

Infectious Disease in Captive Parrots

| Disease | Transmission | Clinical Signs | Diagnosis | Prognosis |
|---|--|---|---|--|
| Aspergillosis | <i>Aspergillus fumigatus</i> (mold) ubiquitous in the environment; infection in multiple birds or humans is likely contracted from the same source rather infected individuals. May be transmitted from parents to chick(s). Infection occurs from exposure to high concentration of mold spores or in food, water, soil, air, etc. –poor ventilation, lack of natural or UV light increases proliferation of spores, over-crowding, stress., compromised health increase risk of infection. | Dyspnea, ataxia, depression, lethargy, anorexia, raspy vocalizations or inability to vocalize, nasal discharge, ocular lesions/inflammation, organ granulomas/aspergellomas; death. Often occurs as a secondary, opportunistic infection in compromised or malnourished individuals living in conditions conducive to proliferation of mold spores. | Anti-body titer, PCR, U of Miami panel (antigen/antibody, protein electrophoresis, galactomannan), necropsy. | Generally poor. Due to the complexity of avian respiratory system and the potential for <i>Aspergillus</i> to colonize in organs, treatment may be prolonged, cause organ damage or be ineffective. Treatment consists of an extended course of itraconazole or terbinafine administered orally or through nebulization. |
| Highly Pathogenic Avian Influenza (Avian Flu, Bird Flu, AI, HPAI) | Feces, contaminated food/water, oral/nasal/ocular discharge, vectors, fomites. Fairly hardy enveloped virus can persist for hours or days at moderate temperatures, indefinitely in temperatures below freezing potentially zoonotic | Lethargy, depression, dyspnea, loss of appetite; oral/nasal/ocular discharge, respiratory distress, inability to stand, walk or perch; sudden death. | Necropsy, possibly testing. Current USDA/APHIS protocols should be followed. | Very poor, nearly 100% fatal. |
| Avian Tuberculosis (Avian TB, <i>Mycobacterium avium</i>) | Feces, contaminated food/water, some insects transmit – pathogens may survive in environment for mos. in some conditions, carriers may be asymptomatic hypothetically zoonotic | Mass under the skin or granuloma, chronic wasting, diarrhea, depression, lameness, swollen joints | Lab work may lead to diagnosis including culture, microscopic examination, endoscopy, biopsy – no one test is reliable – mycobacterium, which is the causative agent, is difficult to detect. Necropsy. | Most common in imported individuals and birds housed outdoors – treatment is possible with strict, long-term drug & isolation regimen – some birds do recover |

| | | | | |
|--|--|---|--|--|
| <p>Chlamydiosis (Psittacosis, Parrot Fever, Ornithosis or <i>Chlamydiophila psittaci</i>)</p> | <p><i>Clamydiophila psittaci</i> organism in feces and respiratory/ocular exudate – organism is hardy and can survive in dried feces and the environment for several mos. – carriers shed organism intermittently, carriers may be asymptomatic zoonotic</p> | <p>Labored breathing, loss of appetite, lethargy/depression, weight loss, diarrhea, nasal/ocular discharge, green/yellow urates, death</p> | <p>Culture, PCR test (requires blood, cloacal/choanal swabs), antibody titer (requires serum), necropsy</p> | <p>Generally very good. Can be treated effectively w/6-week course of doxycycline IM or PO. Re-infection/illness can occur with repeated exposure</p> |
| <p>Giardiasis</p> | <p>Giardia organism in feces, contaminated food/water zoonotic</p> | <p>Feather-picking, weight loss, bright yellow-green urates, nutritional deficiency, diarrhea</p> | <p>Various tests available but the parasite cannot be reliably detected; Diagnosis by response to treatment common; necropsy</p> | <p>Generally good. Treatment can be prolonged w/repetition necessary, esp. w/re-infection by a carrier. All birds in cage/flock should be treated. Treat with metronidazole or ronidazole, immune support.</p> |
| <p>Exotic Newcastle's Disease (Newcastle's, END)</p> | <p>Feces, contaminated food/water, oral/nasal/ocular discharge, vectors, fomites. Fairly hardy, can persist for hours, days, possibly longer under some conditions. potentially zoonotic</p> | <p>Often no signs are present. Some species may be carriers, i.e. Amazons; some species are highly susceptible, i.e., Cockatoos. Dyspnea, depression, paralysis or neuroloc impairment, loss of appetite, weight loss. Sudden death.</p> | <p>Testing, necropsy. USDA/APHIS testing protocol should be followed.</p> | <p>Poor for most species. Some species of Parrots may become ill and clear the infection or become asymptomatic carriers. Other parrot species may become acutely ill and die.</p> |
| <p>Psittacine Beak and Feather Disease (Pbfd, Psittacine Circovirus)</p> | <p>Psittacine Circovirus 1(PsCV1) or Psittacine Circovirus 2 (PsCV-2) in feather dander, feces, blood, parent feeding chick, possibly hen to egg – virus can survive in the environment for up to 6 mos. and possibly 2 yrs. vaccine pending FDA approval</p> | <p>Feather deformity/loss, degradation/deformity of beak – in chicks depression vomiting, feather lesions</p> | <p>PCR test (requires blood) and/or feather biopsy, necropsy</p> | <p>Many adult birds will develop immunity after exposure but may continue shedding. Others develop chronic illness resulting in death. Supportive care only – no available treatment</p> |
| <p>Polyomavirus</p> | <p>Feces, skin and feather dander, oral inhalation of aerosolized virus, possibly hen to egg, adult birds can be asymptomatic carriers – virus can survive in the environment vaccine available</p> | <p>Depression, anorexia, weight, loss delayed crop emptying, vomiting, diarrhea, subcutaneous bleeding, sudden death – mostly affects chicks but sometimes adults</p> | <p>PCR test (requires blood and cloacal swab), titer (requires serum), necropsy is commonly the only diagnosis</p> | <p>Chicks usually die w/in 12-48hrs. of signs – adults can become ill, some recover, some may become carriers. Supportive care only, no treatment available</p> |
| <p>Avian Bornavirus (ABV) & Proventricular Dilatation Disease (PDD) (or [Macaw] Wasting Disease) and</p> | <p>ABV: Enveloped not likely to survive outside host; transmission via contact with contaminated body fluids, exudates, feces or food. Individuals may recover from ABV, carriers may be asymptomatic, mates and flock members may be unaffected. Exact mode of transmission, incubation, prevalence, etiology is unknown. PDD: Etiology associated with ABV exposure/infection. Individuals</p> | <p>PDD/PDS: Depression, weight loss, anorexia, dehydration, vomiting, undigested food in feces, ataxia, impaired balance/flight, abnormal head movement, wing-flipping, toe-tapping,, seizures, and/or mild to severe motor deficit.</p> | <p>PDD: Conclusive diagnosis only w/necropsy. Autonomic, peripheral nerve ganglia are damaged by lymphoplasmacytic infiltrates and neuritis. Crop/(pro)ventricular biopsy demonstrating characteristic lesions, <i>lymphoplasmacytic ganglioneuritis</i>, may provide diagnosis prior to death. PCR tests for ABV are in research & clinical trials but are not reliable.</p> | <p>PDD: Clinical signs w/in days of infection w/death after 11 days to 7-10 yrs.? - supportive care only; some individuals may live months or years w/supportive care. Use of NSAID drugs for inflammation & antiviral drugs for ABV have been used experimentally. No reliable treatment available</p> |

