Zoonotic Diseases of Common Pet Birds: Psittacine, Passerine, and Columbiform Species

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• Pet • Bird • Zoonoses • Psittacine • Avian • Parrot
• Passerine • Columbiform

Psittacine, passerine, and columbiform birds are among the most popular groups of avian species kept as pets. Fortunately, zoonotic transmission of disease from these species is uncommon, but there are some recognized dangers. Most notably, *Chlamydophila psittaci* can be transmitted from pet birds to humans. *Salmonella* spp, although more commonly a food-borne zoonotic agent, can also be transmitted through pet birds. Allergic responses to pet birds, including pneumonitis and contact dermatitis, have also been documented. Bite wounds from pet birds are rarely reported but can cause trauma and develop infection. The other diseases discussed here are considered potential zoonotic diseases of pet birds because of either isolated reports of suspected but unconfirmed transmission to humans or from reports of wild conspecifics being reported to have the disease. For most diseases, humans with underdeveloped or compromised immune systems, including the very young, the elderly, HIV patients, individuals undergoing chemotherapy, or people otherwise immunosuppressed due to other disease are the most at risk.

BACTERIAL ZOONOSES

Chlamydiosis

Chlamydiosis is a zoonotic disease of great interest to pet bird owners and has received a vast amount of attention. Recently, the National Association of State Public Health Veterinarians (NASPHV) has completed an updated compendium to assist in the prevention and control of chlamydiosis among humans and pet birds.[1] A free copy of the compendium along with other resources to aid pet owners with infected birds, pet stores, and aviaries working toward detection and prevention is available at the NASPHV Web site.[1]

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According to the Centers for Disease Control and Prevention, 66 human cases of psittacosis were reported through the Nationally Notifiable Diseases Surveillance System between 2005 and 2009[1]; these statistics are likely an underrepresentation due to incorrectly diagnosed or unreported cases.[2] Most of the cases reported between 2005 and 2009 were attributed to exposure to pet birds infected with the bacterium.[1] Cockatiels, parakeets, parrots, and macaws were the most commonly represented species. Populations considered to be most at risk include bird owners, pet shop employees, and veterinarians. Due to the zoonotic potential, *C psittaci* is reportable in most states.

Chlamydiosis is caused by a small bacterial organism called *C psittaci.*[2–4] This organism is a gram-negative, obligate intracellular bacterium that transitions through at least 2 states during its life cycle. There is an elementary body stage that can infect cells either within the same host or in another host, and a reticulate body stage, which undergoes replication but is not able to infect other cells. An elementary body is extracellular, highly infectious, and metabolically inactive. Elementary bodies are resistant to many environmental stressors and can survive in soil for up to 3 months and in bird droppings for up to 1 month. Elementary bodies are inhaled or ingested by a host and attach themselves to an eukaryotic cell, most commonly a respiratory epithelial cell. After attaching to the cell, the elementary body undergoes endocytosis and forms an endocytoplasmic vesicle. This vesicle allows for the elementary body to remain safe from the host's immune defense system while it undergoes transition into the reticulate body. The reticulate body is the intracellular, metabolically active state that is capable of replication via binary fission. After replication, the reticulate bodies convert to elementary bodies and are released from the cell. Depending on the strain, host, and environmental conditions, the developmental cycle takes 48 to 72 hours. There is also the possibility of a third persistent state in which the organism is present and viable but cannot be eliminated by the host's defense system.[5–6] If this state exists, it is unlikely that a culture could successfully be obtained. The existence of this persistent state is controversial and documentation of its existence in naturally infected birds is lacking.

Birds infected with *C psittaci* may be asymptomatic.[7,8(pp4–96)] This is especially likely for pigeons and passerine birds, but is also seen with psittacine birds. Stress due to reproduction, raising young, transportation, shipping, overcrowding, and inadequate husbandry can increase the likelihood that birds will begin shedding the organism and/or showing clinical signs. Immunosuppressed birds and very young birds are most likely to succumb to severe infection. The organism can be transmitted vertically, and the very young may die soon after hatching or while still in the nest.

The typical incubation period is anywhere from 3 days to several weeks, but clinical signs and active disease may appear without any known risk or exposure.[1] Many of the clinical signs seen in birds, such as lethargy, decreased appetite, weight loss, and ruffled feathers, are very nonspecific. Disease of the respiratory, gastrointestinal, and ocular systems may result in more visible clinical signs. Liver disease due to *C psittaci* commonly results in lime-green diarrhea or bright green urates. Conjunctivitis, dyspnea, and ocular and nasal discharge are often reported. Severely affected birds may become completely anorexic, depressed, and die. These clinical signs are not unique to chlamydiosis but may support a potential diagnosis.

When a person becomes infected with *C psittaci* due to contact with a psittacine bird, the disease process is called *psittacosis* and has historically been referred to as parrot fever.[9] If a person becomes infected with *C psittaci* as a result of contact with a nonpsittacine bird, the term *ornithosis* is applied. *Chlamydiosis* is a broader term

that includes both psittacosis and ornithosis. All of these terms are used somewhat interchangeably in publications regarding zoonoses.

Psittacosis occurs in multiple age groups, but the most severe manifestations of infection are reported in people aged between 35 and 55 years.[4] Children rarely show severe signs when infected, and many individuals shown to be infected with the bacterium show signs mild enough to require minimal to no treatment. The severity of symptoms in people affected with *C psittaci* can range from subclinical to sepsis with multiorgan failure. Many resources describe flulike symptoms such as fever, chills, headache, muscle aches, and a dry cough as symptoms of human infection with *C psittaci.* Headache is the most commonly reported sign, followed by cough, dyspnea, confusion, and abnormal liver tests. Pneumonia diagnosed via thoracic radiographs is also commonly reported. The vast majority of people infected with *C psittaci* show mild signs, and when medical assistance is needed can be treated readily with antibiotic therapy.

