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Herpesvirus strigis: HOST SPECTRUM AND DISTRIBUTION IN INFECTED OWLS

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Abstract: Herpesvirus strigis, a new species of the genus *Herpesvirus*, is a pathogen for several species of owls in the order Strigiformes. Natural infection has been observed in the Eagle Owl (*Bubo bubo* L.), Long-eared Owl (*Asio otus* L.) and Snowy Owl (*Nyctea scandiaca* L.) In addition the Little Owl (*Athene noctua* Scopoli) and Tengmalms Owl (*Aegolius funereus* L.) was experimentally infected. On the other hand the Tawny Owl (*Strix aluco* L.) and Barn Owl (*Tyto albo* Scopoli) proved resistant to a massive experimental infection. Of representatives from nine other orders of birds and mammals, only the Old World Kestrel (*Falco tinnunculus* L.) was found susceptible to this virus.

Distribution of viral antigen in various organs of infected owls, as determined by immunofluorescence and by quantitative virus assay, was in accordance with the occurrence of macroscopic and microscopic lesions.

INTRODUCTION

Since 1915 a viral disease of owls (Order Strigiformes) has been observed in Austria which has been designated hepatosplenitis infectiosa strigum (HSIS) owing to the presence of numerous necrotic foci found mainly in liver, spleen, and bone-marrow.[2] In 1936 the same disease syndrome was described in the USA[7] and since 1965 it has been repeatedly found in Germany.[8,10] The etiological virus can be propagated in chicken embryos or cell-cultures of various birds.[1,8,11] According to its morphological,[5] biological,[1] biochemical and biophysical properties[12] it belongs to the genus *Herpesvirus* and has been designated as *Herpesvirus strigis*.[1]*

As a contribution to the possible sources of infection for owls this paper lists animal species tested for susceptibility to *H. strigis*. In addition, the route of infection in the owl is followed by means of immunofluorescence and by quantitative virus assay.

* Genetive sing. from *strix* (lat.) = the owl.

MATERIALS AND METHODS

Virus

Strain x/42/63 of *H. strigis* isolated from an Eagle Owl with HSIS[1] was used for all experimental infections. Freshly harvested chicken chorioallantoic membranes (CAMs) with typical virus-induced lesions[8] were homogenized and a 1:10 suspension was prepared in phosphate buffered saline (pH 7.4) containing 12000 units of potassium-penicillin G and 10000 units of streptomycin-sulphate per milliliter. Supernatant fluids withdrawn after 5 min centrifugation at 1000 g were used as inocula for embryonating eggs and test-animals.

Test-animals

Birds recorded under "field cases" (Table 1) were submitted by hunters and zoos for post mortem examination. Living game birds came from commercial animal suppliers or from hunters. Chickens were obtained from a poultry farm known to be free from infectious

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laryngotracheitis, fowl pox and Newcastle Disease and belonged either to the commercial lines "Ledbreast x Pilch" or "Harko". Pekin ducklings were from a duck farm where duck viral hepatitis had never been observed. White mice belonged to the MF-strain. Guinea pigs were from the Veterinary University of Vienna laboratory colony.

Diagnostic procedures

Experimentally infected test-animals were clinically observed until death or for at least 2 weeks. Animals that did not die within this period were killed. Tissues from all dead animals, experimental as well as field cases, were examined for gross and microscopic lesions. Attempts were made to reisolate *H. strigis* in embryonating chicken eggs. The techniques were the same as described earlier.[3] A positive diagnosis was based on the characteristic macroscopic and microscopic lesions of HSIS[2] as well as on successful isolation of *H. strigis* on CAM.[3]

Virus-titration and immunofluorescence

Techniques of virus-titration on the CAMs of embryonating chicken eggs[1,3] and of immunofluorescence[4] have been described. Virus doses are expressed as embryo-infective-doses-50% per ml (EID_{50}/ml).

RESULTS AND DISCUSSION

The results of infectivity tests with *H. strigis* using animal species belonging to ten different orders are given in table 1. Included are necropsy results on 11 species of Strigiformes and one species of Falconiformes.

Spontaneous lethal infection by *H. strigis* was observed only in three species of Strigiformes, i.e. Eagle Owl, Long-eared Owl, Snowy Owl, and in two additional owls which could not be accurately identified. Experimental susceptibility to *H. strigis* was established in the Little Owl and the Tengmalms Owl. In addition, Great Horned Owls (*Bubo virginianus* Gmelin) have been shown by American authors[7,9] to be susceptible to both natural and experimental infection. On the other hand, the Tawny Owl and the Barn Owl, proved fully insusceptible when submitted to the same experimental infection. As their immune status had not been determined before exposure, one cannot say whether the individual owls were immune to *H. strigis* or whether there is a species resistance. Natural resistance, however, seems more likely because all members of a given owl species proved invariably either susceptible or insusceptible.

