Introduction

Relationships between wild birds and diseases vary in their ecological complexity and health consequences. Disease is broadly defined as any departure from health resulting in bodily dysfunctions from various causes, including infection, genetic defect, or environmental stress, and characterized by an identifiable group of signs or symptoms. Disease impacts wildlife by reducing the ability of individuals to reproduce or survive (Friend 2006). Some disease systems involve pathogens that cause morbidity or mortality in specific bird species, whereas other disease systems rely on multiple species of apparently healthy birds to spread pathogens. Additionally, birds may play a significant role in the emergence of diseases of human health significance – the spread of highly pathogenic avian influenza of the subtype H5N1 over the past five years across Asia, Russia, the Middle East, Europe, and Africa and the accompanying potential threat of pandemic influenza in humans has highlighted this role.

Disease emergence involving wild birds is invariably the result of a complex network of changes – often human-driven – in the ecology of the bird and/or the pathogen. For example, *Mycoplasma gallisepticum*, historically a poultry pathogen, has caused outbreaks of conjunctivitis (inflammation of the membrane around the eyes) in House Finch across the eastern United States over the past decade. The main mechanism of spread of *M. gallisepticum* involves backyard birdfeeders – particularly tube style feeders in which birds must insert their heads to obtain seed. Infected birds contaminate feeders, which then serve as the source of infection to healthy birds. Thus, supplemental feeding of birds by humans perpetuates the spread of this disease.

While the impact of disease may be significant, only recently have diseases been demonstrated at a theoretical level to have population-level impacts on host species (Anderson and May 1991). In nature, an example of a disease system in which the host is regulated by a pathogen involves a nematode parasite that is responsible for regular population cycles in the British subspecies of Willow Ptarmigan, known as Red Grouse, in Britain (Hudson et al. 1998). When grouse populations harbor heavy worm burdens, negative consequences occur, including reduced breeding success and higher winter mortality, which drive the population cycles. This example also highlights the point that clinical disease – that which is observable based on outward signs of illness – may sometimes result only after the body is challenged with multiple infections, parasites, or stressors, any one of which alone may not cause clinical disease.
Disease is often one among many factors that leads to a species becoming threatened, endangered, or extinct. For example, many species of native Hawaiian birds became extinct during the 20th century due in part to the introduction of the protozoan parasite that causes avian malaria and the mosquito vector that transmits the pathogen to birds (van Riper et al. 1986). Additional threats to these birds include habitat destruction, introduced predators, and other diseases such as avian pox. Presently, the remaining native species on the islands occur predominantly in the high altitude areas that are above the elevational limits of mosquitoes.

Disease Ecology

Disease ecology is the discipline that studies the interactions among hosts, pathogens, and the environment as they relate to the impact of diseases on populations. This population-level approach to understanding disease emergence differs from traditional medical schemes, which aim to treat individual patients. Disease rarely results from isolated interactions, and instead emerges in complex host-pathogen ecological communities, with most diseases involving more than one host species (Collinge and Ray 2006). Changes to the structure of an ecological community may ‘tip the balance’, allowing enzootic disease systems to become epizootics, or allowing new diseases to emerge through the invasion of an exotic pathogen or evolution of a novel pathogen. To comprehend the disease overviews presented below, a brief glossary of disease ecology terms is provided (Table 1).

Table 1: Glossary of key terms used in disease ecology

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Arbovirus</td>
<td>An arthropod-borne virus. Arthropods are invertebrate members of the Phylum Arthropoda, which includes insects, arachnids, and crustaceans.</td>
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<tr>
<td>Enzootic</td>
<td>An infectious disease that is present at a low incidence in a host population at all times</td>
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<tr>
<td>Epizootic</td>
<td>An infectious disease outbreak that results in a large number of diseased animals; the animal equivalent of an epidemic. Epizootics do not persist within a population</td>
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<tr>
<td>Exotic pathogen</td>
<td>A pathogen that is not native to a given area. Exotic pathogens, such as West Nile virus, encounter naïve wildlife hosts when they invade new areas. Naïve hosts often suffer morbidity and mortality because they have not evolved with the pathogen and have no immunity.</td>
</tr>
<tr>
<td>Pathogen</td>
<td>A disease-causing microorganism. Pathogens include bacteria, viruses, fungi, and protozoan parasites</td>
</tr>
<tr>
<td>Parasite</td>
<td>An organism that lives in or on a host. Pathogens are a type of parasite.</td>
</tr>
<tr>
<td>Reservoir host</td>
<td>An animal that becomes infected with a pathogen, maintains the pathogen within its tissues or blood, and serves as a source of infection to other animals. Some hosts may become clinically diseased upon infection with observable signs, whereas others remain clinically normal yet may harbor and shed pathogens.</td>
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<tr>
<td>Serotype</td>
<td>A sub-species level epidemiologic classification of a group of pathogens classified together based on their common surface antigens that induce a characteristic immune response in a host.</td>
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<tr>
<td>Vector</td>
<td>An organism that transmits pathogens from one host to another. Examples of vectors include ticks, mosquitoes, fleas, midges, and kissing bugs.</td>
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<tr>
<td>Zoonoses</td>
<td>Infections or diseases transmissible between animals and humans. Due to the growing human population, which results in wildlife and people sharing more space, zoonoses are increasingly important. Nearly three-quarters of emerging human pathogens are zoonotic (Woolhouse and Gowtage-Sequeria 2005).</td>
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Disease Surveillance and Research
Despite the importance of disease, large gaps remain in our ability to detect the emergence of disease, quantify the prevalence of disease, and determine the impact of disease on wild bird populations. Because a sick bird that exhibits outward signs of weakness may be viewed as easy prey to a predator, birds have evolved to generally hide signs of disease—this is known as the ‘sick bird syndrome’, and it presents problems for individual-based medicine and treatment (Tizard 2008). By definition, disease surveillance is the ongoing, systematic, and continuous collection of health-related data for the analysis, interpretation and action to improve health within populations (Stallknecht 2007). The methods of data collection can be categorized as either passive or active.