There are other less common, more severe expressions of the disease, such as renal complications, hepatitis, pancreatitis, and reactive arthritis.[4] Neurologic and cardiac manifestations are also reported. Some infected individuals develop meningoencephalitis, with the most frequent clinical findings being fever, headache, and confusion. Less commonly, status epilepticus, localized cerebellar ataxia, and brainstem encephalitis are also seen. Cardiac manifestations of the disease include endocarditis, myocarditis, and pericarditis. Symptoms are often present for several months prior to a diagnosis. Endocarditis can be complicated by the development of glomerulonephritis, and surgery is often required even with appropriate and timely antibiotic therapy. Surgical intervention for infective endocarditis is aimed at removing infected tissue, draining any abscesses, repairing heart tissue damaged from the infection, and repairing or replacing affected valves.[10] Mortality with this syndrome approaches 50%.[4] Few other specific clinical presentations have been described in humans, which fortunately are very infrequent, including a fulminant form of psittacosis, gestational psittacosis, and chronic follicular meningitis.[4]

Psittaciforms, passeriforms, and columbiforms are among the 30 bird orders in which *C psittaci* has been documented.[11] Of these 3 orders of birds, most human cases are associated with exposure to psittacine birds, but passerine birds and columbiform birds are also recognized as sources of human infection.[4] Risk of transmission increases with close contact with infected birds that are actively shedding the organism. Birds undergoing stressful situations such as shipping, overcrowding, reproduction, or malnutrition are more likely to shed, resulting in transmission.[1] Birds that are shedding may not show any sign of disease. Infection is acquired through inhalation of aerosolized organisms in dried feces or respiratory tract secretions and through direct contact with infected birds. Persons developing persistent flulike symptoms, headache, respiratory distress, fever, confusion, or cough should consult with a physician and provide details regarding their exposure and interaction with birds.[4]

A combination of tests, including culture, antibody detection, and antigen detection, are recommended when looking for evidence of infection with *C psittaci* infection in birds.[1] Infection can be difficult to detect, especially in asymptomatic birds. There are no pathognomonic lesions that can be viewed on gross necropsy, but cloudy air sacs and enlargement of the spleen and liver support a diagnosis of psittacosis. Tissues or impression smears undergoing chromatic or immunologic staining can sometimes aid in identifying organisms. Liver and spleen are preferred tissues for bacterial culture.

In birds showing clinical signs, the use of a combined conjunctival, choanal, and cloacal swab sample and/or liver biopsy can be used for bacteriologic culture or

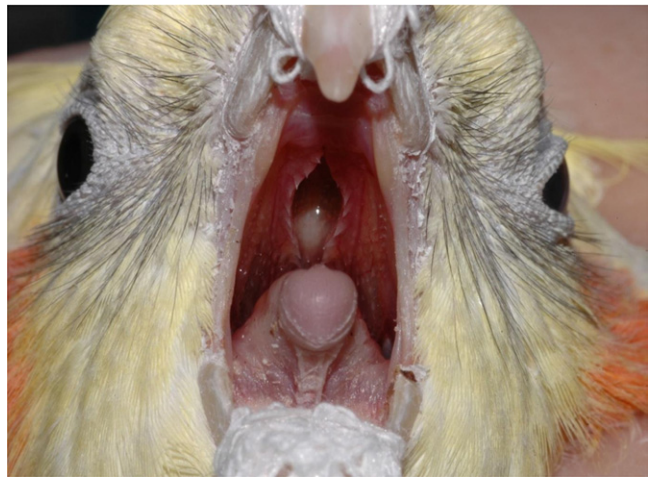

Fig. 1. Sample collection for diagnostic tests, such as bacterial culture and PCR, often includes swabbing the choana of a bird.

polymerase chain reaction (PCR; **Fig. 1**).[1] Depending on the stage of infection and affected tissue, birds may not shed detectable levels of the bacterium in their feces, and for this reason the conjunctival and choanal swabs are preferred to feces. If feces must be used, multiple collections of feces over 3 to 5 consecutive days should be collected and submitted together as a single sample. Samples should be refrigerated after collection and shipped on ice, but not frozen. The individual requirements of each lab may differ, and the sampler is encouraged to contact individual laboratories for their requirements since reliable detection of the bacterium relies heavily on appropriate handling and processing of the samples.

Antibody tests are also available.[1,12] Elementary-body agglutination detects IgM antibody, an early indicator of infection, to the infectious from of *C psittaci* elementary bodies. Indirect fluorescent antibody detects polyclonal secondary antibodies from the host, primarily IgG. Complement fixation is a very sensitive test for antibody but has been associated with a high rate of false-positives in parakeets, young African greys, and lovebirds.[1]

A positive test may reflect either an active infection or an appropriate immunologic response to a previous infection.[12] Antibody might not be found in infected birds that have been acutely infected and are not yet mounting a detectable immune response. Antimicrobial treatment could result in undetectable antibodies, but IgG can persist after successful treatment. To confirm a diagnosis of chlamydiosis, a positive antibody titer must be paired with either 1) a second antibody titer showing at least a fourfold or greater increase in titer, or 2) antigen identification.[1] A positive antibody titer with an elevated white blood cell count, increased serum liver enzymes, and known exposure is not a definitive diagnosis, but are all highly suggestive of chlamydiosis.

Antigen testing detects the presence of the organism even when it is not alive.[1,11] Cross reacting antigens may result in false-positives, and false-negatives can occur when the sample doesn't contain sufficient antigen, which may be due to intermittent shedding. Commercially available antigen tests include enzyme-linked immunosorbent assay and fluorescent antibody test. Positive antigen results should always be

evaluated with respect to the presence or absence of clinical signs in the bird. If the bird is asymptomatic, verification that the bird is shedding the organism can be pursued via isolation of the organism.