| | | | | |
|---|--|---|--|--|
| <p>Proventricular Dilatation Syndrome (PDS)</p> | <p>may recover from ABV infection but PDD, an inflammatory condition of the CNS, may follow - or exist concurrent with - ABV infection. Environmental, genetic, immunological factors in progression from ABV to PDD. Some species appear more/less resistant.</p> <p>Other conditions may cause PDS: heavy metal toxicity, avian gastric yeast or fungal infection, foreign body obstruction, GI papillomatosis.</p> | | <p>PDS: cultures, radiographs, endoscopy, lead/zinc assay, chemistries, biopsy, etc.</p> | <p>PDS: With treatment for underlying causes birds may recover partially or completely.</p> |
| <p>Psittacine Herpesvirus (Pacheco's Disease, Psittacine Papillomatosis, Papillomavirus)</p> | <p>Psittacine herpesvirus shed in respiratory/ocular discharge – virus is fragile and does not survive in the environment long w/o a host – carriers shed virus intermittently and are usually asymptomatic vaccine available but efficacy against multiple strains is undocumented</p> | <p>Several strains that can cause disease. Virulent strain causes sudden death, anorexia, sinusitis, diarrhea (w/blood) yellow-green urates, excessive thirst/urination, conjunctivitis, central nervous symptoms - tremors, head-tilting, violent seizures</p> | <p>PCR test (requires blood, choanal/cloacal swabs), antibody titer (requires serum), necropsy.</p> <p>4 sero-types have been identified to date.</p> | <p>Highly virulent strains can cause death – some species are unaffected carriers, some individuals may recover completely – supportive care only, no reliable treatment available. Anti-viral drugs may be effective.</p> |
| <p>Psittacine Papillomatosis (Papillomavirus, Psittacine Herpesvirus)</p> | <p>Associated with Psittacine Herpesvirus; no Papillomavirus has been detected in birds with Papillomatosis. Close contact – preening, mating, feeding thought to be method of transmission.</p> | <p>Pinkish/whitish, dry, scaly wart-like lesions of oral cavity, cloaca, crop, esophagus, proventriculus, eye/lid, feet, toes.</p> <p>Possibly anorexia, vomiting, dilatation of ventriculus or proventriculus if lesions are located in the GI tract. May cause infertility or mimic prolapse in cloaca.</p> | <p>5% acetic acid will turn papillomatus tissue white but histologic examination of suspect lesions is necessary to confirm diagnosis. Endoscopy may be necessary to obtain biopsy from lesions in the esophagus, intestines, etc. Necropsy.</p> | <p>Physical removal of lesion through surgical excision, laser, radiocautery. Lesions often reoccur. Obstructive or severely ulcerated lesions of GI and reproductive tracts can cause death.</p> |