Chapter 18 Miscellaneous Herpesviruses of Birds

Synonyms

Inclusion body disease of falcons, owl herpesvirus, pigeon herpes encephalomyelitis virus, psittacine herpesvirus

Cause

Herpesviruses other than duck plague and inclusion body disease of cranes (see Chapters 16 and 17 in this Section) have been isolated from many groups of wild birds. The diseases that these viruses cause have been described, but their comparative taxonomy and host ranges require additional study. All of these DNA viruses are classified in the family Herpesviridae, but they belong to various taxonomic subfamilies. The mechanisms for transmitting avian herpesviruses appear to be direct bird-to-bird contact and exposure to a virus-contaminated environment. The virus is transmitted to raptors and owls when they feed on infected prey that serve as a source of virus exposure. The development of disease carriers among birds that survive infection is typical of herpesvirus. Stress induced by many different factors is often associated with the onset of virus shedding by carrier birds resulting in the occurrence and spread of clinical disease.

Species Affected

Herpesviruses infect a wide variety of avian species (Fig. 18.1). Many virus strains appear to be group-specific in the bird species they infect and sometimes only infect a limited range of species within a group. A few of these viruses infect a wide species range. For example, although duck plague only affects ducks, geese, and swans, it affects most species within this taxonomic grouping (see Chapter 16). However, inclusion body disease of cranes has been shown under experimental conditions to infect birds of several families (see Chapter 17). Viruses included in the falcon-owlpigeon complex resulted from experiments to cross-infect birds in these different groups. Herpesviruses as a group have been isolated from almost every animal species in which they have been sought and the viruses also cause disease in humans. In nature, the ability of these viruses to transmit to new hosts is governed by species behavior and host susceptibility to specific types of herpesviruses.

Distribution

To date, avian herpesviruses have been reported from North America, Europe, the Middle East (Iraq), Asia, Russia, Africa, and Australia and they are probably distributed worldwide (Table 18.1). Knowledge of their distribution in wild bird populations is limited to occasional isolated disease events in the wild, isolation of the viruses in association with other disease events, and from surveys of healthy birds. Unfortunately, there are few followup laboratory or field studies to expand information on those viruses that have been isolated. Most of the information on avian herpesvirus comes from disease events that affect or are found in captive flocks. The presence of this group of viruses in wild bird populations is probably more extensive than current data would indicate.

Seasonality

Little is known about the seasonality of disease caused by avian herpesviruses. Late spring appears to be the peak season for duck plague outbreaks (see Chapter 16), but less information about other herpesvirus infections of wild birds is available. The ability of this virus group to establish latent or persistently infected birds reduces the requirement for continual virus transmission to survive in an animal population (see Chapter 16, Duck Plague, and Chapter 17, Inclusion Body Disease of Cranes). Breeding season probably provides the best time of the year for bird-to-bird virus transmission in solitary species. Transmission of herpesviruses via the egg has been shown for some species, but more research is required to determine the importance of egg transmission for virus perpetuation. Seasonality probably plays a more important role for virus transmission in and among bird species that assemble for migration between summer breeding and wintering grounds.

Field Signs

The general signs of disease include depression of normal activity and sudden mortality in a group of birds. Respiratory distress may also be seen. Captive pigeons may show pronounced neurological signs such as extremity paralysis, head-shaking, and twisting of the neck.

Gross Lesions

Birds dying from infection with this group of viruses can have tumors (chicken and pigeon), hemorrhagic lesions (chicken, pheasants, ducks, cranes, peafowl, and guinea fowl), or, more commonly, hepatitis, and disseminated focal necrosis or visual areas of localized tissue death that appear as spots within the normal tissue in the liver, spleen, (Fig. 18.2) and bone marrow along with occasional intestinal necrosis. This broad array of lesions complements and extends those seen for duck plague and inclusion body disease of cranes (Chapters 16 and 17).



Figure 18.1 Relative frequency of disease from herpesvirus infections in birds of North America.

Table 18.1Geographical distribution of avian herpesvirus infections.