PCRs are available for testing on combined conjunctival, choanal, and cloacal swab specimens or blood.[1,13] PCR can be very sensitive and specific, but there are no standardized PCR primers and techniques for handling, and processing samples vary. A list of laboratories that currently offer testing for human and avian samples are listed in the NASPHV's compendium on avian chlamydiosis.[1] PCR and culture for avian samples are available at the Diagnostic Center for Population and Health at Michigan State University (Lansing, MI, USA), the Infectious Diseases Laboratory at the University of Georgia College of Veterinary Medicine (Athens, GA, USA), and Texas Veterinary Medical Diagnostic Laboratory (College Station, TX, USA). Some of these laboratories also offer antibody tests for avian samples. The Comparative Pathology Laboratory at the University of Miami (Miami, FL, USA) offers enzyme-linked immunosorbent assay, indirect fluorescent antibody, and PCR, whereas the Diagnostic Virology Lab of the National Veterinary Services Laboratories (Ames, IA, USA), offers culture and complement fixation. NASPHV recommends any birds that are suspected to be infected with *C psittaci* be evaluated using more than 1 type of test and that all tests are interpreted with regard to the bird's history and clinical signs.

Some veterinarians suggest treating any birds that may be infected regardless of test results, because of the zoonotic risk, but NASPHV discourages prophylactic antibiotic treatment.[1] Currently there is no documentation of antibiotic resistance to *C psittaci* in birds, but antibiotic resistance has been reported to other *Chlamydophila* species and is therefore of potential concern.[14] Historically, the treatment of choice was considered to be doxycycline administration for a minimum of 45 days in most species and 30 days in budgerigars.[1] Treatment for this length of time is still recommended to avoid incomplete resolution of infection. However, recent studies have indicated that other less lengthy protocols may be efficacious.[15]

Information regarding dosing and recipes for food and water administration to birds are readily available in the compendium on avian chlamydiosis by NASPHV.[1] Medicated food has successfully been used to treat chlamydiosis in budgerigars and cockatiels.[16,17] Suggested concentrations of doxycycline hyclate medicated water are available for use in cockatiels, African grey parrots, and Goffin's cockatoos.[1] Exotic doves have also been successfully treated using doxycycline-medicated water.[18] Budgerigars do not maintain therapeutic concentrations using medicated water and should be provided the medication via an alternative route. Some birds may develop toxicosis in response to doxycycline. If toxicosis is suspected, treatment with doxycycline should be discontinued and supportive care provided. Treatment can later be attempted with either a reduced dose of doxycycline or alternative regimen.

Pharmacokinetic studies have been undertaken to determine if the use of other medications or decreasing the length of administration of doxycycline from the recommended 45 days may be effective. Oral administration of azithromycin given every 48 hours or doxycycline given every 24 hours for a 21-day course of treatment have both been shown to be effective in treating cockatiels experimentally infected with *C psittaci.*[15] These results have not yet been tested in naturally infected cockatiels or any other bird species and therefore cannot yet replace the 45-day recommended treatment. Primary motivation to reduce the required length of treatment is to increase the likelihood of owner compliance and therefore decrease recurrence of the disease.

Regardless of which treatment protocol is utilized, infected birds should be isolated from other animals in clean, uncrowded cages.[1] Appropriate husbandry

with adequate nutrition and clean water should be maintained to reduce the risk of secondary infection. Birds should be weighed every 3 to 7 days, and if not able to maintain their weight, supplemental and gavage feeding may be required. High dietary intake of calcium from mineral blocks and cuttlebones should be avoided since it can inhibit the absorption of oral tetracyclines. In hand-fed neonates that require supplementation, calcium and tetracyclines should be given at least 4 to 6 hours apart. Facilities should be thoroughly cleaned and disinfected before termination of treatment to reduce the risk of reinfection. Two weeks after the completion of treatment is the earliest suggested time period for repeat screening.

Birds that are sick should not be sold or purchased.[1] Birds from multiple origins should not be combined without proper quarantine (minimum 30 days) and multimodal testing (antibody, antigen, PCR) for the presence of *C psittaci*. Cages, food containers, and water bowls should be positioned and cleaned to avoid the spread of fecal matter, feather dander, contaminated food, and other substances between cages. Fecal material and discharged food items should be removed daily. Prior to removal, it is recommended that fecal material and contaminated cage items be wetted or sprayed down to avoid aerosolization of the material. Ventilation should be sufficient to avoid accumulation and limit spread of aerosolized organisms.

In multibird households or facilities, healthy birds should be cared for prior to treatment and/or handling of infected birds.[19] All debris and fecal material should be scrubbed from cages. Disinfectants should be used to thoroughly clean any cages that have housed infected birds before they are reused. Bleach and water at a 1:32 dilution (1/2 cup of 5% chlorine bleach in a gallon of water), 1% Lysol, and quaternary ammonia compounds have been recommended as effective disinfectants. Most disinfectants require at least 5 to 10 minutes of contact time and any items that cannot be properly disinfected should be discarded. Rinse thoroughly to avoid irritation from the detergent. Cleaning methods that limit the aerosolization of materials, such as spraying down cages and floors prior to sweeping and mopping should be used. Vacuum cleaners and pressure washers should be avoided because of the risk of aerosolization.

Once clinical signs are noted in birds and a diagnosis of chlamydiosis obtained, human contact and the potential for transmission has likely already occurred. Therefore, any individuals caring for or surrounded by birds should be made aware of the potential zoonotic risk. People cleaning cages or handling infected birds should wear protective clothing that covers the hands, eyes, and head. These individuals should be fitted with respirators of a N95 or higher rating. When potentially infected birds are necropsied, the procedure should be completed in a biological safety cabinet, and detergent and water should be used to avoid infectious particles becoming aerosolized.