Passive Surveillance
The primary technique for the collection of wild bird health-related data includes the detection of sick or dead birds in the wild and submission to a diagnostic facility. Some potential problems with this opportunistic strategy include i) the bird must persist long enough in the environment for someone to detect it, ii) the bird must be collected and delivered in a timely manner, and iii) the diagnostic facility must have the technologies to detect the agent and diagnose the disease. Rarely are all three factors achieved and thus many biases are inherent in passively-acquired data. For example, studies have shown that smaller species of birds have shorter persistence in the environment than larger species (Wobeser and Wobeser 1992, Ward et al. 2006), and urban areas have twice the detection rate of dead birds by people compared to rural areas (Ward et al. 2006). These factors lead to biases, which could inaccurately reflect the magnitude, spatial distribution, and species distribution of an epizootic. Furthermore, it is the general trend that when a new pathogen is introduced to a fully susceptible population, many birds die and are submitted for pathological testing. Subsequently, the number submitted for testing declines, despite the persistence of the pathogen within the population. In the case of emerging zoonoses, the media may initially publicize the epizootic, leading to vigilant humans eager to submit specimens, followed by a decrease in coverage. In Michigan this pattern has been observed when mycoplasmosis and West Nile virus emerged. This may be due to biological factors, in that the new disease reduces the population density of its host so there are fewer birds left to become diseased, or, the remaining individuals may be immune so the mortality rate decreases. Alternately, the decline in submissions may result from the public becoming accustomed to seeing cases on a regular basis and submission of new birds is no longer a priority.

In human medicine, the ‘iceberg phenomenon’ is used to describe the scenario whereby human patients with clinical symptoms of disease represent only a small fraction of the true extent of disease in the population (Lynch et al. 1987). This principle certainly applies to studies of wild bird disease, in which passive surveillance strategies rely on the detection of birds displaying clinical signs, but birds with subclinical levels of disease are not detected. The importance of passive surveillance, however, should not be underestimated. Collecting non-biased data on wild birds can be logistically difficult and too expensive for state and federal budgets, leaving opportunistic data collection as the best alternative. Previous opportunistic surveillance activities have been credited for the discovery of new diseases, such as M. gallisepticum in house finches (Ley et al. 1996) and bovine tuberculosis in white-tailed deer in Michigan (Schmitt et al. 1997).

Active Surveillance
Active surveillance is defined as investigator-driven data collection to meet specific information needs (Stallknecht 2007), which includes the screening of apparently healthy individuals for an agent or antibodies to an agent. The collections typically involve obtaining blood or tissue samples from hunter-harvested birds, birds killed on the roads, bird banding operations, or birds trapped for a specific research purpose. Although some limitations still exist with active surveillance, the strengths are that disease prevalence estimates (number positive as a proportion of the total sampled) are much improved. A recent example of an active surveillance program is the ‘Interagency Strategic Plan for the Early Detection of Highly Pathogenic H5N1 Avian Influenza in Wild Migratory Birds’, designed and implemented by the United States Department of Agriculture. Combined with the Canadian and Mexican efforts, this represents the largest coordinated wildlife disease surveillance program ever implemented (Deliberto et al. 2009).

Disease Research
Both field epidemiology and transmission experiments play an important role in understanding complex avian disease systems. In order to understand disease dynamics in free-ranging birds, population data—such as size, density, age structure, habitat utilization, and home ranges—are necessary to accurately measure pathogen transmission and maintenance across time and space. These intensive studies are also necessary to understand population-level effects of a disease. To complement field studies, controlled experimental studies in a laboratory or semi-field setting are important tools for researchers to study pathogenesis, host and vector competence, and other characteristics of disease. Furthermore, a majority of avian diseases are facilitated by human interactions. Accordingly, research studies that address the growing interface among human, domestic animal, and wild animal health within a shared environment will be increasingly important.
Diseases of Importance to Michigan Birds

In this document, we highlight selected disease systems that impact Michigan birds, or for which Michigan birds play key ecological roles. The criteria for inclusion are diseases that: i) have the potential for local or regional bird population level impacts; ii) are significant diseases in Michigan which require birds for maintenance, or iii) are emerging due in part to the assistance of Michigan birds. The diseases covered here will highlight the historical and current significance of the disease to Michigan birds, and are not intended to comprehensively include all basic disease information such as history, etiology, epizootiology, clinical signs, pathogenesis, pathology, diagnosis, or management. For more information, we refer readers to online resources including the Michigan Department of Natural Resources Wildlife Disease Manual (http://www.michigan.gov/dnr/0,1607,7-153-10370_12150_12220---,00.html) and the National Wildlife Health Center website (http://www.nwhc.usgs.gov).

We cover diverse disease systems, from historic waterfowl diseases (botulism) to newly-emerged pathogens (West Nile virus). We include a disease system that does not negatively impact birds, but for which birds serve as a main mechanism of pathogen spread (Lyme disease), and also a disease not caused by a biological pathogen, but instead by ingestion of lead contamination in the environment. While we do not specifically address nutrition, stress, or co-infection with other pathogens as disease factors in and among themselves, it is likely that their effect on immune function allows or exacerbates other diseases. When available, specific notes on the occurrence of specific diseases within Michigan birds as detected through passive surveillance necropsy records of the Michigan Department of Natural Resources and Environment (MDNR) Wildlife Disease Laboratory over the past two decades are included (Figure 1).