	Continents				
	North America	Europe	Africa	Australia	Other
Raptors					
Booted eagle		•			
Bald and golden eagles	•				
Common buzzard (Old World))	•			
Falcons					
Prairie	•				
Red-headed	•				
Peregrine	•				
Gyrfalcon	•				
Kestrels	•				
Owls					
Eagle owl		٠			
Long-eared owl		•			
Great horned owl	•				
Snowy owl	•	٠			
Pigeon	•	•	٠	•	Egypt
Ringed turtle dove	•				
Storks		•			
Cranes	•	•			China
					Japan
					Russia
Wild turkey	•				
Psittacines (several species)	•	•			Japan
Bobwhite quail		•			
Waterfowl (non-duck plague)	•		٠	•	
Black-footed penguin	•				
Passeriforms					
Exotic finches		•			
Weavers		•			
Finches, including canary		•			
Cormorants			٠		

Gallinaceous birds such as chicken, pheasants, peafowl, and guinea fowl raised in captivity have also been infected.



Figure 18.2 Herpesviruses can produce areas of tissue necrosis, appearing as white spots, such as in this peregrine falcon liver (**A**) and this great horned owl spleen (**B**).



Diagnosis

The primary methods for diagnosing herpesvirus as a cause of disease are virus isolation from infected tissues and finding, during microscopic examination of infected cells, the characteristic accumulations of cellular debris referred to as intranuclear (Cowdry type A) inclusion bodies (Fig. 18.3). These lesions are most often seen in the liver, spleen, and bone marrow. The virus can usually be isolated in chicken or duck embryo fibroblast tissue culture or in embryonated chicken eggs.

Control

Control actions warranted for outbreaks of herpesvirus infections are dependent upon the type of herpesvirus infection and the prevalence of disease in the species or populations involved (see Chapter 4, Disease Control Operations). Euthanasia of infected flocks should be considered for exotic viruses and viruses that are likely to cause high mortality within the population at risk. When depopulation is not appropriate because of the ubiquitous nature of the disease, or for other reasons, disease-control steps should still be taken. Sick birds, as well as those in the preclinical stages of illness, will be shedding virus into their environment; therefore, birds that are suspected of being infected should be segregated from other birds and quarantined for 30 days. Any birds noticeably ill should be isolated from the rest of the contact group. A high level of sanitation should be imposed and maintained for the full quarantine period where birds are housed. Decontamination procedures are needed to minimize disease transmission via virus that is shed in feces and by other means.

Dead birds should be removed immediately and submitted for disease evaluations. Standard bagging and decontamination procedures should be used to avoid off-site transfer of the virus. Personnel should follow good hygiene methods and should not have any contact with other birds for 7 days to prevent mechanically carrying contamination from the quarantine site.

Surviving birds should be tested for virus and virus specific antibody. All birds with antibody are probably virus carriers and they pose a risk as a source for future virus infection. Future use of these birds should take this into consideration. This is especially important when endangered species are involved and for wildlife rehabilitation activities because survivors of herpesvirus infections are potential sources for the initiation of new outbreaks and further spread of the disease.

Human Health Considerations

Avian herpesviruses have not been associated with any disease of humans.



Figure 18.3 Inclusion bodies (arrows) in liver cell nuclei of a great horned owl that died of herpesvirus infection.

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Supplementary Reading

- Burtscher, H., and Sibalin, M., 1975, *Herpesvirus strigis*: Host spectrum and distribution in infected owls: Journal of Wildlife Diseases, v. 11, p. 164–169.
- Graham, D.L., Mare, C.J., Ward, F.P., and Peckham, M.C., 1975, Inclusion body disease (herpesvirus infection) of falcons (IBDF): Journal of Wildlife Diseases, v. 11, p. 83–91.
- Kaleta, E.F., 1990, Chapter 22; Herpesviruses of free-living and pet birds, *in* A laboratory manual for the isolation and identification of avian pathogens, American Association of Avian Pathologists. p. 97–102.