Salmonellosis

Another zoonotic bacterium with reported cases of suspected bird to human transmission is *Salmonella.*[20] There are 2 species of *Salmonella* with thousands of different serovars.[21,22] There are some serovars of *Salmonella* that are specifically adapted for avian hosts, such as *Salmonella* Pullorum and *Salmonella* Gallinarum, which primarily cause systemic disease in poultry.[23] Certain strains of *Salmonella* Typhimurium have been identified as being host adapted for causing disease in pigeons.[24] *Salmonella* infections with various serovars have been documented in psittacine, passerine, and columbiform birds.[25–27]

Several factors will dictate the manifestation and severity of *Salmonella* infection in pet birds.[23,28] The ability of the bird to mount an effective immune response will

depend on the infecting serovar, age of the animal, and presence of concurrent infection, malnutrition, poor husbandry, or stress, which may increase the risk of severe and potentially fatal infection. Some serovars of *Salmonella,* such as *S* Typhimurium or *Salmonella* Enteritidis are more likely to cause severe illness in both birds and humans.

Salmonella has been shown to have the potential to infect multiple avian organs of the respiratory, gastrointestinal, renal, neurologic, cardiovascular, and reproductive systems.[29(pp953–6)] Clinical signs reported in psittacine birds range from mild enteritis to severe anorexia, diarrhea, lethargy, dehydration, and crop stasis or sudden death. *Salmonella* spp have also been isolated from asymptomatic birds. Suspected transmission of *Salmonella* Typhimurium from psittacine birds to humans has been documented.[9,30–31]

Pigeons infected with *Salmonella* spp may also show a wide variation in clinical signs.[26] Infected pigeons may be asymptomatic and therefore the introduction of new birds, exposure to feral birds, or the routine racing and showing of pigeons carries risk of exposure. In the United Kingdom, where pigeon racing and showing is common, any isolates of *Salmonella* spp obtained from racing and show pigeons must be reported to Animal Health due to zoonotic risk. Pigeons that have ingested the bacterium may show signs of bacteremia and sepsis. Decreased appetite, weight loss, and decreased egg production and viability are commonly seen. Some birds may develop swollen and warm joints from septic or infectious arthritis or exhibit evidence of central nervous system disease.

Wild passerines, both ill and asymptomatic, have been repeatedly documented as carriers of *Salmonella* spp and have been implicated in the transmission of the bacterium to mammals and to humans.[32,33] As seen with infection in other avian species, *Salmonella* Typhimurium in passerines may manifest as systemic and multiorgan disease.[29(pp953–6)] Granulomas have been observed in the liver, spleen, and ceca. Finches and canaries, 2 of the most popular passerines species kept as pets in the United States, have also been shown to exhibit ocular lesions and osteomyelitis.[34]

Salmonellosis in people is usually the result of food borne illness or direct contact with an infected reptile or amphibian, rather than interaction with a pet bird.[35,36] Some individuals that ingest *Salmonella* spp may have no to mild signs of illness.[37] Most individuals that become ill from ingesting *Salmonella* spp have symptoms for 4 to 7 days. Clinical symptoms include abdominal cramps, headache, fever, nausea, vomiting, and copious watery diarrhea. Occasionally the symptoms may be severe enough that hospitalization in indicated. Rarely, serious complication or death may result from infection. In addition to causing gastrointestinal distress, some patients experience other systemic manifestations of infection with the bacterium, including, but not limited to, arthritis, hepatitis, and neuritis. The very young, the elderly, and people with underlying health issues are most likely to suffer severe disease.

The bacterium is typically spread via fecal to oral transmission but can also be spread through direct contact with infected animals or people.[35] The bacteria may be spread mechanically via contaminated clothing, shoes, equipment, and on rodents such as rats and mice.[26] This disease can also be transmitted vertically into the egg or through crop milk. Food and water contaminated with feces from infected animals, both wild and domestic, is also a potential source of infection.

Definitive diagnosis of salmonellosis requires successful culture of the organism. Antemortem, repeat, or pooled fecal samples are cultured. The use of selective enriched culture media is required, and laboratories must be told that salmonellosis is suspected. The shedding of the bacterium is often intermittent and false-negatives

are common. Postmortem, multiple samples from a wide range of tissues should be submitted for culture. In flocks with large die off, multiple birds should be submitted for evaluation.

The severity of the disease presentation dictates the intensity of treatment.[26] Supportive care, including fluids and gavage feeding, may be indicated. Antibiotic use is controversial because resistance is common; if warranted, antibiotic choice is best determined by the result of culture and susceptibility testing. Enrofloxacin and amoxicillin are common choices. Maintaining cleanliness within the cages or lofts and providing clean water and food are essential in allowing for recovery. Length of treatment may last from 10 days to 3 weeks to allow for clearance of the bacteria.

Prevention of *Salmonella* spp infection relies heavily on cleanliness.[37] Appropriate husbandry must be maintained, providing for clean surroundings, clean food and water, and avoiding contact with ill and infected animals. Any materials with infected feces should be removed from the enclosure. Sodium hypochlorite (bleach) at a concentration of 0.05% and alkaline peroxide at a concentration of 1% have been shown to be effective against *Salmonella.*[38] The effectiveness of some disinfectants against *Salmonella* adhered to surfaces or contained within a biofilm may be reduced.[39] Suggestions for disinfectants to combat *Salmonella* in these situations include those containing 70% ethanol. Virkon S was also effective at eliminating *Salmonella* found on surfaces. Rodent control should be implemented.[26] Humans working with animals that may carry *Salmonella* spp should wear disposable gloves, frequently wash their hands, discard any items sullied with feces, and avoid eating, drinking, or putting hands near the face and mouth without appropriately washing with soap and warm water.[37]

Mycobacteriosis

Mycobacterium spp are Gram positive, aerobic, acid-fast bacillus that infect birds, mammals, and humans.[40] The most commonly isolated *Mycobacterium* spp from pet birds are *Mycobacterium avium* and *Mycobacterium genevense.* Other species of *Mycobacterium* are infrequently identified in pet birds, including *Mycobacterium tuberculosis,* the agent responsible for tuberculosis in people. *M avium* subsp *Hominis suis* has also been diagnosed in a 6-month-old female, blue-fronted Amazon parrot with inappetence, slight emaciation, heavy biliverdinuria, ascites, and melena.[41] This subspecies rarely causes disease in birds, but has been shown to cause severe disease in humans, especially immunocompromised individuals.

