Disease Transmission from Companion Parrots to Dogs and Cats: What is the Real Risk?

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Numerous pet caregivers harbor unwarranted concerns regarding the potential of disease transmission from companion parrots to their dogs and cats. Such concerns may result in an overemphasis on testing and treatment for some of these potential pathogens in companion parrots, rehoming of companion parrots, or prevention of the adoption of parrots because of concern over the health of the household dogs and cats. A number of bacterial, viral, fungal, and parasitic diseases are postulated to undergo transmission from parrots to dogs and cats. In reality, transmission of Pasteurella multocida from companion cats to parrots is of greater concern. The following discussion reviews common conditions presumed to undergo such transmission and demonstrates the lack of data to support such concerns.

Mycobacterium spp
Infections with Mycobacterium avium or Mycobacterium genavense are not uncommon in psittacine species.1,2 M. genavense is an atypical mycobacterium that is an important source of infection in parrots.2 Rare reports of infection with Mycobacterium tuberculosis or Mycobacterium bovis exist, and such infections probably occur secondary to close contact with infected humans.2

Mycobacterium spp are ubiquitous in the environment and are found in soil with heavy fecal contamination or other organic debris.2 Additional sources include surface water or marshy shaded areas.2 Transmission occurs by ingestion or inhalation of soil or water contaminated by feces or, less commonly, urine, and vertical transmission is also possible.2 In parrots, the primary site of entry and initial colonization is the gastrointestinal tract.2 Inhalation may lead to direct colonization of...
respiratory tract, and focal skin disease secondary to inoculation into mucosal or dermal tissues or by contaminated needles may also occur. There is no apparent gender predilection.

Clinically, affected birds demonstrate muscle wasting, loss of subcutaneous and intracoelomic adipose tissue, and poor quality feathers. Initially the bird has a good appetite, followed by anorexia as the disease progresses. Other findings include wasting with increased appetite, poor feather quality, lethargy, weakness, pallor, chronic or intermittent diarrhea, abdominal distension, and rarely, ascites or pericardial effusion. Bone disease is reported to have a wide prevalence (2%–93%) and can result in acute or chronic lameness or a shifting leg lameness. Respiratory disease and reproductive failure are less commonly described.

Grossly, hepatosplenomegaly is commonly reported. Granulomatous lesions occur in the gastrointestinal tract and liver. Tubercles are white, tan, or yellow. Miliary foci to nodules several centimeters in diameter may occur within the gastrointestinal wall, liver, spleen, and bone but may occur in other viscera. Generalized disease may be associated with diffuse enlargement of affected organs. The gastrointestinal tract is often distended and thickened, and the intestinal mucosa may display a shaggy-carpet appearance. The carpometacarpal and elbow joints are most commonly involved, and the skin overlying joints may be thickened and ulcerated. Granulomas in the lungs or compression of air sacs secondary to hepatomegaly can lead to dyspnea or exercise intolerance. Rare nodules are reported within the infraorbital sinus, nares, and syrinx. Tubercles in the skin are rare, and dermatitis results in diffuse nonpruritic thickening and subcutaneous masses. Infections of the eyelids, nictitating membranes, retrobulbar tissue and pecten, and cornea are also reported in birds. Additional reports of lesions in the oropharynx, larynx, and external auditory canal exist. Reproductive failure due to infection of the adrenal glands, pancreas, and gonads resulting in subsequent endocrine abnormalities have infrequently been reported. Pulmonary necrosis, as well as granulomatous cardiopulmonary arteritis, are less common presentations.

Histologically, mycobacterial infections typically present as a granulomatous enteritis, splenitis, or hepatitis with variable intracytoplasmic acid-fast bacteria. Additionally, macrophages within the dermis, mucous membranes, and subserosa of the peritoneum and air sacs are reported. Granulomatous intestinal lesions are associated with expansion of the intestinal villi by diffuse infiltrates of epithelioid macrophages, multinucleated giant cells, fewer lymphocytes, and proliferation of epithelial cells within the glands of Lieberkühn. Infection with \textit{M avium} typically results in large numbers of acid-fast bacilli, whereas \textit{M bovis} and \textit{M tuberculosis} result in small numbers of acid-fast bacilli. In parrots, granulomas do not possess regions of central calcification or extensive necrotic centers. The diffuse form is more difficult to recognize with resultant diffuse infiltration with large foamy histiocytes.