Only owls with a yellow or orange colored iris, including the Eagle Owl, Long-eared Owl, Snowy Owl, Little Owl, Tengmalms Owl, and Great Horned Owl, have proved to be susceptible, whereas two species with dark irises, i.e. Tawny Owl and Barn Owl, proved resistant. Perhaps this observation is of no significance but it may prove of interest should susceptibility to *H. strigis* infection be investigated further.

Among representatives of the other nine orders tested, only the Old World Kestrel proved experimentally susceptible to *H. strigis*. However, no spontaneous infection in this species has hitherto been observed in Austria.

In the USA, Maré and Graham[9] produced experimental infection with *H. strigis* in the Great Horned Owl, the Ring necked Turtle Dove (*Streptopelia risoria*) and the American Kestrel (*Falco sparverius* L.). On the other hand, these authors and Ward *et al.*[13] reported on a spontaneous herpesvirus infection in various species of Falconiformes i.e. the Prairie Falcon (*Falco mexicanus* Schlegel) the Red-headed Falcon (*Falco chiquera* Daudin) and the Peregrine Falcon (*Falco peregrinus* Tunstall.). The isolated herpesvirus proved experimentally pathogenic to the Prairie Falcon, the American Kestrel, and the Merlin (*Falco columbarius* L.) as well as to the Great Horned Owl and the Screech Owl (*Otus asio* L.) and to the Ring-necked Turtle Dove. Gross and microscopic lesions in all species, spontaneously and experimentally infected with either herpesvirus, were quite similar to those of HSIS. From these findings and from

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TABLE 1. Susceptibility to **Herpesvirus strigis** of several animal species belonging to different orders.

Order / Species	Field cases	Experimental data				
	number positive/ number examined	number of positive cases/total number exposed	number per age group	age	route of inoculation	dose EID_{50} log 10
1 2	3	4	5	6	7	8
Strigiformes						
Eagle Owl (*Bubo bubo* L.)	31/42	/	/	/	/	/
Long-eared Owl (*Asio otus* L.)	8/13	5/5	4	adult	i.m.	5.0
			1	7 weeks	i.m.	4.7
Snowy Owl (*Nyctea scandiaca* L.)	2/3	/	/	/	/	/
Little Owl (*Athene noctua* Scopoli)	0/4	3/3	1	adult	i.m.	4.8
			2	adult	aerosol	/
Tengmalms Owl (*Aegolius funereus* L.)	—	1/1	1	adult	p.o.	5.0
Tawny Owl (*Strix aluco* L.)	0/6	0/2	1	adult	p.o.	5.2
			1	adult	i.m.	5.0
Scops Owl (*Otus scops* L.)	0/11	/	/	/	/	/
Barn Owl (*Tyto alba* Scopoli)	0/4	0/2	1	adult	p.o.	5.0
			1	adult	i.m.	5.0
Pygmy Owl (*Glaucidium passerinum* L.)	0/2	/	/	/	/	/
Ural Owl (*Strix uralensis* Pallas)	0/2	/	/	/	/	/
Fish Owl (*Ketupa ketupa* Horsfield)	0/1	/	/	/	/	/
Owl species not accurately determined	2/4	/	/	/	/	/
Falconiformes						
Old World Kestrel (*Falco tinnunculus* L.)	0/6	1/1	1	juvenile	s.c.	4.7