Clinical signs of mycobacteriosis in birds vary widely dependent on the *Mycobacterium* spp, resulting in infection, the species of bird affected, the duration and severity of exposure, and the organ system infected.[41] Weight loss is the most consistent finding reported across multiple species. Respiratory disease can occur, but diarrhea, coelomic distension, and poor feathering are more frequently reported.[42] Weight loss and failure to respond to routine antibiotic therapy are commonly documented in pet birds shown to be suffering from mycobacteriosis.

People afflicted with acquired immune deficiency syndrome are commonly afflicted with what is known as the Mycobacterium complex (*M avium* and *Mycobacterium intracellulare*) and are the individuals most likely to develop systemic mycobacteriosis.[40,43–44] The likelihood of infection increases directly with the severity of immunosuppression. Individuals undergoing organ transplant or those experiencing disease resulting in immunosuppression are also at risk. Fever, weight loss, abdominal pain, fatigue, chronic diarrhea, and anemia are reported in people with systemic mycobacteriosis.

Localized disease also occurs in humans including central nervous system infection, boney or soft tissue lesions, cervical lymphadenitis, or endocarditis.[43]

M tuberculosis causes pulmonary disease known as tuberculosis in humans characterized by a persistent cough, chest pain, and coughing up blood and/or sputum.[45] Other signs include weight loss, fatigue, fever, and generalized malaise. Other parts of the human body may also be impacted by this infection including the kidneys, spine, and brain. Tuberculosis was once the leading cause of death in the United States and is still a common fatal illness in other parts of the world. Many individuals who become infected with *M tuberculosis* develop latent infection because their immune systems are able to fight the disease. Latent infections can transition to active infection within the body, causing illness when a person's immune system is not able to fight the infection. Infection can stay latent weeks to several years, and those with suppressed immune systems, such as those with acquired immunodeficiency system, are at increased risk of becoming ill.

Mycobacterium spp are very stable in the environment and can remain in the soil for years.[40] Infection is typically secondary to ingestion of the bacterium, inhalation of the organism, or from introduction of the organism into open cutaneous lesions. *Mycobacterium* organisms are found worldwide and have been isolated from soil, water, animals, birds, and foods. Environmental sources such as contaminated water, food, and soil are considered the most likely sources of infection for people and animals. Possible transmission of *M tuberculosis* from humans to pet birds, including an African grey and a green-winged macaw, has been reported, and there is concern that a pet bird could harbor this organism and serve as a carrier and source of infection to other birds and humans.[46,47] *M tuberculosis* has also been identified in pet passerines.[48] No confirmed transmission of *Mycobacterium* from a bird to a person has been reported.

Characteristic hemogram findings for birds diagnosed with mycobacteriosis include nonregenerative anemia, and leukocytosis with heterophilia and monocytosis.[40] Depending on the organs most affected, lesions may include pulmonary granulomas, enlarged liver, engorged intestinal loops, or boney lesions. A presumptive diagnosis of mycobacteriosis may be made if acid-fast bacilli are detected in biopsy or necropsy specimens. However, mycobacterial culture or polymerase chain reaction analysis is required for definitive diagnosis. Care must be taken when acid-fast organisms are detected in fecal samples, as nonpathogenic acid-fast organisms, including saprophytes, may be present. Due to the prolonged period of time often necessary to culture *Mycobacterium* spp, diagnosis in birds is often based on histological evidence of mycobacteriosis in diseased tissue (liver, spleen, intestine) and PCR.[49]

Therapy is controversial due to the potential for persistent infection, antibiotic resistance, and the zoonotic potential.[40] If owners want to pursue therapy for a pet bird, it is recommended that the veterinarian have owners sign a release that explains the possibility of transmission of *Mycobacterium* from the bird.[50] Successful treatment has been achieved in pet birds with multimodal antibiotic therapy.[40] Several protocols are available with most, including drugs such as clarithromycin or azithromycin, rifampin or rifabutin, and ethambutol. Treatment takes many months, and monitoring success of treatment is difficult as the organism can be difficult to detect in low numbers.

Reports of transmission of *Mycobacterium* spp to humans from pet birds is lacking. However, there is a recognized potential for human transmission with pet birds potentially serving as reservoirs. Prevention of mycobacteriosis relies heavily on prompt detection of the bacterium and avoiding humans and birds that are showing signs of illness. Maintaining overall health through proper diet and exercise supports

a healthy immune system that reduces risk of infection. Making sure that food, water, and the environment are free of contamination can aide in prevention. *Mycobacterium* spp can be challenging to destroy and are often resistant to commonly used disinfectants.[51] Bleach, one of the most commonly used disinfectants is only effective at high concentrations and quaternary ammoniums are not effective. Phenolics are the best disinfectant for inactivating *M tuberculosis* and potentially other *Mycobacterium* spp. Care must be taken to avoid generating droplets or aerosols when cleaning, as inhalation is a primary means of transmission. If a person believes that they or their bird have been exposed to individuals infected with *Mycobacterium* spp they should seek medical or veterinary attention respectively.

Other Bacteria

There are multiple other potential bacterial zoonotic pathogens, including *Mycoplasma,* a bacterium characterized by the lack of a cell wall, which causes conjunctivitis, tracheitis, air sacculitis, and chronic respiratory disease most commonly in poultry and wild passerines, but also in pet birds.[52] Multiple gram-negative bacteria such as *Pasteurella* spp, *Klebsiella* spp, *Yersinia* spp, *Campylobacteriosis* spp, and *Escherichia coli* are also potential zoonotic pathogens identified in pet birds. Documented evidence of zoonotic transmission to humans from pet birds is lacking for these bacterial pathogens but the potential is present. Appropriate hygiene and husbandry, quarantine of sick birds, and maintaining clean food and water sources will reduce risk of infection and possible transmission.

