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Infectious and Parasitic Diseases of Captive Passerines

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The small passerines, canaries and finches, are social birds often bred and housed in flock aviaries. The aviary may be a mixed aviary housing different species or a breeding aviary concentrating on a single species. Multiple birds in contact with each other provide the means by which infectious disease can spread. Dietary and husbandry requirements vary for the species of passerines housed and can also influence disease outbreaks when they are less than optimal. Stress factors, including nutritional, husbandry (overcrowding, aviary maintenance), breeding, and the introduction of new birds, may play a significant role in disease outbreaks. An overview of viral, bacterial, fungal, and parasitic issues affecting passerines housed in aviaries will be addressed.

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Viral Diseases

Paramyxovirus (PMV) may cause high morbidity and mortality in a flock situation. African Silverbills (*Lonchura malabarica cantans*), Zebra Finches (*Poephila guttata*), and Gouldian Finches (*Chloebia gouldiae*) are commonly infected.¹⁻³ There are 9 serotypes of paramyxovirus, with serotype 1 causing Newcastle disease. Serotypes 1, 2, and 3 have been associated with disease in passerines, whereas serotypes 1, 2, 3, and 5 have been associated with disease in psittacines.^{4,5} Two groups of strains, strains of turkey and psittacine origin, represent PMV-3, with the psittacine strains causing disease in passerines and psittacines.

In passerines, the clinical signs of paramyxovirus include conjunctivitis, anorexia, yellow diarrhea, voluminous stools with undigested starch and fat (as a result of pancreatic insufficiency), dyspnea, and occasionally neurologic signs. Passerines may be carriers for months before the onset of clinical signs.⁴ Gross postmortem lesions may include pulmonary edema, pancreatic atrophy, or pale myocardium. Inclusion bodies may be found in the brain, pancreas, and heart. Serology to diagnose PMV-3 may be used

for an antemortem diagnosis; this test may cross react with PMV-1. Virus isolation with inoculation of chick embryos or cell cultures and identification by hemagglutination inhibition with antisera specific for PMV-3 is necessary to confirm the diagnosis. Pancreatitis in combination with the clinical signs is highly suggestive of this infection in these birds.

Avipoxvirus is a common problem in canaries but rarely in finches. It has also been documented in the wild House Finch (*Carpodacus mexicanus*).⁶ Three forms of the avipoxvirus are seen and may occur simultaneously.^{2,3,7} The *cutaneous* form may first present as conjunctivitis and blepharitis. These symptoms give way to development of yellow-to-brown papules which progress to vesicles that open, dry, and form a wart-like lesion. These lesions are found on the unfeathered portions of the skin (head, eyes, beak, nares, and feet). The *diphtheric* form results in the formation of a necrotic membrane covering the mouth and larynx. The *respiratory* form presents with acute onset of dyspnea, cyanosis, and ruffled feathers. Mortality may reach 100% within 3 days of the onset of clinical signs. A desquamative pneumonia resulting in airsacculitis, necrotizing bronchitis, and hemorrhagic lungs is usually present.⁷ In all 3 forms, Bollinger bodies (intracytoplasmic lipophilic inclusion bodies) are characteristic of the avipoxvirus and are usually present in the affected tissues. They may be visualized on histopathology with a cytologic (Wright's) stain.⁸ Mosquitoes, mites, and contact through traumatized epithelial surfaces transmit avipoxvirus from bird to bird. An attenuated-live vaccine for avipoxvirus is available and should be used in high risk situations as a pre-

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ventative measure 1 month before the onset of mosquito season.^{2,3,7,9} High-risk birds are those living in outdoor aviaries with a dense mosquito population. The vaccination should be repeated annually. Caution must be exercised when vaccinating during an outbreak to prevent the transmission of the virus during the handling and vaccination procedures. Canaries will develop immunity for 3 to 6 months following vaccination.⁷

Polyomavirus infections may cause high morbidity and mortality in finches. Adult finches may be asymptomatic carriers and exhibit intermittent shedding and production of infected young. Early embryonic death and acute mortality have been reported in 2- to 3-day-old nestling finches, as well as in fledgling and young adult finches.^{2,7,10,11} A chronic form of polyomavirus infection is seen with those fledglings that have survived the original infection. They often present with poor feather development and tubular-shaped mandibles. Gross necropsy and histopathologic findings are similar to those in psittacines and include perirenal hemorrhage, serosal or subserosal intestinal hemorrhage, liver hemorrhage, myocarditis, and splenomegaly.^{1,7} Affected tissues may exhibit basophilic intranuclear inclusion bodies containing polyomavirus antigen, which is identified by fluorescent antibody staining techniques. Viral-specific DNA probe analysis of cloacal and fecal swabs or of whole blood may provide an antemortem diagnosis of polyomavirus infection.⁷