Parasitic Diseases

Trematode: Verminous hemorrhagic ulcerative enteritis

Trematodes (flukes) are parasites with a complex life cycle involving two intermediate hosts (usually snails) and a definitive host (such as birds) where reproduction occurs (Huffman 2008). A wide range of birds are exposed to trematodes but most do not cause disease. One pathogenic species, *Sphaeridiotrema globulus*, is responsible for large-scale bird epizootics, including frequent occurrence in Michigan. The disease caused by *S. globulus* is called Verminous Hemorrhagic Ulcerative Enteritis (VHUE) and primarily occurs in waterfowl (Figure 2). Recurrent epizootics have occurred in the Mississippi River (Herrmann and Sorensen 2009) and the St. Lawrence River (Huffman 2008). Birds that are most susceptible to this disease, resulting in severe blood loss and anemia, are those that consume *S. globulus*-infected snails. In Michigan, the disease is primarily found in swans, with 106 Mute Swans, 2 Trumpeter Swans, and 27 Tundra Swans diagnosed by the state diagnostic lab between 1988 and 2009. The impact of *S. globulus* on wild bird populations is difficult to determine. The exotic species of snail, *Bithynia tentaculata*, is widespread throughout the Great Lakes region and is implicated as the intermediate host responsible for the establishment and maintenance of *S. globulus* in the upper Mississippi River (Herrmann and Sorensen 2009). Although no definitive answers are available, swan VHUE epizootics in Michigan might be limiting Mute Swan populations, and this disease must be considered in the restoration efforts of Trumpeter Swan in Michigan.

Figure 1: Tom Cooley, wildlife biologist with the Michigan Department of Natural Resources and Environment, begins a necropsy on a Common Loon at the Wildlife Disease Laboratory. Photo credit: MDNR Wildlife Disease Laboratory.
Avian Disease

Figure 2: Tundra Swan collected from Michigan with Verminous Hemorrhagic Ulcerative Enteritis displaying clinical signs of hemorrhages in the intestinal tract. The scalpel is pointing to the adult fluke (S. globulus) which are small, spherical, flat, and seed-like in appearance. Photo credit: MDNR Wildlife Disease Laboratory.

Nematode: Proventricular Worm
Nematodes are an incredibly diverse group of worms with 15,000 described parasitic species. Nematodiasis, a condition of infection with nematode worms, is widespread in many orders of birds occurring in Michigan, most of which are non-pathogenic and presumably have little impact on bird populations. Herein, we focus on a group of nematode parasites in the family Acuariidae. In particular, the proventricular worm (gizzard worm), Dispharynx nasuta, is a common parasite of birds in the Galliformes, Columbiformes, and Passeriformes. With an indirect life cycle, D. nasuta uses the sowbug (Procellio scaber) or pillbug (Armadillidium vulgare) as intermediate hosts (Peterson 2004). D. nasuta is considered pathogenic to Ruffed Grouse and has been considered the leading cause of 'grouse disease' in the northeastern U.S. (Ruff and Norton 1997). Ruffed Grouse populations throughout their range are known to experience population cycles (Williams et al. 2004, Zimmerman et al. 2008) and a recent study determined that predation was not contributing to these cycles, but other factors including parasites are likely regulating Ruffed Grouse populations (Zimmerman et al. 2008).

A closely related parasite, Trichostrongylus tenius, is a parasite of Red Grouse in northern England and has been implicated with regulating population cycles. The parasitic nematodes impact Red Grouse by reducing survival of adults and fecundity of females and have been implicated in contributing to Red Grouse population cycles (Hudson et al. 1998, Redpath et al. 2006, New et al. 2009). Regulation in this context implies that the parasite reduces the host population when it is above a certain threshold, and allows the population to increase under other conditions (Peterson 2004). Although these parasites do not jeopardize host population extinction, other abiotic conditions could further lead to population declines.

Although only one Ruffed Grouse has been diagnosed with Proventricular Nematodiasis caused by D. nasuta over the past two decades in Michigan (this case occurred in Oscoda County), we do not have a good estimate of the prevalence of these parasites in the state and their contribution to Michigan grouse population cycles. A passive surveillance program would not likely detect this disease given that these parasites do not usually cause direct mortality. Furthermore, even if a grouse dies in northern Michigan, the chances of it being recovered are slim. In the late 1970’s and early 1980’s, an active surveillance program that utilized hunter-harvested birds was implemented in Michigan, and reimplementation of such a program would provide a better measure of prevalence and the potential for population regulation.
Protozoa: Trichomoniasis

Avian trichomoniasis is caused by *Trichomonas gallinae*, a protozoan parasite that has a cosmopolitan distribution and primarily infects pigeons, doves, and raptors. The primary host is the Rock Pigeon and transmission among doves and pigeons occurs through feeding of young and eating contaminated food and water. Raptors are exposed by consuming infected doves or pigeons. Small outbreaks of this disease are common, and periodic large scale epizootics have occurred in Mourning Doves – the largest on record being in Alabama during 1950-1951 when 50,000-100,000 doves died. In Michigan, 71 doves have been diagnosed with trichomoniasis between 1988 and 2009, most of which were individuals collected between July and October. The impact of trichomoniasis on Michigan bird populations is unknown.

Trichomoniasis has a long history and is possibly the oldest known wildlife disease recorded (Forrester and Foster 2008). Recently, Wolff et al. (2009) discovered fossilized dinosaurs (*Tyrannosaurus rex*) with evidence of trichomoniasis-type infections, similar to the *T. gallinae* in modern birds. Other authors consider trichomoniasis as a possible contributing factor to the extinction of the Passenger Pigeon, although there is little to support this hypothesis (Forrester and Foster 2008).