In dogs and cats, mycobacterial infections may be caused by a number of different but closely related bacteria. Relevant members of the tuberculosis complex group in dogs and cats include \textit{M tuberculosis}, \textit{M bovis}, and \textit{Mycobacterium microti}. Other mycobacteria that can be potentially pathogenic in cats and dogs include \textit{Mycobacterium lepraemurium} and opportunistic nontuberculous mycobacteria such as members of the \textit{Mycobacterium chelonae-abscessus} group, \textit{Mycobacterium fortuitum} group, \textit{Mycobacterium smegmatis} group, and others. In general, mycobacterial infections are rare in both cats and dogs. The majority of cases are seen in cats and present as skin lesions. Most infections in dogs and cats are due to \textit{M bovis} or, in the case of cats, \textit{M microti}. Infection with \textit{M tuberculosis} is increasingly rare. Many cases in dogs and cats are subclinical. Infection usually occurs after
prolonged exposure, and disease is seen mainly in adult animals.\textsuperscript{3} No gender predisposition is seen in dogs, but male cats seem to be overrepresented.\textsuperscript{3} Certain breeds seem to be predisposed, including Siamese, Abyssinian, Bassett hounds, and Miniature Schnauzers.\textsuperscript{3}

Depending on the route of infection, infected dogs and cats may present with systemic signs related to the alimentary or respiratory tract or with localized disease affecting the skin.\textsuperscript{3} The usual presentation for tuberculosis in cats is cutaneous.\textsuperscript{3} These lesions probably arise from infected bite wounds, local spread, or hematogenous dissemination to the skin.\textsuperscript{3} The lesions often involve the face, extremities, tail base, or perineum. Less frequently, lesions involve the ventral thorax.\textsuperscript{3} Lesions typically appear as firm, raised dermal nodules. Ulceration may be present, as well as nonhealing wounds with draining tracts. Granulomatous inflammation may extend into adjacent subcutaneous tissues, muscle, and bone. Skin lesions are commonly associated with localized or generalized lymphadenomegaly.\textsuperscript{3} On occasion, submandibular or prescapular lymphadenomegaly may be the only clinical finding.\textsuperscript{3}

When the infection spreads to the lungs, tubercles arise in the lungs and hilar lymph nodes, and affected animals present with weight loss, anorexia, dyspnea, and cough.\textsuperscript{3} Additionally, there may be associated sneezing and nasal discharge.\textsuperscript{3} Pneumothorax and pleurisy may also occur with pleural and pericardial effusions.\textsuperscript{3} Pulmonary cases in dogs have occasionally presented with hypertrophic pulmonary osteopathy.\textsuperscript{3}

In the alimentary form, tubercles arise in the intestines and mesenteric lymph nodes, and affected animals present with weight loss, anemia, vomiting, and diarrhea.\textsuperscript{3} Occasionally, tubercles arise in the tonsils.\textsuperscript{3}

In dogs and cats, a range of clinical signs may develop with disseminated disease including splenomegaly, hepatomegaly, generalized lymphadenomegaly, weight loss, and fever.\textsuperscript{3} With bone involvement, lameness may develop. Ocular involvement may result in granulomatous uveitis, retinal detachment, and central nervous signs due to extension along the optic nerve.\textsuperscript{3} Mycobacterial conjunctivitis may also be seen alone or associated with more generalized changes.\textsuperscript{3}

To the authors' knowledge, there are no known published cases of transmission of \textit{Mycobacterium} spp from parrots to dogs and cats. It is more likely that companion pets acquire such infections from living with infected people or through opportunistic infections from environmental exposure.

\textbf{Chlamydophila psittaci}

\textit{Chlamydophila psittaci} is a zoonotic intracellular bacterial organism that was first reported in humans and psittacine birds in 1895.\textsuperscript{4} Most parrots and over 130 species in other taxonomic orders of birds have been found to be capable of being infected by this agent. The potential host spectrum of \textit{C. psittaci} also includes practically all domestic mammals including humans and many wild mammals, some amphibians, and arthropods. \textit{Chlamydophila} spp have an intracellular life cycle and are periodically shed by infected birds.\textsuperscript{4} Carriers can shed the organism intermittently and typically do not demonstrate any clinical signs.\textsuperscript{1} The organism propagates in the epithelial cells of the respiratory tract and then generalizes to other organs.\textsuperscript{5}