Papillomavirus is common in finches, especially the European Goldfinch (*Carduelis carduelis*). This virus causes a slow-growing, epithelial wart-like proliferation on the skin of the feet and legs. The dorsal and plantar surfaces of the unfeathered legs may have smooth hard projections. Severe infections may result in the loss of digits. Often termed "tassel foot," this condition is often confused with *Knemidokoptes* mite infections.^{3,7}

Eastern equine encephalomyelitis (EEE) is a disease caused by a virus belonging to the Togaviridae family. This virus is endemic in the southeastern United States, Central, and South America. The virus, found in free-ranging reservoir-host birds, is most commonly transmitted by certain species of mosquitoes. Respiratory distress and posterior paresis has been documented in the Gouldian Finch.^{1,7} Western equine en-

cephalomyelitis and St. Louis encephalitis viruses have been isolated from the House Finch. Research has not confirmed if these birds may act as a reservoir host for transmission of this virus to outdoor aviary passerines.¹²

Cytomegalovirus may cause respiratory problems in the Lady Gouldian Finch and other Australian finches.^{2,7,13} Clinical signs of disease include depression, anorexia, dyspnea, and swollen, crusted eyelids. Affected birds often die 5 to 10 days after the onset of clinical signs.¹³ Histopathology reveals intranuclear inclusion bodies in the conjunctiva, liver, and esophageal mucosa.¹³

Influenza virus has been reported in finches and a coronavirus-like virus has been isolated from the trachea of canaries exhibiting mild respiratory signs.²

Bacterial Infections

Passerines generally do not have large numbers of organisms in their gastrointestinal tract. Individual passerines may be subject to a variety of bacterial infections, however, and this review focuses on those infections that may act as an infectious agent between birds.

Escherichia coli, *Enterobacter* spp, *Klebsiella* spp, and *Salmonella* spp—all members of the family Enterobacteriaceae—may cause primary or secondary infections in passerines. Concurrent infections with viral, parasitic, or fungal agents may be present and should be ruled out as inciting factors for bacterial disease. Clinical signs will vary depending on the organism present and may include diarrhea, septicemia, metritis, conjunctivitis, or rhinitis.^{14,15} Passerine nestlings with diarrhea ("sweating disease") are usually infected with an Enterobacteriaceae organism.

Salmonella typhimurium var *copenhagen* (paratyphoid) can cause granuloma formation in the liver, spleen, and rudimentary ceca, as well as ocular lesions and osteomyelitis in the finch and canary.^{3,7,16,17} This infection resembles *Yersinia pseudotuberculosis* both clinically and at necropsy.

Yersiniosis (*Yersinia pseudotuberculosis*) is seen in passerines during the winter months. Transmission may occur via food and water contaminated with feces. These bacteria may cause dyspnea, diarrhea, and death in canaries and finches. Necropsy findings may be identical to those found with *Salmonella* infections, including

granuloma formation in the liver, spleen, and intestines. Culture is necessary to differentiate these 2 organisms.

Campylobacter fetus var *jejuni* may affect both the canary and finch. The Bengalese or Society Finch (*Lonchura domestica*) is often a carrier without clinical signs. Tropical finches, especially nestlings and juveniles of the family Estrildidae (African, Asian, and Australian finches), are often susceptible to these bacteria. Pale, voluminous feces due to the presence of undigested starch are a common clinical sign.^{3,18}

Staphylococcus aureus, *S. epidermidis*, and *Streptococcus* spp infections are often seen. Clinical signs will vary and may include abscesses, dermatitis, pododermatitis, arthritis, sinusitis, conjunctivitis, omphalitis, and embryonic death.

Chlamydophila psittaci is relatively uncommon in the canary and finch. The infection rate appears to be between 0% and 1.4% each year.³ The clinical signs are nonspecific and may include fluffing, diarrhea, conjunctivitis, or nasal exudates.