Infectious Diseases

Bacterial

*Salmonellosis*

Salmonellosis results from infection with bacteria of the genus *Salmonella*, which includes approximately 2,500 serotypes that have been recovered from birds, mammals, and reptiles. Salmonellae are intestinal pathogens with a fecal-oral transmission route, and cross-species transmission is common. While Salmonellae are one of the main causes of human food-borne illness throughout the world, wild birds are rarely linked as the source of infection to humans; rather, wild bird infection usually reflects contamination of the environment by humans or livestock (Cizek et al. 1994). Nonetheless, salmonellosis is a significant public health and veterinary concern, as all members of this genus are potentially pathogenic to humans and animals. Salmonellae have been devastating to the poultry industry, especially before the implementation of pathogen control measures, as it has caused fatal septicemia in domestic flocks referred to as 'fowl typhoid' or 'pullorum disease'. Furthermore, poultry-associated Salmonellae pose a significant zoonotic threat to humans (Antunes et al. 2003). Host adaptation occurs with *Salmonella* pathogens, in which certain bacterial strains are evolved to exist in a long-term carrier state in specific hosts, and will occasionally emerge to cause disease within that host upon circumstances favorable to the pathogen, including periods of compromised immune function in a host (Kingsley and Bäumler 2000). The virulence of such host-adapted strains to non-target hosts is reduced.

Salmonellae can persist in the environment outside of a host’s body – in water, soil, feed, litter, manure, plastics, or other materials – and wild birds most commonly acquire infections from the environment. Among wild birds, the strain most commonly isolated is a serotype of *S. enterica* called typhimurium, which has not historically been considered as host-adapted, but some types appear to be adapting to songbird species that frequent bird feeders (Daoust and Prescott 2007). While most bird infections are sub-clinical, salmonellosis is a cause of sporadic mortality particularly among young birds in large breeding colonies and songbirds around feeders in winters. As is the case for a majority of intestinal pathogens, the duration and concentration of fecal shedding of the organism is influenced by the dose of exposure, host-adaptation of the strain, the composition of the normal microflora of the intestine, and immune function of the host.

In Michigan, salmonellosis has been confirmed in 572 birds over the past two decades, comprised of 20 species across 59 counties. The most commonly infected species include the Common Redpoll and the Pine Siskin, which together account for 65% of documented cases, and House Sparrows and American Goldfinch, which together account for an additional 21% of cases. Infections have been most common in winter at bird feeders, when bird densities are high. Over 92% of the Michigan cases occurred between the months of November and April. Supplemental feeding promotes disease emergence by creating high densities of birds, high concentration of feces, and stress due to social interactions (Daoust and Prescott 2007). The spatial and temporal pattern of disease emergence in Michigan reflects the irruptive movement patterns of the most impacted species. Such irruptions are unpredictable, sudden winter movements of flocks of birds to areas outside their normal range, often driven by a lack of food on the normal wintering grounds. The winter of 1997-1998, for example, was a period of irruptive migration of Common Redpoll, when dense flocks migrated simultaneously into their wintering grounds from their Canadian breeding grounds, leading to high densities of stressed birds at feeders in Michigan which resulted in 138 documented cases of salmonellosis in this species. Similarly, the winter of 2008-2009 was a period of irruptive migration of Pine Siskin, and 29 cases were confirmed in this species over that time period. Those feeding birds can help to reduce transmission through weekly disinfection of feeders using a 10% bleach solution, altering the location of feeders to disperse contamination of the ground, and sweeping away the partially-eaten feed and feces. If diseased birds are noted, feeders should be removed to disperse birds, and all feeding within the neighborhood of diseased birds should cease for at least two weeks or until sick birds are no longer observed.

Lyme disease

Lyme disease is a tick-borne bacterial disease of humans and dogs caused by infection with *Borrelia burgdorferi*. The blacklegged tick is the most important vector in the
eastern United States. While the endemic foci for Lyme disease in the United States has historically been circumscribed to the northeast and upper Midwest, risk is increasing in new geographic areas as the tick and bacterium are expanding in range (Bacon et al. 2007). Because ticks do not move great distances on their own, significant range expansion of the tick is due to movement of the host upon which the tick is blood-feeding within a 3-7 day time period. Ticks become infected when feeding on an infected mammal or bird, and then may subsequently transmit the pathogen to a new host during the next blood meal.

Compared to the other diseases highlighted in this chapter, birds are not known to become sick when infected with the Lyme disease pathogen. Birds are, however, important in the ecology of Lyme disease for two reasons. First, migratory birds travel great distances during short time periods, and this movement may allow for infested birds to transport blacklegged ticks over large areas, expanding the distribution of the tick and correspondent Lyme disease risk (Reed et al. 2003, Ogden et al. 2008, Brinkerhoff et al. 2009); Figure 3). In areas of Lyme disease endemcity, the species of birds that are most heavily parasitized by blacklegged ticks are ground-dwelling birds, including thrushes (Turdidae), brown thrashers (Mimidae), wrens (Troglodytidae), sparrows (Emberizidae) and several species of wood warblers (Parulidae) (Brinkerhoff et al. 2009), as they are more likely to encounter and become infested by ticks. Second, many species of bird are competent reservoir hosts for *B. burgdorferi* as they can maintain this pathogen within their tissues and blood to serve as a source of infection to ticks that feed upon them. Thus, movement of infected birds may represent a mechanism of pathogen introduction to new areas. In Lyme disease-endemic areas, over 70 passerine species and one species of woodpecker have been found to be parasitized by blacklegged ticks with an overall infestation prevalence of 14%.

In Michigan, Lyme disease has historically occurred only in a focal area of the southern Upper Peninsula, as this was the extent of the distribution of the blacklegged tick in the state (Walker et al. 1999). Over the past decade, however, the blacklegged tick has been invading new areas throughout the southern and western Lower Peninsula (Foster 2004, Hamer et al. 2007). Because blacklegged ticks do not currently occupy all suitable habitats in Michigan, it is predicted that this vector will continue to expand in range, and research is underway to elucidate the role of birds in the expansion of Lyme disease risk (Hamer et al. 2010).