Clinical signs of disease are quite variable and are dependent on the virulence of the infecting strain as well as the host. Young birds with an incompletely developed immune system may develop acute systemic infections when exposed to particularly virulent strains of \textit{C. psittaci}. Most commonly, clinical signs noted in psittacine birds may include respiratory and/or gastrointestinal abnormalities. Clinical signs associated with liver disease are not uncommon in psittacine bird species.
diarrhea, biliverdinuria). Some birds may occasionally develop central nervous system abnormalities, keratoconjunctivitis, nasal discharge, and in the cockatiel, flaccid paresis and paralysis have been reported. Multisystemic disease can also develop in cockatiels and other species of parrots.5

Gross lesions include hepatic and splenic enlargement. In parrots, infections of the air sacs result in diffuse cloudy opacification with occasional tan-yellow plaques.5 Affected livers may have minimal gross changes, but many are enlarged and discolored, containing foci of necrosis.5 The spleen may range from dark red to purple, or the spleen may be pale as a result of increased inflammatory infiltrates.5 The conjunctiva may be diffusely reddened with serous or purulent exudates.5

Histologically, there is a fibrinous air sacculitis.5 Within the gastrointestinal tract, infection may result in diffuse mucosal necrosis and a moderate lymphoplasmacytic and histiocytic inflammatory infiltrate. C. psittaci can also result in marrow granulocytic hyperplasia.5 Within the liver, there is a mononuclear inflammatory infiltrate, which may be diffuse within the sinusoids.5 Many of the macrophages contain green-brown pigment consistent with bile pigment and/or hemosiderin.5 Multifocal to confluent hepatic necrosis is also common.5 In chronic disease, portal fibrosis and bile duct hyperplasia occurs.5 Urinary tract lesions include lymphoplasmacytic and histiocytic interstitial inflammation.5 The most consistent lesion seen in the spleen is histiocytosis.5 There is hyperplasia of histiocytes of the perivascular sheaths and a diffuse proliferation of plasma cells.5 Arthritis and nonsuppurative meningitis may also occur.5 There is typically conjunctival necrosis with a lymphohistiocytic infiltrate.5 Organisms can be recognized as basophilic punctuate structures within the cytoplasm of macrophages or hepatocytes.5 These organisms can be visualized with Gimenez stains and other special stains.5 Uncommon conditions associated with chlamydophilosis include otitis media, bursitis, and solitary nephritis.5

In cats, Chlamydophila felis, previously known as C. psittaci, is a primary conjunctival pathogen.7 Cats may also shed this organism from other nonocular sites as well.7 Infection with this organism is not documented in dogs.

To the authors’ knowledge, there are no known published cases of transmission of C. psittaci from parrots to other domestic animals.

**SALMONELLOSIS**

*Salmonella* spp are gram-negative bacteria that typically function as primary pathogens in parrots. *Salmonella* spp are members of the large family of Enterobacteriaceae and are widely distributed geographically. Some serotypes have been shown to penetrate the mucosal barrier, and noninvasive serotypes result in carrier states.5 *Salmonella typhimurium* is the most common psittacine isolate.5 Previously, the disease was a significant problem among wild-caught birds that were closely confined in quarantine stations.5 Currently, this infection is more likely to be identified in parrots from aviaries that have a significant rodent problem.5

Affected birds typically die suddenly due to septicemia.1 Clinically, affected birds develop profuse watery diarrhea, polyuria and polydipsia, dyspnea, pneumonia, depression, inappetance, and neurologic signs.1

The classic lesions of salmonellosis include hepatomegaly, splenomegaly, pneumonia, and a catarrhal to hemorrhagic enteritis.5 Gastrointestinal gross lesions include intestinal redness, exudation, and mucosal ulceration to varying degrees of severity.5 Gas or fluid distension of the gastrointestinal tract may occur, and there is generally focal soiling of the feathers of the vent consistent with diarrhea.5 Systemic infections with *Salmonella* spp may result in multifocal white nodules within the hepatic parenchyma (paratyphoid nodules).5
Histologically, intestinal necrosis with fibrin and heterophilic infiltrates are suggestive of a bacterial enteritis. There may be extension of the inflammatory process into the submucosa as well as the tunica muscularis, and crypt dilatation and abscess formation may be seen. Within the liver, paratyphoid nodules consist of randomly distributed nodular accumulations of histiocytes with fewer numbers of lymphocytes and plasma cells and variable necrosis. In most cases there is a multifocal to coalescing necrotizing splenitis with nodular aggregates of lymphocytes, macrophages, and heterophils. Additionally, salmonellosis may result in lymphohistiocytic meningitis, encephalitis, and myelitis, as well as osteoarthritis associated with bacterial septicemia. Bacteria are not always readily identifiable upon histologic examination, and additional special stains, bacterial culture, or molecular diagnostics may be necessary.