Mycoplasma spp is difficult to isolate and is often not documented in those cases. Many of these birds respond to tylosin therapy. Wild House Finches captured for establishment of captive flocks have been found to be actively infected with *Mycoplasma gallisepticum*. These birds present with conjunctivitis.¹⁹ Flock management strategies are underway to develop preventive measures for *Mycoplasma* spp infections in these captive flocks.

Avian mycobacteriosis (*Mycobacterium avium-intracellulare* complex, *M. genavense*) can affect passerines. The classic formation of granulomas in the organs is seldom seen in these birds. The typical clinical presentation is sudden death; however, most birds examined are thin in appearance, which suggests chronic disease. Histopathology may demonstrate acid-fast bacilli within macrophages in the liver, spleen, lung, myocardium, proventriculus, ventriculus, and intestine. *M. avium* is shed in the feces, thus increasing the chance for exposure in the aviary situation.^{2,7,20}

Fungal Infections

Most fungal infections are a result of poor husbandry, including improper ventilation that permits accumulation of fungal spores, or of immunosuppression caused by many factors, including malnutrition and concurrent disease.

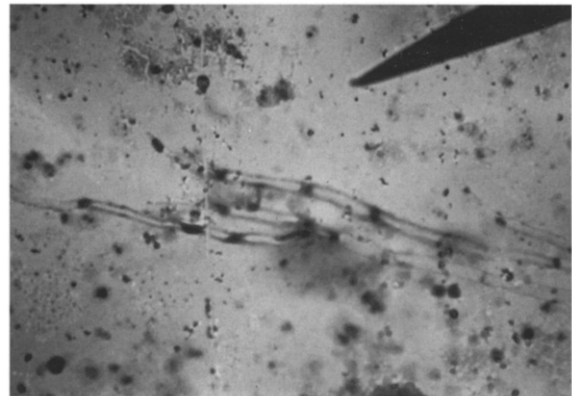


Figure 1. Gram's or cytologic stains of the oropharynx may reveal pseudohyphae suggesting tissue invasion by *Candida albicans*.

Candida albicans may be present in low numbers in healthy birds. Overgrowth may cause regurgitation, anorexia, crop stasis, and diarrhea. The organism has been demonstrated to invade the upper digestive tract mucosal lining and the koilin lining of the ventriculus.^{21,22} Finches seem to be predisposed to candidal invasion of the ventriculus.^{3,7,21,22} The presence of blastospores, pseudohyphae, and/or mycelia indicates tissue invasion (Fig 1).

Avian gastric yeast, originally known as "megabacteria," is a filamentous microbe staining positively for Gram's stain and periodic acid-Schiff (PAS) reagent and is found on the mucosal surface of the ventriculus at the junction with the proventriculus within the koilin layers.^{23,24} Clinical signs include chronic weight loss, dysphagia, vomiting, regurgitation, diarrhea, and death. Treatment protocols have been attempted with varying results. Currently, amphotericin B (Fungizone, Bristol-Myers Squibb Co., Princeton, NJ) used at 100 mg/kg orally every 12 hours is the most commonly used drug to treat this condition. A fluconazole (Diflucan, Roerig Pfizer Inc., New York, NY) regimen of 5-15 mg/kg orally every 12 hours or 50 mg/L in drinking water for up to 60 days has also been reported.^{23,24}

Aspergillus spp can cause respiratory signs in the canary and finch. Vomiting and diarrhea may be present with invasion of the gastrointestinal tract. Although not truly an infectious disease passed from one bird to another, aspergillosis may affect a large number of birds in an

aviary and exposed to improper ventilation and an accumulation of fungal spores.

Microsporium gallinae and *Trichophyton* spp can cause feather loss on the head and neck region and hyperkeratosis of the skin.⁷

Parasites

Protozoal infections in passerines are varied and include coccidiosis, atoxoplasmosis, cryptosporidiosis, toxoplasmosis, trichomoniasis, giardiasis, and cochlosomiasis.