![Figure 3: Larval ticks in the ear of a White-throated Sparrow captured at Rose Lake State Wildlife Area, East Lansing, MI. Photo credit: Gabriel Hamer, 2007.](image-url)
Avian Disease

**Mycoplasmosis**

Conjunctivitis – swelling or infection of the membrane of the eyelids – was first reported in wild House Finches in New York in 1994, when birds with crusty eyelids and impaired vision were observed (Ley et al. 1996, Fischer et al. 1997). The pathogen infecting the birds was identified as *Mycoplasma gallisepticum*, and this represented the first time that this pathogen (commonly a cause of respiratory disease in poultry) was isolated from passerines. Within three years, the pathogen spread across the entire eastern range of the House Finch, causing a decline of eastern populations (Hochachka and Dhondt 2000), and the disease remains enzootic today. More recently in 2002, the disease was first observed among the native western House Finch population (Duckworth et al. 2003), and has been confirmed in five additional avian species: American Goldfinch, Purple Finch, Tufted Titmouse, Pine Grosbeak, and Evening Grosbeak (Fischer et al. 1997, Hartup et al. 2000, Luttrell et al. 2001, Mikaelian et al. 2001). House Finch native to the western United States were captured and sold in pet shops in the eastern states in the mid-1900s, and released into the wild shortly thereafter. The eastern House Finch population expanded dramatically from the original small number of released birds, and currently ranges across the entire eastern and Midwestern states and southern Canada.

While there are biological reasons that likely contributed to the rapid spread of this disease in eastern House Finch populations, including reduced genetic diversity from a population bottleneck and increased population density in comparison to the western populations, the spread of this disease is enhanced through the use of bird feeders. Supplemental bird feeding provides opportunities for increased contact between infected and susceptible birds with the feeder serving as a reservoir of infection. Additionally, supplemental feeding may prolong the life of diseased birds that otherwise would not have found food, allowing them to be infectious for a longer period of time. Since its emergence, mycoplasmosis has been tracked mainly through volunteer-based passive surveillance (a form of ‘citizen science’) in which citizens who regularly feed birds were able to report sightings of both healthy and sick birds at their feeders to the Cornell Laboratory of Ornithology.

In Michigan, mycoplasmosis was first diagnosed in House Finches in 1995, with numerous reports in the first few years, and sporadic reports thereafter (Figure 4). That confirmed cases are not as numerous in recent years is likely an artifact of the public becoming accustomed to observing diseased finches as well as increasing immunity within the population. In addition to House Finch, a Downy Woodpecker in the state was confirmed to be infected with *M. gallisepticum*. As with prevention of Salmonellosis, transmission of mycoplasmosis can be reduced through weekly disinfection of feeders using a 10% bleach solution.

Figure 4: House Finch captured in Lansing, MI, with conjunctivitis. Photo credit: Sarah Hamer, 2006
Duck plague (i.e. duck virus enteritis) is a highly contagious disease of waterfowl caused by a herpesvirus (Friend 1999b). The virus is distributed globally with irregular epizootics occurring in North America since the late-1960s. The virus has been documented in a wide range of waterfowl species, but the most susceptible species include Blue-winged Teal, Redhead, Wood Duck, and Gadwall (Hansen and Gough 2007). Transmission is horizontal with host-to-host contact due to virus shedding and contamination of the environment. The largest epizootic in the United States occurred in 1973 at Lake Andes National Wildlife Refuge in South Dakota when 40,000 Mallards died. This outbreak lead to the realization that wildlife disease could actually cause population-level impacts, providing the motivation for the establishment of the United States Geological Survey's National Wildlife Health Center in 1975. The last known cases of duck plague in Michigan occurred in Muscovy and Mallard ducks in 1979.

West Nile virus (WNV) first arrived in North America in 1999 and rapidly spread from New York City to much of North and South America in one decade, and has become one of the most widely distributed arboviruses in the world (Weaver and Reisen 2009). The viral agent is maintained by a mosquito-bird cycle, primarily involving *Culex* spp. mosquitoes which transmit WNV to a variety of avian hosts, and occasionally humans, horses, or other mammals which serve as incidental hosts (Kilpatrick et al. 2007); (Figure 5). By a process known as amplification, this mosquito-bird cycle typically increases the virus to peak levels in late summer (Hamer et al. 2008). In the last decade, approximately one million people in the United States are estimated to have been exposed to WNV, resulting in nearly 30,000 human clinical cases, and just over 1,000 fatalities (Gyure 2009).

Figure 5: West Nile virus transmission cycle. Figure credit: Gabriel Hamer.
Experimental studies have demonstrated that birds vary in their competence as a host, which is measured by susceptibility to the virus, the amount of virus growth in the bird, and the duration that the bird is considered infectious to other mosquitoes (Kilpatrick et al. 2007). Several studies have used different approaches to determine the impact of WNV on wild bird populations. Yaremch joint et al. (2004) used a radio-telemetry study to discover 68% mortality of American Crows in 2002 in Central Illinois, when WNV first arrived in the Midwest. Several studies have utilized the U.S. Geological Survey’s North American Breeding Bird Survey (BBS) to detect declining trends in bird populations due to WNV (LaDeau et al. 2007, Wheeler et al. 2009). Consistently, birds in the Corvidae family (crows and jays) are highly susceptible to WNV and have significant declines in bird populations, presumably due to WNV. For example, the Yellow-billed Magpie, endemic to California, has declined in abundance since the virus was first detected in the state in 2005, and population-level declines are evident in local surveillance (Smallwood and Nakamoto 2009), Christmas Bird Counts, and BBS data (Crosbie et al. 2008).

Due to their high death rates and relative absence in urban environments, American Crows are not the most important species responsible for amplification and maintenance of WNV. Scientific evidence suggests that the American Robin is the most important host for WNV in the eastern half of the United States (Hamer et al. 2009). The risk of WNV to bird populations varies spatially, but the species with apparent population-level declines in some parts of the United States include American Crow, Blue Jay, American Robin, Eastern Bluebird, House Wren, Tufted Titmouse, House Finch, and Chickadee species (LaDeau et al. 2007, Wheeler et al. 2009). Like many species, raptors in general exhibit both susceptibility and tolerance/resistance to the virus, and any local population-level impacts that may have occurred have not manifested in Breeding Bird Survey data. Local-scale surveys of the American Kestrel, for example, show both reduced abundance in nest boxes and high levels of antibodies in surviving birds after the virus passed through (Medica et al. 2007). Due to the patchy distribution of affected birds, standardized bird counts in the non-breeding season, such as the Christmas Bird Count, are not often sensitive enough to detect impacts of the virus on subpopulations because the counts are of accumulations of birds that arrived from many different breeding areas in which only a subset may have experienced WNV transmission.

