillosis	• Snow Goose (*Chen caerulescens*)	27
03/1992	Quivira National Wildlife Refuge	Aflatoxicosis	• Mallard (*Anas platyrhynchos*)	5

Approximate start date (mm/yyyy)	Location	Disease	Species	Estimated number affected
08/1993	Cheyenne Bottoms Wildlife Area	Avian Botulism	• Green-winged Teal (*Anas crecca*) • Mallard (*Anas platyrhynchos*) • Blue-winged Teal (*Anas discors*) • Northern Shoveler (*Anas clypeata*) • Northern Pintail (*Anas acuta*) • Ruddy Duck (*Oxyura jamaicensis*) • Redhead (*Aythya americana*) • Wood Duck (*Aix sponsa*) • Gadwall (*Anas strepera*) • Widgeon (*Anas americana*) • Coot (*Fulica americana*) • Grebe • Rail • Heron • Gull • Shorebird • Egret • Unidentified	2,035
09/1994	Cheyenne Bottoms Wildlife Area Pool 1A	Avian Botulism	• Green-winged Teal (*Anas crecca*) • Blue-winged Teal (*Anas discors*) • Northern Shoveler (*Anas clypeata*) • Mallard (*Anas platyrhynchos*) • Ruddy Duck (*Oxyura jamaicensis*) • Northern Pintail (*Anas acuta*) • Gadwall (*Anas strepera*) • Widgeon (*Anas americana*) • Cinnamon Teal (*Anas cyanoptera*) • Pelican *(Pelecanus erythrorhynchos*) • Yellowlegs (*Tringa melanoleuca*) • White-faced Ibis (*Plegadis chihi*) • Pectoral Sandpiper (*Calidris melanotos*) • Avocet (*Recurvirostra americana*) • Least Sandpiper (*Calidris minutilla*) • Common Snipe (*Gallinago delicate*) • Black-necked Stilt (*Himantopus mexicanus*) • Coot (*Fulica americana*) • Egret • Gull • Grebe • Unidentified	604
08/1995	Cheyenne Bottoms Wildlife Area Pool 1A	Avian Botulism	• Mallard (*Anas platyrhynchos*) • Redhead (*Aythya americana*) • Northern Shoveler (*Anas clypeata*) • Blue-winged Teal (*Anas discors*) • Wood Duck (*Aix sponsa*) • Ruddy Duck (*Oxyura jamaicensis*) • Gadwall (*Anas strepera*) • Green-winged Teal (*Anas crecca*) • Mottled Duck (*Anas fulvigula*) • Coot (*Fulica americana*) • Egret • Heron • Rail • Shorebird • Unidentified	259

Approximate start date (mm/yyyy)	Location	Disease	Species	Estimated number affected
03/1998	• Lovewell Reservoir • Jamestown Wildlife Area • Rock Pond City Lake	Avian Cholera	• Mallard (*Anas platyrhynchos*) • Northern Pintail (*Anas acuta*) • Blue-winged Teal (*Anas discors*) • Green-winged Teal (*Anas crecca*) • Widgeon (*Anas americana*) • Northern Shoveler (*Anas clypeata*) • Gadwall (*Anas strepera*) • Redhead (*Aythya americana*) • Canvasback (*Aythya valisineria*) • Scaup (*Aythya affinis*) • Ruddy Duck (*Oxyura jamaicensis*) • Bufflehead (*Bucephala albeola*) • Ring-necked Duck (*Aythya collaris*) • Goldeneye (*Bucephala clangula*) • Canada Goose (*Branta canadensis*) • White-fronted Goose (*Anser albifrons*) • Snow Goose (*Chen caerulescens*) • Ross's Goose (*Chen rossii*) • Coot (*Fulica americana*) • Unidentified	4,906
07/1998	Cheyenne Bottoms Wildlife Area	Avian Botulism	• Blue-winged Teal (*Anas discors*) • Green-winged Teal (*Anas crecca*) • Cinnamon Teal (*Anas cyanoptera*) • Mallard (*Anas platyrhynchos*) • Northern Pintail (*Anas acuta*) • Northern Shoveler (*Anas clypeata*) • Gadwall (*Anas strepera*) • Widgeon (*Anas americana*) • Wood Duck (*Aix sponsa*) • Redhead (*Aythya americana*) • Ruddy Duck (*Oxyura jamaicensis*) • Scaup (*Aythya affinis*) • Ring-necked Duck (*Aythya collaris*) • Black Duck (*Anas rubripes*) • Canada Goose (*Branta canadensis*) • Pelican *(Pelecanus erythrorhynchos*) • Double-crested Cormorant (*Phalacrocorax auritus*) • Killdeer (*Charadrius vociferus*) • Coot (*Fulica americana*) • Green Heron (*Butorides virescens*) • Black Tern (*Chlidonias niger*) • Avocet (*Recurvirostra americana*) • Teal • Egret • Gull • Heron • Ibis • Stilt • Rail • Grebe • Unidentified	2,304