Coccidiosis

Canaries may become infected with coccidian in the first few days of life but do not exhibit clinical signs until 2 months of age.^{2,3} *Isospora serini* and *Isospora canaria* have been isolated from the canary.²⁵ A disseminated infection may result from invasion of *I serini*, whereas *I canaria* usually results in the typical coccidian infection restricted to the intestinal epithelium. The duodenum may become edematous and hemorrhagic with trophozoites present in the affected duodenum. Wet mounts of the droppings may reveal large numbers of the oocysts.²⁵ Research in captive Nashville Warblers (*Vermivora ruficapilla*) demonstrated coccidial infections secondary to the immunosuppressive effects of a lymphoid neoplasm. Concurrent coccidiosis and lymphosarcoma has been described in captive passerines, but it is unlikely that the coccidia had a primary role in the lymphogenesis.²⁶ Control of coccidiosis includes strict hygiene and treatment with coccidiostat drugs. A regimen of sulfa-chlorpyridazine (Vetisulid, Solvay Animal Health Inc., Mendota Heights, MN) in the drinking water at 300 mg/L for 5 days, stopped for 2 days, and then continued for 4 treatment cycles may significantly reduce or totally clear fecal oocyst shedding for an extended period of time. This drug appears to affect only the intestinal stages of the parasite.²⁷ Toltrazuril (Baycox, Bayvet Bayer Corporation, Shawnee Mission, KS) at 75 mg/L drinking water for 2 days each week for 4 treatment cycles in canaries, and 12.5 mg/kg orally every 24 hours for 14 days in the Bali Mynah (*Leucopsar rothschildi*), has been used in attempts to affect the systemic stages of coccidial infections.^{27,28} Trimethoprim/sulfamethoxazole (Bactrim, Roche Alphaforma USPD Inc., Balti-

more, MD) and sulfadimethoxine (Albon, SmithKline Pfizer Inc., New York, NY) have also been used with limited success.

Atoxoplasmosis

Atoxoplasma parasites are most likely related to the coccidial genus *Isospora*. As stated previously, *Isospora* coccidia typically remain within the intestine. *Atoxoplasma* spp undergo an asexual life cycle in mononuclear blood cells and can spread hematogenously to the liver, lung, or spleen. Asexual and sexual cycles are also present in the intestinal mucosa.^{3,7,25,29-33} Because *Atoxoplasma* spp are commonly found in tissues other than the intestines, the genus was named and promoted as distinct in the early literature. However, *Isospora serini* does have an extraintestinal form in canaries, a fact that has led some researchers to believe that *I serini* is the coccidial parasite responsible for this disease.²⁵ Because *Isospora* spp are often very species-specific, it is possible that other species of *Isospora* may be involved in this disease syndrome in different passerine species.

Atoxoplasmosis has been reported in canaries, (*Serinus canarius*), sparrows (*Passer domesticus*), Greenfinches (*Carduelis chloris*), Bali Mynahs (*Leucopsar rothschildi*), and Bullfinches (*Pyrrhula pyrrhula*).³² Canaries aged 2 to 9 months are usually clinically affected. Mortality rates may approach 80%.^{3,33} Acute infections are the typical manifestation of this disease, and clinical signs may include huddling, ruffled feathers, diarrhea, coelomic distension due to hepatomegaly, neurologic signs, and death. Coccidial oocysts are rarely found in the feces or intestinal contents of dying birds because of the acute nature of the disease. Necropsy may reveal an enlarged thick liver with necrotic foci, an enlarged dark-red spleen, and an edematous duodenum with vascularization. Impression smears from the lung, liver, and spleen may reveal the parasite in the cytoplasm of the monocytes and lymphocytes.^{2,3,7} Adult carriers that are healthy in appearance may shed the oocysts in the feces for up to 8 months.³³ Transmission is usually by ingestion of oocysts from the environment. These oocysts are hardy, can remain infective in the environment for months to years, and are not routinely susceptible to disinfectants. Most treatment options are targeted toward the intes-

tinal stages of the organism and include the coccidiostatic drugs previously discussed. Toltrazuril (Baycox) therapy has been attempted to affect the systemic stages of this infection.^{27,28} Clinical outbreaks may be related to the stress of overcrowded aviaries, hygiene practices, and nutritional inadequacies.