The first WNV-positive bird in Michigan was discovered in 2002, and since then 151 birds have tested positive for WNV at the MDNR Wildlife Disease Laboratory. Although most of these birds have been Corvids and raptors, this passive surveillance data is difficult to use for assessment of population-level effects, especially since national and state budgets have reduced the funding to test all dead birds arriving in diagnostic labs. Based on BBS trends in the state of Michigan, a few populations of birds appeared to have some level of decline since WNV arrived in 2002, such as American Crows, Blue Jays, and Tufted Titmouse, although it is difficult to determine cause and effect. The most apparent population decline after 2002 occurs in American Crows, but this decline has been followed by a recent increase in 2009, which may suggest a rebounding population. Recent studies have identified increasing antibody prevalence in American Crow populations (Wilcox et al. 2007, Reed et al. 2009), supporting the hypothesis that acquired immunity among some populations will decrease the mortality rate due to WNV. Now that WNV is established in the state, we expect continued transmission from year to year with variable levels of intensity, but bird populations in the state do not appear to be in jeopardy.

**Avian Pox**

Avian pox is caused by a viral pathogen in the poxvirus group and infects a wide range of bird species worldwide (Hansen 1999). Birds may be exposed to the pathogen horizontally, including direct contact with other infected birds or indirectly after contact with an infected surface such as a bird feeder. Additionally, mosquitoes and other vectors can mechanically transmit the virus from host to host. Once infected, birds develop lesions on the skin of toes, legs, heads, or mouth, which sometimes lead to death.

The majority of wild bird avian pox infections are mild and self-limiting (van Riper and Forrester 2007). Some epizootics have resulted in extremely high mortality rates, especially on islands with naive bird populations, such as Hawaii. In Michigan, 79 birds, over half of which were Wild Turkey, have been diagnosed with avian pox over the past two decades. Bird banders in Michigan commonly detect birds with lesions consistent with pox virus (Figure 6).

**Avian influenza**

Avian influenza viruses (AIVs) encompass a diverse group of RNA viruses of the family Orthomyxoviridae. AIVs infect domestic poultry and wild aquatic birds, and are typically of low pathogenicity (LP). LPAIVs are transmitted naturally among waterfowl, gulls, and shorebirds and no disease results as these birds are the natural reservoirs of LPAIV worldwide (Stallknecht et al. 2007). These strains may cause respiratory disease or reductions in egg production in domestic poultry. AIVs are transmitted among wild birds through a fecal-oral route when cloacal shedding of the virus occurs, as well as through exposure to contaminated water. High pathogenicity (HP) forms of the virus are named for their ability to cause disease in domestic chickens, and have historically caused epizootics referred to as ‘fowl plague’ with severe systemic infections and high mortality rates. Prior to 2002, the scientific consensus was that while HPAIVs posed significant risks to poultry, there was little or no risk to wild bird populations (Stallknecht et al. 2007). Beginning in 2002 in Asia however, wild and captive exotic birds began displaying signs of morbidity and mortality from infection with HPAIV of the H5N1 lineage (named for
the types of hemagglutinin and neuraminidase proteins on the surface of the virus). This HPAI H5N1 virus was responsible for epizootics in both poultry and wild birds as it spread throughout Asia in the subsequent years, and reached central Europe and Africa by mid-2006 (Stallknecht et al. 2007). Globally, H5N1 virus has been reported in 104 species of wild and domestic birds, with significant mortality loss reported for some species (USGS 2006). Mallard, Northern Pintail, Blue-winged Teal, Redhead, Eurasian Wigeon, and Gadwall appear to be able to become infected with and shed HPAIV without becoming diseased (Munster et al. 2005; Keawcharoen et al. 2008; Brown et al. 2006). While the mechanism of global spread of this virus is unknown, it is likely that both infected migratory birds as well as trade of infected poultry have contributed (Stallknecht et al. 2007).

In general, AIVs are non-infectious to humans due to species barriers to transmission that relate to virus-receptor specificity (Kuiken et al. 2006). There is however, a potential for viral reassortment and mutation that would facilitate human infection and therefore risk of a pandemic (Peiris et al. 2007). Although rare considering the number of infected birds, bird-to-human transmission of the Asian HPAI H5N1 virus has been demonstrated among poultry workers in Southeast Asia. Furthermore, limited, direct human-to-human transmission from poultry workers to their immediate family members has also been documented, although the infrequency with which this has occurred and the inability to rule out common exposure to a contaminated environment do not suggest that the virus has gained the ability to be transmitted in the absence of the avian reservoir (Ungchusak et al. 2005).