In dogs and cats, infections typically begin with ingestion of organisms in contaminated food or water with subsequent invasion of M cells in the Peyer patches. Clinical signs in these species are often associated with acute disease characterized by fever, malaise, anorexia, diarrhea, and vomiting. The diarrhea is often watery or mucoid and can be bloody. Most animals, though, are asymptomatic, and in rare cases systemic sepsis may occur.

To the authors’ knowledge, there are no known published cases documenting disease transmission of Salmonella spp between parrots and dogs and cats.

Yersinia pseudotuberculosis

Yersiniosis is rare in parrots and is typically transmitted via the fecal-oral route. Following ingestion, bacteria invade the intestinal mucosa by attaching to and penetrating the mucus layer overlying mucosal epithelial cells then adhere to and colonize intestinal brush border membranes. After penetrating the mucosa, bacteria are phagocytized by neutrophils and macrophages in the mucosa and submucosa. The organisms are able to survive within the cytoplasm of these phagocytic cells and are subsequently systemically spread.

In parrots, yersiniosis results in acute illness, diarrhea, and general ill health. Weight loss, decreased physical activity, and lethargy may be noted.

Gross lesions include hepatomegaly, discoloration of the liver, and miliary white spots throughout the liver, kidneys, and spleen. There may be variable associated foci of necrosis with associated accumulations of caseous material and necrotic debris.

Histologically, yersiniosis results in multifocal random foci of necrosis with variable associated accumulations of neutrophils and macrophages with fewer numbers of lymphocytes and plasma cells. Foci of necrosis may develop associated foci of mineralization and can be variable in size with some foci becoming quite large. Associated with foci of necrosis, there are large colonies of coccobacilli, which are suggestive of infection with Yersinia spp. Confirmatory bacterial culture is warranted in such cases.

Yersinia pseudotuberculosis can be ingested by cats eating infected rodents or birds. The bacteria then infect the gastrointestinal tract, liver, and lymph nodes. Clinically, affected cats demonstrate marked weight loss, diarrhea, anorexia, lethargy, jaundice, and mesenteric lymphadenomegaly.

To the authors’ knowledge, there are no known published cases documenting disease transmission of Yersinia spp from companion parrots to other companion animals in the household.
AVIAN INFLUENZA

Influenza viruses come in three groups: groups B and C affect humans and rarely birds, whereas group A affects birds and rarely humans. The primary reservoir is thought to be wild aquatic birds. Avian influenza has affected parrots, causing no illness to sudden death. There are no pathognomonic clinical signs for this condition, and clinical signs may vary depending on the age and species, presence of concurrent infectious disease, and environmental factors. Affected birds may demonstrate depression, diarrhea, or neurologic signs. Avian influenza virus should be considered as a differential diagnosis for clinical signs of gastrointestinal tract disease in psittacines. This virus is rarely seen in parrots, and most psittacine infections occur either in quarantine stations where it was contracted from other birds or has been caused experimentally. Avian influenza virus varies in its pathogenicity, and the serotypes that affect poultry are not usually a great risk to parrots.

Gross findings include dehydration and evidence of regurgitation such as occluded nares and feed material on the beak, head, and within the oral cavity. The crop may be full of feed material upon evaluation. Evidence of vent soiling consistent with diarrhea is also evident. Additionally, hemorrhagic enteritis is a common finding. Occasionally, a nonsuppurative meningoencephalitis is identified. Polymerase chain reaction with matrix gene as target is needed for confirmation of infection.