Viral Zoonoses

Avian paramyxovirus, avian influenza, and West Nile virus (WNV) are potential zoonotic infectious agents found in pet bird species. There are not currently any documented cases of direct transmission of these viruses from pet birds to humans, but pet birds have the potential to serve as reservoirs for viral infection. Poultry and wild bird populations, which are discussed elsewhere in this publication, are more likely to harbor these viruses, and therefore, discussion of these viruses within this chapter on pet birds will be abbreviated.

Newcastle Disease Virus

Avian paramyxoviruses (APMV) have been observed in pet birds, but most published descriptions of APMV are based on poultry. APMV includes Newcastle disease virus (NDV), which is caused by serotype 1 (APMV-1).[53] There are nine serovars of avian paramyxovirus, and each serotype is characterized by the type of bird affected. PMV-1 is of great concern in the poultry industry and is a potential zoonotic disease. In humans, NDV infection may result in mild flulike symptoms, conjunctivitis, or laryngitis. Classic Newcastle disease was observed to infect pigeons during poultry outbreaks in the United Kingdom.[26] There is documentation that imported psittacine birds may carry APMV, and passerines have also been shown to suffer from avian paramyxovirus.[54–56] Pigeons may become infected with a variant strain similar—but distinct from—the classic NDV, which is often referred to as PMV-1 or PPMV-1.[26] Monoclonal antibodies are utilized to differentiate this strain from classic exotic Newcastle disease. Vaccination of pigeons for PMV-1 is now a requirement for show and racing pigeons in the United Kingdom.

The virus is shed through respiratory secretions and feces, and exposure to NDV is usually due to ingestion or inhalation of contaminated substances.[53] Direct transmission is possible, and vectors such as insects, humans, and rodents can exacerbate

the extent of infection. To confirm a diagnosis, isolation of the virus from infected tissues to identify serotype and virulence must be completed. Antemortem samples typically include cloacal and tracheal swabs. In birds, treatment is supportive and most cases are fatal. Prevention through use of personal protective equipment and good husbandry practices is encouraged to avoid infection.

Influenza

Avian influenza or influenza A is in the family Orthomyxoviridae and has been associated with respiratory disease in multiple avian species, mammals, and humans.[57] There is great variance in clinical signs dependent on the strain and associated virulence and the susceptibility of the infected species.[58] The virus is classified according to surface proteins called hemagglutinin (H) and neuraminidase (N); there are 16 H and 9 N unique proteins currently identified. Many wild birds, especially waterfowl, infected with the virus are asymptomatic and serve as a reservoir for a strain that may result in disease in domestic birds. A highly pathogenic strain, H5N1, has caused significant poultry losses and human disease and death predominantly in Asia over the past decade. This virulent strain also caused death in birds that are typically reservoirs for the virus, including free-ranging ducks, shorebirds, and passerines.[59]

Mild to severe respiratory signs are often accompanied by depression, anorexia, diarrhea, or neurologic signs in birds.[58] Inhalation or direct contact with respiratory, fecal, or ocular secretions are the main modes of transmission.[58,59] Antemortem diagnosis is conducted via viral isolation from tracheal and/or cloacal swabs.[58] Vaccinations are available for birds and have been used in an effort to protect some valuable zoological species of birds; however, due to the potential zoonotic and economic impact, there are strict legal restrictions governing their use.[60] Potential disadvantages of the use of the vaccine include the masking of clinical signs of infection, which could translate into human exposure and possible infection.

There are no current reports of H5N1 in pet birds within the United States; however, there is a report of low pathogenic H5N2 isolated from a 3-month-old red-lored Amazon parrot with severe lethargy and gastrointestinal distress, including regurgitation and melena.[61] The bird was kept in quarantine, given supportive care, and recovered from clinical signs within 4 days. The bird was released 9 weeks after presentation when virus isolation and PCR were negative for the previously identified virus. Avian influenza subtypes H5 and H7 are reportable in the United States, and most often result in depopulation within the poultry industry due to the risk of the virus mutating into a highly pathogenic form. However, as demonstrated in the aforementioned case, not all pet birds are destroyed.

Precautions recommended by the US Department of Agriculture can be taken to protect pet birds from acquiring infectious diseases such as Newcastle disease and avian influenza.[62] First, owners should limit access to their pets. Allowing contact with individuals whom own their own birds, have exposure to sick birds, or work in occupations dealing with birds such as pet stores or poultry plants should be avoided. Owners should make sure their hands and clothes are clean prior to handling their pets. Food and water should be replaced daily. Cages should be kept clean and droppings removed from any toys or materials kept within the household prior to disinfection. If acquiring a new pet bird, proof that the bird was legally imported or bred within the United States should be requested from the seller. Sick birds should never be purchased and new birds should be quarantined for at least 30 days. If a pet bird has been to a show, club meeting, or other event involving exposure to multiple birds, that pet should be kept separate from any other birds in the household for at

least 2 weeks. Any birds showing signs of illness should be examined, and multiple deaths within a pet bird collection should be reported to the U.S. Department of Agriculture.

WNV

WNV is a flavivirus for which wild birds have been identified as the main reservoir.[63] The virus causes neurologic and ocular disease in birds and has been associated with neurologic and respiratory manifestations in mammals including humans. The virus was first identified in 1999 in the Western Hemisphere, where wild and zoo birds in the New York City area started dying from the virus. Species commonly kept as pets, including passerine and psittacine birds, have been fatally infected with WNV and may serve as a potential reservoir for human infection.[64,65]

Mosquitoes, primarily *Culex* spp, are the primary route of transmission.[63,64,66] The mosquito becomes infected when feeding on birds infected with the virus and then spreads the virus to mammals, including humans, when the mosquito bites. Oral transmission of the virus through ingestion of infected food items or from direct bird to bird contact as occurs in courtship behavior has been reported. Direct contact, organ transplant, intrauterine contact, and receiving blood donation products have been documented as transmission routes in human infections.