In 2006, the US Interagency Early Detection System for Highly Pathogenic H5N1 Avian Influenza in Wild Migratory Birds was implemented by USDA Wildlife Services in response to the spread of HPAI H5N1 in Europe and Asia (Deliberto et al. 2009, Pedersen et al. 2010). This effort, in conjunction with the US Department of the Interior, all 50 state fish and wildlife agencies, several tribal wildlife agencies, as well as Canadian and Mexican collaborations, constituted the largest wildlife disease surveillance program ever implemented. The aim was not only to detect potential HPAIV in wild birds, but also to provide insight on the variable strains of LPAIV in birds. Using standardized methodology, over 261,000 wild bird samples and 101,000 fecal samples were collected in the US, using a variety of sampling strategies from April 2006 to March 2009 (Deliberto et al. 2009). A majority of the samples were collected from hunter-harvested birds; also sampled were live, apparently healthy birds, sentinel birds (caged birds strategically placed in areas and repeatedly sampled to detect infection), sick or dead birds involved in morbidity or mortality events, and spent fecal samples from the ground. Over 86% of samples were collected from dabbling ducks, geese, swans, and shorebirds due to their importance as reservoir hosts for the virus (Deliberto et al. 2009). In this effort, Michigan was classified as a ‘Level One’ state, in which the highest level of sampling was conducted based on known distribution of AIVs, species-specific migratory pathways, geographic size and location, wetland habitat, juxtaposition with shorelines, band recovery data, and input from waterfowl biologists. Results provided evidence, with 99.9% confidence, that the US population of wild birds was free of HPAIV given the expected minimum prevalence of 0.001% (Deliberto et al. 2009). Across all states, the apparent prevalence of LPAIV in birds ranged from 9.7-11%, with a majority of virus subtypes detected in ducks. The most common subtype isolated was H5N2 and this type was most commonly associated with Mallards which accounted for the largest percentage of sampled birds, as well as the largest proportion of total virus detections (Pedersen et al. 2010).
Avian Disease

While this viral strain is not currently highly pathogenic in poultry, mutations of the LP virus into a HP virus are possible (Fouchier et al. 2007). In Michigan, at least five unique LPAIVs have been detected through this effort (Pedersen et al. 2010). The regular contact and interaction between poultry, wild birds, and humans underscores the importance of understanding diseases that may emerge at this interface and the continued need for well-coordinated and standardized surveillance.

Mycotic

Aspergillosis

Aspergillosis is a respiratory disease of birds and mammals, including humans, caused by infection by a fungus of the genus Aspergillus. Aspergillus species have a near worldwide distribution, where they are saprophytic – that is, they live upon dead or dying organic matter – and they thrive in warm and moist conditions which favor spore formation, including crop litter in cut fields, grass along rivers, moist grain, hay, or silage, damp straw or rotting plant or animal materials (Friend 1999a). Thus, birds that are most at risk for becoming infected include species exposed to these environmental risk factors, such as wild aquatic and scavenger species, and birds that feed in agricultural fields, as well as domestic birds that are fed tainted feed. Wild birds that are transported or held in captivity have also developed disease (Converse 2007). The most commonly documented species causing disease in wild birds is A. fumigatus. Birds become infected upon inhalation of fungal spores in the air near a contaminated environmental source; the fungus is not transmissible directly from bird-to-bird or from bird-to-mammal (Merton 1964). The inhaled spores then lodge within the lungs and the vegetative form of the fungus (the mycelium) produces branching hyphae that infiltrate the lungs and other tissues, resulting in pneumonia and other disease manifestations. Some mycelia produce mycotoxins which can further damage blood cells, the neurological system, or other organs (Moss 2002). Aspergillosis may manifest acutely or chronically. In the acute form, birds otherwise in good body condition die rapidly after pneumonia develops, and epizootics may result when a large number of birds are exposed to a common contaminated environmental source. Conversely, in the chronic form, some birds that are exposed to the fungus may live with the infection for a long period of time before other concurrent disease, poor nutrition, or other forms of stress compromise immune function, allowing the fungus to flourish.

Over the past two decades in Michigan, aspergillosis has been identified as the cause of death in over 200 birds, with the Common Loon, Canada Goose, Mallard, Red-tailed Hawk, and Ring-billed Gull responsible for 55% of passive surveillance cases. While the ubiquitous distribution of pathogenic Aspergillus species in the environment suggests that wild birds will continuously be exposed, preventative measures that can be taken to reduce the risk of wild bird infection in an agricultural setting include preventing wild bird access to spoiled animal feeds as well as plowing fields of waste grain before winter if grains have become molded from excess rain or snow, and keeping bird feeders and nest boxes free of moldy grain or damp materials (Converse 2007).

Toxins, Poisons, and Pollutants

Heavy metals: Lead poisoning

Lead toxicosis is the most commonly reported waterfowl disease caused by heavy metals and has been documented in free-ranging birds since the 1800s (Sanderson and Bellrose 1986, Friend 1999c). The major source of exposure of wild birds is through the ingestion of lead shot and lead fragments from hunting activities and other lead materials such as fishing sinkers and jig heads (Figure 7). The lead enters the gizzard and is dissolved and absorbed into body tissues, causing paralysis and eventual death. Raptors can be exposed by bioaccumulation when they consume carcasses containing lead. The magnitude of the problem was slowly realized through research in the 1970s and 1980s when an estimated 1.6 to 2.4 million waterfowl died each year from lead poisoning caused by lead shot ingestion. As a result, federal legislation banned the use of lead shot for waterfowl hunting in 1991 in the United States and 1997 in Canada (Degernes 2008). In addition, non-toxic shot is also required in some states for hunting other game birds, such as Mourning Doves. In 2007, the California state government passed the Ridley-Tree Condor Preservation Act which requires the use of lead-free bullets for big game hunting in the range of the California Condor.

Although hunters have shifted to non-toxic shot such as steel, tungsten, or bismuth when hunting waterfowl, birds are still dying of lead poisoning in Michigan and elsewhere. Over the past two decades, the MDNR Wildlife Disease Laboratory has diagnosed 249 birds with lead poisoning (mostly waterfowl, loons, and Bald Eagles), and the annual numbers have not been declining since 1991 when the ban on lead shot was implemented. One reason for this could be the long-term persistence of lead shot in wetlands, which will likely require over 25 years to dissipate (Flint and Schamber 2010). Although the reduction of the use of lead shot has shown signs of reducing lead toxicosis nationwide, in recent years, reduction in the availability in nature of other sources of lead, such as fishing sinkers, has received more attention, resulting in bans of lead fishing tackle and voluntary programs that encourage the use of non-toxic alternatives (Friend 1999c).
Biological toxins: Botulism (Type C and E)
Globally, botulism is an extremely important avian disease and in North America it has been responsible for over a million bird deaths in a single year (Rocke and Bollinger 2007). This disease of both animals and humans is caused by neurotoxins produced by the bacteria, *Clostridium botulinum*. There are at least seven different neurotoxins produced by different strains of *C. botulinum*, the most common being type C and type E. Both types result in birds affected with varying degrees of paralysis, hence the common name 'limberneck' for the disease.