Dogs are susceptible to highly pathogenic avian influenza (H5N1) infection. Affected dogs typically develop fever but not fatal disease. Contact exposure experiments of influenza virus–infected cats with uninfected dogs did not result in interspecies transmission. There is a single report of a domestic cat infected with highly pathogenic avian influenza (H5N1) following ingestion of an infected pigeon carcass. Affected cats often demonstrate many of the following: necrotizing pneumonia with hyaline membrane formation, necrotizing and lymphoplasmacytic meningoencephalitis with gliosis, necrotizing myocarditis, necrotizing hepatitis, and necrotizing adrenalitis.

The potential for transmission of avian influenza from parrots to cats remains unsubstantiated. The only documented case of bird-to-cat transmission evidently required ingestion of an infected, nonpsittacine carcass. To the authors’ knowledge, there are no known published cases of such transmission from birds to dogs.

ASPERGILLOSIS

Aspergillus fumigatus is a noncontagious opportunistic angioinvasive fungus that results in acute and chronic respiratory and systemic disease. A fumigatus is a ubiquitous fungus found in nature and in the companion bird environment. Soil, moldy litter, moldy grain, and bedding material contaminated with feces are common sources of environmental Aspergillus isolates.

Like any infectious disease, the pathogenesis of aspergillosis is a function of interactions between the host, the agent, and the environment. Host factors commonly implicated include immunosuppression, stress, trauma, and toxicoses. Overall, it seems to be uncommon for immunocompetent birds to develop aspergillosis. Stress seems to be a significant contributor to immunocompetency in both wild and captive birds. Some common stressors may include capture, travel, importation, reproductive activity, overcrowding, and excessive human traffic. Host factors that are commonly associated with risk of aspergillosis include malnutrition (hypovitaminosis A), preexisting disease, and prolonged antibiotic or steroid treatments. Agent factors that can have a role in the development of disease include challenge with a large number of spores or increased invasiveness of the organism because of its
capability of being highly invasive given the right set of circumstances. Gliotoxins produced by *A. fumigatus* are immunosuppressive (cytotoxic to lymphocytes). Lipopolysaccharide of bacteria, inhaled along with spores of *A. fumigatus* in particulate matter, may reduce pulmonary function and the ability to clear debris from the respiratory tract, potentiating fungal colonization. *A. fumigatus* is unique among other members of the genus for its thermotolerance; it can grow at a wide range of temperatures (20°C–50°C). Environmental factors contribute to risk of disease through chronic exposure to a small number of spores or acute exposure to a massive number of spores. If the environment is conducive to fungal growth and spore production, the stage is set for increased exposure and increased likelihood of disease. Fungal growth and sporulation are promoted by environmental conditions of warmth and high humidity, followed by a drying period. Poor husbandry practices, allowing the accumulation of debris, discarded feed, feces, and litter support fungal growth. Poor ventilation increases the concentration of spores in the environment. Types of bedding frequently implicated in promoting the growth of *Aspergillus* spp include corncob, walnut shell, and eucalyptus leaves.

*Aspergillus* spp thrive in high humidity and warm temperatures. Immunosuppression, stress, and other factors related to confinement, poor husbandry, malnutrition, preexisting disease, and prolonged use of antibiotics and/or steroids predispose to disease in companion birds. Disease is common in overpopulated, poorly ventilated, and dusty environments. Infection is often a common sequel to other respiratory tract disease. Predominantly seed diets may result in vitamin A deficiency and subsequent squamous metaplasia of the oral and respiratory epithelium with secondary establishment of fungal growth. Gray parrots and Pionus species demonstrate increased susceptibility and are commonly represented in the literature. Localized infections of the nasal passages are common in Amazons. Parrots may carry spores in their lungs and air sacs without ill effect.

Infection develops because of inhalation of fungal spores or penetration of broken skin and eggshells, resulting in infection of developing embryos during the incubation process. Disease of the lower respiratory tract, lungs, and air sacs develop typically secondary to the trachea, syrinx, and bronchi being affected. Infections may spread from the respiratory tract to pneumatized bone or enter the peritoneal cavity by direct extension through air sac walls. Vascular invasion and embolism results in systemic spread of the organism.