Cryptosporidiosis

Cryptosporidiosis has been reported in the Gouldian Finch.^{1,34} Weight loss, depression, fecal pasting, pale or bulky droppings, and occasional feather loss at the head are all possibilities with this infection. Lymphoplasmacytic infiltration of the proventricular mucosa and upper gastrointestinal tract was demonstrated in cases where spherical protozoal organisms consistent with cryptosporidial organisms were detected on affected epithelial surfaces.¹ An antemortem diagnosis may be attempted with pooled fecal samples for flotation or acid-fast staining; however, intermittent shedding often yields false negative results. Postmortem diagnosis involves the use of histology, enzyme-linked immunosorbent assay testing of antigens and indirect immunofluorescent assays.¹ Treatment has been attempted with paromomycin (Humatin, Parke-Davis, Morris Plain, NJ) at an oral dosage of 100 mg/kg every 12 hours for 7 days, but results may vary.^{1,34}

Toxoplasmosis

Infections with *Toxoplasma* spp in the canary may become a problem if the oocysts excreted in the feces of cats contaminate the aviary or the bird's feed. The acute phase of infection presents with respiratory signs. Necropsy reveals a catarrhal pneumonia, myositis, hepatomegaly, and splenomegaly. Trophozoites may be identified on impression smears of affected lungs or histologic sections of affected tissues including the brain.³⁵ Birds that do not die from the acute infection often develop iridocyclitis, chorioretinitis, parophthalmia, and blindness several weeks after the original infection.^{2,3} Antemortem testing with an anti-*T. gondii* antibody titer has been attempted with variable results.³⁵ Treatment has been attempted with pyrimethamine (Fansidar, Roche Pharmaceuticals, Manati, Puerto Rico 00674) at 0.5 mg/kg orally every 12 hours for 14 to 28 days. Clinical signs possibly associated with *T. gondii* in an Alala Ha-

waiian Crow (*Corvus hawaiiensis*) resolved following the use of diclazuril, a benzeneacetonitrile anticoccidial agent, at an oral dosage of 10 mg/kg every 24 hours on days 0, 1, 2, 4, 6, 8, and 10.³⁵

Trichomoniasis, Giardiasis, and Cochlosomiasis

These flagellated protozoa inhabit the gastrointestinal tract and may be associated with clinical signs of regurgitation, gagging, diarrhea, and emaciation.^{2,7,36} *Trichomonas* infections are commonly seen in the Australian finches but are only sporadically reported in canaries.^{2,3} The crop and esophagus becomes thickened and opaque, and lined with a caseous material. A thick mucoid exudate may spread from the crop and oropharynx to the nares and sinuses. These birds may present with sinusitis and respiratory signs.^{3,37} *Cochlosoma* infections have been reported in a variety of finches. The most noted is the Gouldian Finch and the Bengalese Finch.³⁷ It is suggested that the Bengalese Finch, when used as a foster parent for the nestling Gouldian Finch, may pass the organism to the nestlings and cause a high mortality in these birds at ages between 6 and 12 weeks.^{1,7,37} Diagnosis may be made with wet-mount direct smears of warm feces or histopathology of the affected tissues. Treatment regimens for these organisms have included carnidazole (Spartrix, Wildlife Pharmaceuticals, Fort Collins, CO) 0.25 mg to 0.5 mg per bird as a single oral dose, metronidazole (Flagyl, Sidmark Laboratories Inc., East Hanover, NJ 07936 fish health formulation) at 1.5 g/gallon of drinking water daily for 7 days in canaries, or ronidazole (Ridzol 10%, Merck & Co. Inc., West Point, PA 19486) at 40 mg/L drinking water daily for 7 days.^{37,38}

Plasmodium

Plasmodium spp are intraerythrocytic parasites that are responsible for the disease avian malaria. Depending on the geographical location, canaries in outdoor aviaries may be susceptible to this devastating and often fatal disease.³⁹ The culicine mosquitoes (*Aedes*, *Anopheles*, and *Culex* spp) are the vectors responsible for spreading the organism. In some areas of the United States, malarial outbreaks may have a seasonal influence that relates to the mosquito season,



Figure 2. Plasmodium infections may cause an enlarged (black) liver that is apparent on necropsy.