Type C botulism has been known as an important disease in waterfowl and other wetland birds, particularly in the western US, since biologists first took note of it in the early twentieth century. The primary transmission cycle for this disease is described as a carcass-maggot cycle (Rocke and Friend 1999). Briefly, decaying organic matter in stagnant water provides the ideal anaerobic and hot environment conducive to toxin production by the growth and multiplication of *C. botulinum*. Invertebrates ingesting the carcass concentrate the toxins and are consumed by wetland birds. Once more birds start to die, they contribute to *C. botulinum* growth and perpetuate the cycle. This cycle typically occurs in late summer when the lack of rain and hot temperature creates stagnant water environments. In Michigan, these outbreaks have traditionally occurred in wetlands near the Detroit River and Saginaw Bay. Type C botulism has been implicated for its large scale population level impacts in the western US; in California in 1996, 8,500 American White Pelicans were killed, an estimated 15-20% of the western metapopulation (Rocke et al. 2005). Type C botulism outbreaks in Michigan have been less frequent, less dramatic, and have less of an impact on avian populations. However, the impact on threatened or endangered birds such as the Piping Plover and the potential for outbreaks in zoo exhibits warrants wetland management actions to reduce late-summer stagnant water conditions. These management actions are encouraged at many state and federally-owned wetlands and at wetlands outside the state where Michigan breeding birds migrate or winter.

Type E botulism occurs primarily in fish and fish-eating birds of the Great Lakes Region and has resulted in extensive epizootics occurring more frequently in recent years (Riley et al. 2008). Much less is known about the growth conditions conducive to *C. botulinum* growth and toxin production in the Great Lakes and an interdisciplinary
team of researchers are investigating the transmission ecology. One hypothesis for the mechanisms leading to epizootics involves two invasive exotic species of mussels, quagga mussel (*Dreissena bugensis*) and zebra mussel (*Dreissena polymorpha*), that filter-feed and improve water clarity allowing light to penetrate deeper into the lake. The increased light improves algal growth, creating an environment with low oxygen levels as the algae proliferates and decomposes on the lake bottom. Once the correct anoxic environment for *C. botulinum* is established, the toxins accumulate in mussels and fish, particularly the invasive round gobie (*Neogobius melanostomus*), which are then consumed by fish-eating susceptible avian species. The inconsistent epizootics of large numbers of birds washing ashore have been associated with local environmental and limnological triggers (Perez-Fuentetaja et al. 2006). Although much remains unknown about this complex disease system, much attention has been focused on it in recent years and a task force consisting of state, federal, and provincial agencies in the Great Lakes Region met in Michigan in 2008 to synthesize data and direct management strategies.

From 1999 to 2008, an estimated 70,000 avian mortalities were reported in the Great Lakes due to type E botulism, typically occurring in October and November. The majority of these birds were gulls, loons, waterfowl, and shorebirds (Figure 8). The most likely species with a population level impact due to type E botulism include: Common Loon, Herring Gull, Great Black-backed Gull, and Piping Plover. Of these, Common Loon have potentially been impacted at the population level during the fall epizootics on Lakes Michigan, Huron, and Erie. Events of loon epizootics are often followed by drops in the BBS counts the following year, suggesting population level impacts, but the populations also appear to rebound quite readily (Figure 9). The loons that are being exposed to botulism toxins (presumably through fish) are staging on the Great Lakes during migration. Of the 36 Common Loons collected in Lake Michigan, processed by the MDNR Wildlife Disease Laboratory and diagnosed with type E botulism, all but one were adult birds. This age bias toward adults was also observed from a loon epizootic in Lake Huron in 1999, where 100% of the dead birds were adults. The disproportional impact on adult birds could have a greater impact on subsequent recruitment. The recent drop in populations of loons in Michigan, Wisconsin, and Minnesota since 2007 might be related to the large type E botulism epizootics in Lake Michigan in 2006 and 2007. Fortunately, little botulism activity occurred in 2008. Many government agencies in the Great Lakes Region are carefully monitoring the population impacts of avian botulism so more knowledge should be forthcoming.

![Figure 8: Dead loons, grebes, and ducks along Lake Michigan's shoreline during the 2007 epizootic of Botulism type E. Photo credit: MDNR Wildlife Disease Laboratory.](image-url)
Conclusion
Rates of disease emergence will continue to increase given globalization, species invasions, climate change and its influence on species distributions, habitat destruction, and other factors. A common theme preceding disease emergence is the increasing interface among humans, wild animals, and domestic animals – at this interface, pathogens are shared among species. The role of wild birds in pathogen maintenance and spread of disease has been of increasing scientific interest over the past decade given the emergence of West Nile virus and avian influenza. Elucidating the migratory patterns of wild birds will assist in understanding the spread of diseases of importance not only for wild bird health, but also for the health of domestic birds, wild mammals, livestock, pets, and humans. Despite decades of scientific research, wild bird populations are still plagued by historically important diseases, including botulism and trichomoniasis. Importantly, disease in wild birds is often the result of multiple factors with a cumulative effect of decreasing bird health, including poor nutrition, breeding, migratory, or territorial stress, and chronic, subclinical infection with pathogens. As we continue to try to protect the health of wild populations, surveillance of birds for disease presence will be of continued importance. Michigan’s bird enthusiasts can play an important role by staying informed about the important wild bird diseases in our state, regularly sanitizing bird feeders, and being vigilant for the abnormal occurrence of sick or dead birds that can be reported to the Michigan Department of Natural Resources, Wildlife Disease Laboratory online (http://www.michigandnr.com/diseasedwildliferreporting/disease_obsreport.asp).

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