Infected birds often display varying degrees of neurologic compromise, including recumbency and paralysis of the pelvic and thoracic limbs. Virus isolation is best achieved from the brain, spleen, and kidneys. PCR is available and typically performed on oral or cloacal swabs, but is not always successful in antemortem diagnosis.

Clinical symptoms in humans develop 3 to 14 days after being bitten by an infected mosquito. Approximately 80% of people infected with WNV will show no symptoms.[67] Up to 20% of people will show mild signs including fever, headache, nausea, vomiting, and rash. Clinical signs can last for days to weeks. Rarely severe clinical signs such as high fever, neck stiffness, disorientation, tremors, convulsions, weakness, vision loss, numbness, and paralysis may occur. The duration of illness may last weeks and neurologic detriments may be permanent.

Treatment for infection with WNV is primarily supportive. There is no specific treatment, and in birds, infection severe enough to result in neurologic impairment is often fatal. Minor clinical signs identified in people such as fever and muscle aches may pass without any therapy. Severe cases of infection in humans will require hospitalization, intravenous fluid therapy, breathing assistance, and additional supportive care.

Limiting exposure to mosquitoes is the primary goal in prevention. Standing water and areas that promote insect breeding should be treated with larvicides. People should wear repellents when outdoors. Poultry houses and other avian enclosures, including aviaries for zoological species and pet birds, should be constructed to limit insect exposure. Many facilities within the United States that keep captive avian species have started vaccination programs using a commercially available equine vaccine, but efficacy is unknown.[58] Due to concerns over safety and efficacy, vaccination for WNV is not routinely done for pet birds but may be considered in pet birds kept outside with exposure to wild birds and mosquitoes.

Fungal Zoonoses

There are multiple fungal organisms that can infect both birds and humans.[68] *Aspergillus* spp and *Candida* spp are frequently responsible for respiratory or gastrointestinal illness in pet birds, respectively, and can also result in severe disease

in immunocompromised individuals. There is currently no evidence that humans acquire these fungal infections directly from birds, but rather acquire infections from environmental exposure.

Some fungal organisms, including *Cryptococcus* spp and *Histoplasmosis* spp, grow well in soil with high nitrogen levels, which is often due to the presence of bird and/or bat feces. *Histoplasmosis capsulatum* is most commonly associated with dove and pigeon feces, and the avoidance of areas that contain high levels of bird and bat droppings are recommended. In most cases, infection with these organisms goes unnoticed and humans are asymptomatic. In a few cases, symptoms of respiratory disease, including cough, headache, chest pain, and fever, can result. In rare cases, histoplasmosis can become disseminated and spread to organs outside the lungs. *Cryptococcus neoformans* has a tendency to infect the central nervous system and can result in meningoencephalitis. When symptoms are severe or the person is immunocompromised, infection without appropriate antifungal treatment can be fatal.

Birds rarely develop clinical signs associated with colonization of *C neoformans.* There is one report of a Moluccan cockatoo suffering from systemic cryptococcal disease and one report of a cockatoo that exhibited cutaneous lesions.[69,70] Human infection with these organisms is not from direct transmission from a bird, but rather from exposure to the organisms in the environment, and therefore—strictly speaking—is not considered zoonotic. However, some of these fungal organisms may be found in the feces of caged birds and therefore, pet birds may serve as a potential reservoir for infection.[71–75] Individuals who are immunocompromised are considered at risk, and rarely, otherwise healthy individuals may become ill from infection with *Cryptococcus* spp.[76]

There are 2 reports of meningitis from *C neoformans* in the literature that are believed to be due to exposure to a pet bird's contaminated and aerosolized excreta. An elderly, immunocompromised woman was diagnosed with meningitis from an isolate of *C neoformans* identical to one recovered from the feces of an asymptomatic Umbrella cockatoo cared for in the same household.[77] Exposure to aerosolized cockatoo excreta containing *C neoformans* was cited as the suspected cause of human infection. Although the bird had shared the same home with the woman for 7 years, the bird was housed on a different floor of the house and the woman was not directly involved in caring for the bird or cleaning its cage. In the second case, an immunocompetent woman was diagnosed with *Cryptococcus* meningitis after exposure to a magpie bird kept as a pet in her parent's household.[78] The magpie's feces cultured positive for *C neoformans.* No direct contact with the bird was identified and the woman had only lived in the house with the bird for 3 months. Consistent with the suspected aerosol exposure described in these cases, experimentally, *C neoformans* has successfully been isolated from air near caged birds.[79] Cutaneous nodular lesions due to dermatologic infection with *Cryptococcosis* spp have also been observed in an immunocompromised pet cockatoo owner, but the origin of the infection was not published.[80]

Parasitic Zoonoses

Species of birds kept as pets have been diagnosed with *Giardia* spp and *Cryptosporidium* spp.[81–83] However, to the author's knowledge there are no reports of direct transmission from a pet bird to a human in the literature. There are reports of wild birds serving as possible reservoirs for the parasites, increasing the possibility of pet birds also serving as reservoirs.[84–89]

Most *Giardia* spp isolated from birds, such as *Giardia ardeae* and *Giardia psittaci* are not considered zoonotic due to their host specificity.[81] However, 1 species

isolated from psittacine birds, *Giardia duodenalis* may be infectious to humans. *G duodenalis* trophozoites isolated from a parrot (*Cacatua galerita*) were used to colonize the small intestinal tracts of domestic kittens and lambs.[90] The experimental infection resulted in diarrhea in most of the kittens, but the lambs remained asymptomatic. This indicates that this *Giardia* sp may infect some pet bird species, resulting in a potential source of infection and disease for some mammals, potentially including humans.