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TABLE 1 — Continued

1 2	3	4	5	6	7	8
Galliformes						
			40	1 day	p.o.	4.7
Chicken			40	1 day	i.p.	4.7
(*Gallus gallus*	—	0/200	40	1 day	i.c.	2.7
domesticus L.)			40	4 day	i.m.	4.0
			40	4 day	i.t.	4.9
Columbiformes						
Pigeon			7	adult	i.p.	4.7
(*Columba livia*	—	0/15	3	juvenile	i.p.	4.7
Gmelin)			5	adult	i.m.	4.7
Anseriformes						
Pekin Duck			5	1 day	s.c.	4.7
(*Anas*	—	0/10	5	1 day	p.o.	4.7
platyrhynchos L.)						
Psittaciformes						
Budgerigar						
(*Melopsittacus*	—	0/1	1	adult	i.m.	4.6
undulatus Shaw)						
Caprimulgiformes						
Swift						
(*Apus apus* L.)	—	0/1	1	adult	i.m.	4.7
Ciconiiformes						
White Stork						
(*Ciconia ciconia* L.)	—	0/1	1	adult	i.m.	4.0
Passeriformes						
Blackbird						
(*Turdus merula* L.)	—	0/1	1	juvenile	i.m.	4.6
Hooded Crow						
(*Corvus corone*	—	0/1	1	adult	i.m.	5.0
cornix L.)						
Sparrow						
(*Passer domesticus* L.)	—	0/1	1	juvenile	i.m.	4.3
Rodentia						
			5	adult	i.p.	4.7
White mouse			5	adult	i.m.	4.7
(*Mus musculus* L.)	—	0/34	10	adult	p.o.	5.4
			14	newborn	i.c.	2.0
Guinea pig			2	adult	i.m.	4.7
(*Cavia aperea*	—	0/4	2	adult	i.p.	5.0
porcellus L.)						

— : not observed
/ : not determined
i.m. = intramuscular
p.o. = per os
i.p. = intraperitoneal
s.c. = subcutaneous
i.c. = intracerebral
i.t. = intratracheal

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results of comparative serological tests it was concluded that the falcon herpesvirus and *H. strigis* are closely related to each other.

A herpesvirus originally isolated by Cornwell *et al.*[6] from pigeons seems related to these two herpesviruses only serologically, whereas infectivity tests gave no corresponding results.

Table 2 shows the distribution of *H. strigis* antigen in experimentally infected owls as determined by the fluorescent antibody technique and by quantitative virus determinations on CAMs.

TABLE 2. Distribution of **Herpesvirus strigis**-antigen as demonstrated by immunofluorescence[1] and by titration[2] in experimentally infected owls.

Organ	Degree of immunofluorescence	Cells affected	Titer EID_{50}/0.1 ml log 10
Liver	+ + +	liver cells & Kupffer cells	2.5
Spleen	+ + +	mainly reticulum cells	3.0
Bone marrow	+ + +	mainly reticulum cells	1.5
Bursa of Fabricius	+ + +	mainly lymphatic tissue	/
Thymus	+ + +	mainly reticulum cells	/
Lung	+ + + (aerosol) + + (i.m.)	mesenchymal cells & bronchial epithelium	3.4
Trachea	+ + + (aerosol) — (i.m.)	epithelial & mesenchymal cells of propria mucosae	2.5
Heart	—	—	—
Kidney	+	epithelial & mesenchymal cells	2.5
Pancreas	+ or —	epithelial cells	/
Brain	—	—	—
Palate	+ +	mesenchymal & epithelial cells	1.5
Esophagus	+	suberosal mesenchym	/
Proventriculus	+ or —	mesenchymal cells of propria mucosae	/
Gizzard	—	—	/
Small intestine	+ + + (aerosol) + (i.m.)	mesench. cells of propria mucosae & of suberosa	/
Large intestine	+ +	mesenchymal cells of propria mucosae	2.3
Breast-muscle (inoculation-site)	+ + +	interstitial cells	/

[1] 4 Owls used: 2 Long-eared Owls injected intramuscularly (i.m.) with 5 log 10 EID_{50}
2 Little Owls exposed to aerosol (infective dose not determined)
[2] 1 Long-eared Owl used: injected i.m. with 5 log 10 EID_{50}
—: not observed /: not determined

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Distribution of viral antigen in owl tissues, as demonstrated by immunofluorescence, is quite similar to that observed in experimentally infected chicken-embryos.[4] Formation of antigen starts in the cell-nucleus with subsequent spreading to cytoplasm. Within organs, the antigen appears mostly in a spotted pattern, and rarely in a diffuse pattern. In infected birds viral antigen is limited to the sites of macroscopic and microscopic lesions.[2]

Affected cells are mainly mesenchymal. Fluorescent epithelial cells are rare and seem to be affected secondarily by virus spreading from adjacent mesenchymal cells. Viral affinity to mesenchymal cells also becomes apparent by the more intense affection of mesenchymal organs, e.g. spleen, bone marrow, bursa of Fabricius, and thymus, compared with the insignificant antigen content of mainly epithelial organs and tissues such as kidney, pancreas, proventriculus, and esophagus.

Results of fluorescent antibody titers are in remarkable accordance with quantitative virus assays done with individual organs of one experimentally infected owl.

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