Clinical disease may be acute or chronic. Acute onset typically results in fatal respiratory disease. Dyspnea, tail bobbing, cyanosis, lethargy, anorexia, polyuria and polydipsia, sudden death, tachypnea, open-mouth breathing, gurgling respirations and vomiting may occur. Weight loss is common. Chronic disease results in a change of behavior, reduced level of activity, decreased appetite, exercise intolerance, weight loss even with good appetite, respiratory compromise, and tachypnea and dyspnea late in disease. Biliverdinuria, polyuria and polydipsia, ascites, regurgitation, diarrhea, and abnormal droppings also occur in chronic disease. Ataxia and torticollis may occur with involvement of the central nervous system. Ocular disease may result in blepharospasm, photophobia, severe periorbital swelling, and conjunctival hyperemia. The time from onset of clinical signs to death may be less than 1 week to 6 weeks.

Acute disease results in whitish mucoid exudates in the respiratory tract and marked congestion of the lungs with thickening of air sac membranes and miliary foci of inflammation within the caudal thoracic and abdominal air sacs and peripheral lung fields. Abdominal enlargement due to ascites may also occur. Mycotic tracheitis results in granuloma formation within the trachea, syrinx, and primary bronchi with
resultant change in vocalization ability or obstructive airway disease. Chronic disease results in multiple nodules, which may coalesce into plaques and larger granulomatous lesions. Adhesions between air sac membranes, lungs, and abdominal viscera are common.

Air sacculitis is the most frequently encountered form of disease with extension to the lungs, most commonly with the posterior thoracic and abdominal air sacs affected. Chronic rhinitis and sinusitis, distension of the infraorbital sinus, and periorbital soft tissue swellings are common. Unilateral or bilateral nasal discharge that is serous to purulent with rhinoliths and oronasal granulomas and subsequent upper airway obstruction causing wheezing and secondary bacterial sinus infections is common.

Infection of the central nervous system with encephalitic and meningoencephalitic lesions may occur. Ocular disease is rare and develops secondary to preexisting upper respiratory infection. Corneal epithelial erosions and stromal necrosis with perforation of the cornea or panophthalmitis with functional loss of eye may develop.

Histologically, infections with *Aspergillus* spp result in multifocal granulomas primarily in the respiratory tracts of parrots, including the trachea, syrinx, bronchi, lungs, and air sacs. Lesions consist of multifocal-coalescing variable accumulations of epithelioid macrophages with associated multinucleated giant cells, occasional fibroblasts depending upon the degree of chronicity, and intralRESional fungal hyphae. Fungal hyphae are 2 to 4 μm in diameter with parallel cell walls and septae and demonstrate acute angle dichotomous branching. Histologically, fungal hyphae of *Aspergillus* spp are quite characteristic and can be used to diagnose aspergillosis with a degree of confidence.

*Aspergillus* spp are a common cause of upper respiratory infection in dogs. Canine sinonasal aspergillosis is characterized by colonization and invasion of the nasal passages and frontal sinuses by *A. fumigatus*. The disease primarily affects young to middle-aged dogs and is progressive. German Shepherds and Rottweilers are at increased risk. Colonization and invasion of the nasal mucosa results in destruction and necrosis of the nasal turbinates and often results in frontal sinus osteomyelitis. Facial pain, anorexia, sneezing, and mucoid to hemorrhagic nasal discharge and crusting are common. Life-threatening epistaxis and secondary meningoencephalitis may occur.

Disseminated infections typically involve multiple organ systems with no history of nasal or pulmonary involvement. *Aspergillus terreus*, *Aspergillus deflectus*, *Aspergillus flavipes*, and rarely *A. fumigatus* have been reported in association with these cases. Dissemination occurs following inhalation of spores and subsequent hematogenous dissemination. German Shepherds seem predisposed to systemic disease. Associated clinical signs are variable and based upon the organ system involved. Uveitis, ophthalmitis, and chorioretinitis may precede the onset of generalized disease. The most common clinical signs are bone pain, paraparesis, draining sinus tracts, weight loss, pyrexia, lethargy, muscle wasting, and fever. Infections with *Aspergillus* spp are often associated with immune suppression in the patient.

To the authors’ knowledge, there are no known published cases documenting disease transmission of aspergillosis from parrots to dogs and cats.