Cryptosporidium spp reside in the gastrointestinal system of infected humans and animals.[91] The organism can be passed in droppings, and contaminated water is the most frequently cited source of infection. Research indicates that wild birds including songbirds, parrots, and pigeons may be a mechanical vector for this parasite, increasing the risk of pet bird and human exposure.[75,81–83]

Ectoparasites found on birds have the potential to cause dermatologic lesions in humans.[20] Mites, including *Ornithonyssus sylviarum* and *Dermanyssus gallinae,* most commonly infect poultry and wild birds and are very rare in pet birds.[92] Human skin serves as an incidental host and lesions are mostly localized, but can be intensely pruritic. Papular to papulovesicular eruptions in response to the mite can also occur. These mites cannot reproduce on a human host and thus, the infection is self-limiting. The sections of this publication focusing on zoonotic infections from poultry and wild bird populations should be consulted for additional information.

Hypersensitivities and Dermatologic Conditions

Hypersensitivity pneumonitis (HP) is a lung disease characterized by lymphocytic inflammation and formation of granulomatous pulmonary lesions resulting from an inhaled antigen.[93] Dust and mites encountered during occupational exposure have historically been the most frequently cited antigens, however case reports of HP resulting from exposure to pet birds, including psittacine and columbiform species, are increasing in frequency. Exposure to feathers, feather dander, and bird droppings have all been linked to allergic alveolitis. In addition to pet bird exposure, bedding filled with feathers from species of waterfowl and poultry has also been implicated in causing HP.

There are documented reports of pet birds suffering from a suspected similar allergic pneumonitis.[94] Most instances involve South American psittacine birds exposed to the feather dander in the environment from another avian species, most notably cockatoos and cockatiels. This disease is commonly called chronic obstructive pulmonary disease or macaw pulmonary hypersensitivity because of the overrepresentation of blue and gold macaws (*Ara ararauna*).[95] Although early stages of the disorder often go unnoticed, advanced stages are often characterized by polycythemia and exercise intolerance.[96] Atrial smooth muscle hypertrophy is the most prominent lesion, but proliferation of parabronchial lymphoid tissue and lymphoid nodular formation may also occur. Treatment is based on symptomatic therapy, air purification, and removal of the inciting antigen.

In people, HP can present as either an acute or chronic form.[93,97] The acute form often results in symptoms 4 to 6 hours after exposure to the antigen. Cough, fever, chest pain, dyspnea, and generalized malaise are common symptoms of acute exposure to an antigen to which the individual has hypersensitivity. In the chronic form, signs are more gradual, but often progressive and can include breathlessness that is exacerbated during exercise, a dry cough, decreased appetite, and unplanned weight loss. There may be a genetic predisposition in people that results in the development of HP.[93] Diagnosis is often based on history of antigen exposure, clinical symptoms, blood work, and imaging (including chest radiographs and CT). In some

cases bronchoscopy with biopsies, pulmonary function tests, and antibody panels to detect specific hypersensitivities may also be performed. Treatment involves identifying and avoiding the antigen, which can be difficult when the bird is living in the home as a pet. Removal of carpeting, regular cleaning, and air filtration can reduce antigen burdens in the home. For individuals breeding birds, a change in occupation may be necessary. Glucocorticoids may be given to reduce inflammation in the chronic form of the disease. When exposure to the antigen continues and treatment is not initiated, irreversible pulmonary fibrosis and/or emphysema may result.

Birds most commonly reported to be associated with the development of HP include pigeons and budgerigars.[98] It is possible that this overrepresentation may be due to the popularity of these species as pets. Other avian species frequently cared for as pets, including canaries and various other psittacine birds such as cockatiels, lovebirds, and rosella parrots have also been associated with HP in humans.[99,100]

Cutaneous reactions from skin allergies can also result from dermatologic exposure to pet birds.[101] Additionally, as most pet bird owners know, pet birds can bite and cause significant skin lesions. Despite the fact that animal bites are the most commonly documented zoonotic risk from pets, bites and scratches from pet birds are rarely reported in peer-reviewed literature. However, secondary infection developed following a bite from a pet cockatoo in a 68-year-old woman.[102] She was bitten on her right hand between the second and third digits; the woman did not seek professional medical advice until 30 days after the bite occurred. Culture of the lesion was positive for *Mycobacterium chelonae/abscessus.* The wound was surgically excised and long-term antibiotic therapy for 12 months was eventually successful. A second report documents a 59-year-old diabetic woman who was diagnosed with pyoderma gangrenosum after being bitten and scratched by a crow.[103] Pyoderma gangrenosum, a rare noninfectious neutrophilic dermatosis is typically associated with an underlying systemic disease. The initial bite wound cultured positive for *Citrobacter koseri* and *E coli,* both of which have been cultured from the gastrointestinal tract of birds. Although bird bite reports are rare in the literature, the potential for severe tissue damage, infection, and systemic illness exist, and those suffering bites from pet birds should perform appropriate wound care, including cleaning and flushing, and seek medical attention when appropriate. Research is needed to more fully assess the risk associated with bites sustained from pet birds, particularly infection that can occur due to inoculation of bacteria into the wound.

SUMMARY

In summary, true zoonotic infection resulting from exposure to pet birds is rare. Most birds with potentially zoonotic diseases do not present serious risk to healthy individuals, but all individuals interacting with birds should observe proper hygiene practices to lessen the risk of transmission. Individuals handling pet birds or their excretions should ensure proper sanitation. Veterinarians play multiple critical roles in limiting exposure to zoonotic and potentially zoonotic diseases. Routine and preventative veterinary care can aid in the recognition and treatment of disease. By developing a thorough understanding of the diseases and methods of transmission, veterinarians can effectively communicate risks and appropriate precautions to their staff and pet bird owners.

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