primarily summer and fall. Clinical signs of disease will vary, depending on the stage of infection. Birds may present with some degree of exercise intolerance, lethargy, and anorexia. With time, the birds may also show signs of dyspnea, vomiting, weight loss, and seizures. Identification of the parasite within the red blood cell of peripheral blood smears provides an antemortem diagnosis. As the number of infected red blood cells increases, hemolytic anemia may become apparent. On necropsy, an enlarged dark (almost black) liver, enlarged spleen, pericardial effusion, and pulmonary edema may be seen (Fig 2). Many of the internal organs may contain schizonts, a characteristic stage of plasmodium.^{39,40} Without treatment this disease is usually fatal. Treatment may be accomplished with chloroquine phosphate (Aralen, Sanofi Synthelabo Inc., New York, NY 10016) and primaquine (primaquine phosphate, Sanofi) used in conjunction and formulated for drinking water. Recently, a regimen of mefloquine

hydrochloride (Lariam, Roche Pharmaceuticals, Manati, Puerto Rico 00674) at 30 mg/kg orally every 12 hours for the first day, every 24 hr for the next 2 days, and once weekly thereafter for 6 months, has been effective at eliminating the tissue stage of this parasite.

Other Parasites

Cestode infections are commonly seen in the insectivorous finches fed live insects as a food source. Zebra Finches, Firetails, and Parrot Finches (*Erythrura psittacea*) are prone to heavy infections.^{7,38,41} Signs include emaciation, diarrhea, and death due to intestinal obstruction. The proglottids may be passed intermittently in the feces. Treatment is with praziquantel or oxfendazole. Enclosures that are insect-proof will limit access to the intermediate host responsible for this infection.

Nematodes affecting the gastrointestinal tract of passerines are varied. Spiruroid nematodes such as *Dispharynx nasuta*, *Spiroptera incerta*, or *Acuaria skrjabi* inhabit the proventricular mucosa and koilin lining of the ventriculus. *Capillaria* spp most commonly have direct life cycles. These nematodes may cause white plaques in the mouth and pharynx and are often found in the intestines in passerines. Eggs have a bipolar plug and may be found in wet mounts from swabs of oral lesions or feces. Treatment options for all nematodes include ivermectin, fenbendazole, oxfendazole, piperazine, pyrantel pamoate, or levamisole hydrochloride. Treatment choice will depend on the parasite identified and the passerine species affected, as well as on safety and efficacy data.^{38,42}

Mite infections may affect passerines internally or externally. *Sternostoma tracheacolum* (air sac mite) inhabits the respiratory tract of passerines, especially the Gouldian Finch and canaries. The Bengalese Finch is not susceptible to the air sac mite, and use of these finches as foster parents for the Gouldian Finch will reduce transmission of these mites from the infected Gouldian parents to offspring.^{1,7} The mites are found in the trachea, syrinx, lungs, and air sacs. Clinical signs may include respiratory distress, wheezing, coughing, sneezing, nasal discharge, loss of voice, and gasping. This mite may cause airsacculitis, tracheitis, or focal pneumonia. *Dermanyssus gallinae*, the red mite, may cause high mor-

tality in fledgling and adult birds. This mite spends its day in the nest and crevices of the aviary and ventures out at night to bite the bird. Hallmark clinical signs are respiratory symptoms, general depression, and anemia. Treatment must include an insecticide for the bird and removal of the birds from the aviary for thorough cleaning and disinfection. *Ornithonyssus sylviarum*, the northern mite, is a problem in aviaries. This blood-sucking mite spends its life on the host. *Knemidokoptes pilae* is often called the scaly face or scaly leg mite. This mite may be carried latently after infection in the nest or by direct transfer from bird to bird. This mite tunnels into the stratum corneum layer of the skin to cause proliferative crusty lesions on the featherless parts of the face, legs, and vent. Scrapings of the affected area will often reveal the mite. This condition should be distinguished from "tassel foot" in the goldfinch, which is caused by a papillomavirus.^{3,7,43} Treatment for all mite infections may include the use of ivermectin topically or orally at 200 to 400 µg/kg once weekly for 3 to 6 weeks. Some situations may require dusting the infected birds with an insecticide or hanging a pest strip in a well-ventilated room that is not accessible to the birds. Caution must be used with dusts and sprays in the nestling.³

Conclusion

The infectious diseases mentioned in this text represent the most common examples seen in passerine aviaries. Certainly the stress factors of nutrition, husbandry practices, breeding, and exposure to multiple birds play an important role in the development of disease. It would not be uncommon for the ill bird to present with more than 1 problem. Viral infections with secondary bacterial infections are not uncommon. Fungal disease usually affects those birds that are immunosuppressed or suffering from some other ailment. Treatment regimes are based on the diseases present and often require thoughtful planning to treat a large number of aviary birds.

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