**Cryptococcus neoformans**

*Cryptococcus neoformans* var *neoformans* is an encapsulated saprophytic fungus with worldwide distribution and is often found in soils contaminated with bird droppings. This organism rarely causes disease in birds, but disseminated infections have been
reported in a green wing macaw, Moluccan cockatoo, thick-billed parrot, and North Island brown kiwi.14

Clinical signs are based upon location of the infection. Infections of the respiratory tract may result in dyspnea, tail bobbing, or increased respiratory noise, whereas infections of the digestive tract may result in weight loss, diarrhea, or the passing of whole seed in the feces. Finally, central nervous system infections may result in neurologic signs such as ataxia, disorientation, impaired mutation, or even seizures. Infections may sometimes be inapparent clinically and only diagnosed upon post-mortem examination.

Infection of the respiratory tract, digestive tract, and central nervous system results in necrotic granulomatous lesions and thick, pale gelatinous exudates.14 The lower temperature of the upper respiratory tract makes it more susceptible to infection.14 Upper respiratory tract involvement may produce facial granulomas that distort the rhamphotheca.14 Chronic rhinosinusitis has been reported in a Major Mitchell’s cockatoo due to Cryptococcus neoformans var. gattii with resultant encephalitis and meningitis causing blindness and paralysis.14

Histologically, infections with C. neoformans does not typically incite much of an inflammatory response because of the thick mucopolysaccharide capsule of the organism.5 When present, inflammatory infiltrates consist of macrophages with possibly associated multinucleated giant cells, lymphocytes, and plasma cells.5 Organisms may be intracellular or extracellular and are 5 to 7 μm in diameter with a 2- to 3-μm clear capsule, which stains positive with mucicarmine and a central 3- to 4-μm spherical basophilic body. Organisms are numerous and are readily identifiable with fungal stains such as periodic acid–Schiff or silver stains.

Infection occurs via inhalation of the yeast from the environment and is not considered contagious.16 Debris and droppings in and around avian habitats, especially pigeon habitats, contain large numbers of yeasts.16 Lesions consist of either granulomatous inflammation with few organisms or gelatinous masses of organisms with little inflammation.16 Cats are more commonly affected, and there is no breed, age, or sex predilection noted.16 Clinical findings are typically associated with upper respiratory, nasopharyngeal, cutaneous, ocular, or central nervous system involvement.16 The lungs are not typically affected. Upper respiratory signs include bilateral mucopurulent nasal discharge with or without blood. Proliferative lesions result in destruction of the nasal turbinates and tissue overlying the bridge of the nose.15 Oral ulcerations may occur, and granulomatous chorioretinitis variably with retinal detachment and meningoencephalitis is common.16

In dogs, disease typically affects young dogs under the age of 4.16 American Cocker Spaniels, Labrador Retrievers, Great Danes, and Doberman Pinschers are overrepresented.16 Central nervous system, upper respiratory, ocular, and cutaneous clinical signs are common.16 The brain is affected in most dogs.16 The upper respiratory tract is the second most common site with resultant upper airway stridor, nasal discharge, sneezing, epistaxis, or firm swellings over the bridge of the nose.16 Optic neuritis may result in blindness, and granulomatous chorioretinitis often occurs.16 The skin is less likely to be affected in dogs.16

To the authors’ knowledge, there are no known published cases documenting disease transmission for companion birds to dogs and cats.

**HISTOPLASMOSIS**

*Histoplasma capsulatum* is an infectious but not contagious fungal organism reported in poultry and zoo parrots only.14 The organism is soilborne and endemic in the eastern and central United States.14 *H. capsulatum* is commonly associated with fecal
material from pigeons and gallinaceous birds.\textsuperscript{14} The organism has the ability to grow within dirt substrates of enclosed aviaries and results in disease similar to \textit{Cryptococcus} \textit{spp}.\textsuperscript{14}

An initial pneumonia can progress to disseminated disease with formation of necrotic granulomas in multiple organs.\textsuperscript{14} Osteomyelitis and mineralized soft tissue granulomas within the shoulder and antebrachium in a Moluccan cockatoo have been reported.\textsuperscript{14}

Histologically, histoplasmosis results in multifocal granulomatous inflammation composed of nodular accumulations of epithelioid macrophages, multinucleated giant cells, and fewer lymphocytes, plasma cells, and heterophils.\textsuperscript{5} Variable numbers of intracellular fungal yeast may be identified within macrophages and multinucleated giant cells. Fungal yeasts are 2 to 4 $\mu$m in diameter and spherical with a 1- to 2-$\mu$m central basophilic body surrounded by a clear halo that develops because of artifactual shrinkage.\textsuperscript{5}