Approximate start date (mm/yyyy)	Location	Disease	Species	Estimated number affected
08/2000	Cheyenne Bottoms Wildlife Area Pool 4B	Avian Botulism	• Mallard (*Anas platyrhynchos*) • Blue-winged Teal (*Anas discors*) • Redhead (*Aythya americana*) • Northern Pintail (*Anas acuta*) • Wood Duck (*Aix sponsa*) • Green-winged Teal (*Anas crecca*) • Widgeon (*Anas americana*) • Northern Shoveler (*Anas clypeata*) • Gadwall (*Anas strepera*) • Ruddy Duck (*Oxyura jamaicensis*) • Scaup (*Aythya affinis*) • Coot (*Fulica americana*) • Pelican *(Pelecanus erythrorhynchos*) • Grackle (*Quiscalus quiscula*) • Grebe • Shorebird • Gull • Heron • Egret • Unidentified	432
07/2003	Quivira National Wildlife Refuge	West Nile	• American White Pelican *(Pelecanus erythrorhynchos*)	36
06/2005	Lee Richardson Zoo	Avian Botulism	• Mallard (*Anas platyrhynchos*)	75
03/2006	Quivira National Wildlife Refuge	Mycotoxicosis	• Sandhill Crane (*Antigone canadensis*)	200
09/2007	Cheyenne Bottoms Wildlife Area	Avian Botulism	• Northern Pintail (*Anas acuta*) • Green-winged Teal (*Anas crecca*)	6
03/2008	McKinney Lake	Avian Cholera	• Snow Goose (*Chen caerulescens*)	550
12/2008	Dundee, Kansas	Avian Cholera	• White-fronted Goose (*Anser albifrons*)	243
11/2010	Quivira National Wildlife Refuge	Avian Cholera	• Snow Goose (*Chen caerulescens*)	6
11/2011	Cheyenne Bottoms Wildlife Area	Aspergillosis	• Ross’s Goose (*Chen rossii*)	1
12/2011	Cheyenne Bottoms Wildlife Area Pool 1A	Avian Cholera	• Ross’s Goose (*Chen rossii*)	50
12/2011	Cheyenne Bottoms Wildlife Area	Avian Cholera	• Snow Goose (*Chen caerulescens*) • Greater White-fronted Goose (*Anser albifrons*)	50
12/2011	Cheyenne Bottoms Wildlife Area	Avian Cholera	• Greater White-fronted Goose (*Anser albifrons*)	50
12/2011	Cheyenne Bottoms Wildlife Area	Aspergillosis	• Ross’s Goose (*Chen rossii*)	50
01/2014	Coldwater City Lake	Avian Cholera	• Mallard (*Anas platyrhynchos*) • Snow Goose (*Chen caerulescens*)	*
02/2014	Lovewell Reservoir	Avian Cholera	• Mallard (*Anas platyrhynchos*) • Snow Goose (*Chen caerulescens*)	3,000

* - Indicates no data reported.

Appendix B - Disease Management Plan

I. Introduction

A. Purpose description

B. Area description, features and general information

II. Biological data

A. Common species to area

B. Movement and patterns

C. Weather patterns

D. Past disease history

III. Surveillance of management area

A. Purpose

B. Schedule

IV. Reporting procedures

A. Outline beginning to end of disease event and who should be notified

V. Response

A. Control procedures

1. Individual diseases require unique approaches, advised to seek recommendations in the Field Manual of Wildlife Diseases, Friend and Franson 1999

2. Field investigation and diagnostic laboratory assistance

B. Resources

1. Personnel

a) State

b) Federal

2. Equipment

a) Vehicles

b) Aircraft

c) Heavy Equipment

d) Boats

e) Radio

f) Incinerators

g) Pumps

h) Hazing devices

i) Disposable equipment

3. Decontamination

a) Personnel

b) Equipment

c) How to decontaminate contaminated areas

4. Habitat and population manipulation control

5. Media relations

VI. Suggested attachments

A. Maps

B. List of wildlife and wetland locations

C. List of species of concern

1. Federal and state endangered species

D. List history of disease

E. Contacts

1. Federal

2. State

F. Incident report

G. Investigation report

H. Information distribution list

I. Specific disease procedure

J. List of diagnostic facilities

K. List of media outlets