In dogs and cats, this infection typically originates in the lungs and potentially the gastrointestinal tract with subsequent dissemination to the lymphatics, liver, spleen, bone marrow, eyes, and other organs.\textsuperscript{17} Cats seem more susceptible to infection than dogs.\textsuperscript{17} This organism is not contagious, and infection is via ingestion or inhalation.\textsuperscript{17} Following phagocytosis by macrophages, where they grow as facultative intracellular organisms, hematogenous and lymphatic dissemination results in multisystemic disease.\textsuperscript{17} In dogs the lungs, gastrointestinal system, lymph nodes, liver, spleen, bone marrow, eyes, and adrenal glands are commonly infected.\textsuperscript{17} In cats the lungs, liver, lymph nodes, eyes, and bone marrow are commonly affected.\textsuperscript{17} Lesions consist of multiorgan granulomas with intrahistiocytic fungal yeasts.

To the authors’ knowledge, there are no known published cases of transmission of \textit{H capsulatum} from companion parrots to dogs and cats.

\textit{Cryptosporidium} \textit{spp}

\textit{Cryptosporidium} \textit{spp} are protozoa that function as an intracellular but extracytoplasmic parasite. Transmission occurs via the fecal-oral route. \textit{Cryptosporidium} infections usually result in disease in immunocompromised birds, more specifically enteritis in small birds such as cockatiels, budgerigars, and lovebirds.\textsuperscript{14} Upper respiratory tract disease is described in birds of prey.

Histologically, a mild-moderate lymphoplasmacytic proventriculitis with mucosal, glandular, and ductular hyperplasia has been documented.\textsuperscript{17} Organisms are described as intracellular but extracytoplasmic and are histologically identified as apical 1-$\mu$m spherical basophilic structures intimately associated with the surface of mucosal epithelial cells.

\textit{Cryptosporidium parvum} infection in dogs and cats is typically asymptomatic but has been associated with self-limiting diarrhea in cats.\textsuperscript{9} In immunocompromised animals, severe hemorrhagic diarrhea has been reported.\textsuperscript{9}

There are no published cases documenting disease transmission of \textit{Cryptosporidium} \textit{spp} from companion parrots to dogs and cats in the same household.

\textbf{GIARDIASIS}

\textit{Giardia} \textit{sp} is an intestinal protozoa identified in a number of different species. Morphologically identical cysts are identified in the feces of a number of different species including dogs, cats, and parrots.\textsuperscript{18} Despite their similar morphologic appearance, infectivity studies have demonstrated a lack of cross-infectivity among different species.\textsuperscript{18,19} \textit{Giardia lamblia} is typically isolated from mammals, whereas \textit{Giardia psittaci} is recognized in birds.\textsuperscript{18}
Giardia spp are an infrequent cause of diarrhea and have been associated with feather-destructive behavior in cockatiels.¹ Giardial organisms may be difficult to identify antemortem because they are typically found in the upper small intestine.¹ Giardiasis often results in minimal grossly visible changes at necropsy, although excessive fluid and mucus and mucosal hyperemia may be noted.⁵ Histologically, lesions range from nonexistent to villous atrophy and lymphoplasmacytic inflammation.⁵ Organisms may be found within the intestinal crypts but can extend the entire length of the villi.⁹ Giardiasis is described as very common in budgerigars, although most infected animals are asymptomatic.⁵ Infection is also common in cockatiels.⁵

In dogs and cats, diarrhea and abdominal discomfort are the most commonly recognized clinical signs.¹⁸ Infection is typically asymptomatic, and clinical signs range from mild self-limiting acute diarrhea to severe or chronic diarrhea associated with weight loss.⁹ Diagnosis is typically made by identification of the characteristic trophozoites or cysts in fecal samples, although ELISA antigen capture assays are also commercially available.

To the authors' knowledge, there are no known published cases documenting disease transmission of Giardia spp from parrots to companion dogs and cats.

**SUMMARY**

In conclusion, there are a number of bacterial, viral, fungal, and parasitic diseases that are presumed to be transmittable from companion parrots to dogs and cats. Such disease transmission has not been documented to occur; therefore, concerns for such transmission are apparently unwarranted, and pet caregivers should feel comfortable introducing a companion parrot into their household without increased risk of disease to their pet dogs and cats.

**REFERENCES**