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SHORT COMMUNICATIONS

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Herpesviral Inclusion Body Disease in Owls and Falcons is Caused by the Pigeon Herpesvirus (Columbid herpesvirus 1).

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ABSTRACT: A herpesviral disease of Rock Pigeons (*Columba livia*), called "inclusion body disease" or "inclusion body hepatitis," was first described in the 1940s. The disease involves hepatic and splenic necrosis with associated intranuclear inclusion bodies and occurs primarily in young squabs. A similar herpesviral disease occurs in falcons and owls. Serologic and restriction endonuclease digestion studies indicate that herpesviruses from pigeons, falcons, and owls are very closely related and that most reported cases of disease in falcons and owls involve prior documented or possible ingestion of pigeons. These findings led to the hypothesis that an endemic herpesvirus of pigeons may be causing disease in falcons and owls. In order to test this hypothesis, we sequenced a fragment of the herpesviral DNA polymerase gene from naturally infected owls, falcons, and pigeons with inclusion body disease collected between 1991 and 2006. Sequences from all three sources were almost identical, and we therefore propose that the usual agent of inclusion body hepatitis in owls and falcons is columbid herpesvirus 1.

Key words: Columbid herpesvirus 1, falcon, herpesvirus, inclusion body disease, inclusion body hepatitis, owl, Rock Pigeon.

A virus of Rock Pigeons (*Columba livia*) associated with inclusion body disease, and now tentatively called columbid herpesvirus 1 (CoHV-1) by the International Committee on Taxonomy of Viruses (ICTV), was first described in 1945 (Smadel et al., 1945) and was characterized in detail in a series of reports in the late 1960s and 1970s. Production of typical cytopathic effects in cell culture using a variety of avian cell lines (Cornwell and Weir, 1970a), intranuclear inclusion body formation when grown in 10-day-old chick embryos (Cornwell and Weir, 1970b), and detection by electron microscopy of intranuclear viral particles structurally consistent with a herpesvirus (Cornwell and Weir, 1967) suggested the etiology was a herpesvirus. Based on biologic criteria such as in vitro growth characteristics, clinical presentation, and pathology, CoHV-1 was originally classified as a member of the Betaherpesvirinae subfamily (Gunther et al., 1997). Subsequent sequence data indicated that the virus is actually most closely related to the alphaherpesvirus that causes Marek's disease, and therefore CoHV-1 has been reclassified as an unassigned member of the Alphaherpesvirinae subfamily (Ehlers et al., 1999).

The pigeon herpesvirus is carried subclinically and is shed by a potentially large percentage of adult pigeons (62.7% in a study in Belgium; Vindevogel et al., 1980, 1981). Disease in pigeons mainly occurs in squabs between ten and sixteen weeks of age (Callinan et al., 1979). Affected squabs are usually from uninfected parents or are debilitated for some other reason (Vindevogel et al., 1980). Clinical disease is not always evident prior to death but, if present, may involve depression, anorexia, conjunctivitis, oral and pharyngeal ulceration, dyspnea, and diarrhea with a duration of a few hours to as long as one week (Callinan et al., 1979). Lesions generally include one or more of the following: upper respiratory tract inflammation and ulceration, upper gastrointestinal tract inflammation and ulceration, hepatic necrosis, splenic necrosis, and renal necrosis (Cornwell and Wright, 1970). Eosinophilic intranuclear inclusion bodies are present

in parenchymal cells and sometimes in epithelial cells adjacent to areas of necrosis. A similar disease in raptors involving hepatic, splenic, and bone marrow necrosis with intranuclear inclusion bodies was first described in a great horned owl (*Bubo virginianus*) in 1936 (Green et al., 1936) and in a prairie falcon (*Falco mexicanus*) in 1971 (Ward et al., 1971). Since then, herpesviral inclusion body hepatitis has been reported naturally and experimentally around the world in a variety of Falconidae (Maré and Graham, 1973; Graham et al., 1975), and Strigidae (Burtscher and Sibalin, 1975). The herpesviruses in falcons and owls are currently listed by the ICTV as falconid herpesvirus 1 (FHV-1) and strigid herpesvirus 1 (StHV-1). The disease in raptors has a very similar clinical, gross, and histologic presentation as that found in pigeons (with the addition of bone marrow necrosis), but little is known about its transmission or epidemiology in raptors.

Historically, herpesviral isolates from pigeons, falcons, and owls have been described as distinct viruses. However, early serologic studies demonstrated varying degrees of cross-reactivity among the three viruses and no relationship with other avian herpesviruses (Maré and Graham, 1973). An early serologic study using cultured virus and indirect fluorescent antibody testing reported that FHV-1 and CoHV-1 were indistinguishable and that the StHV-1 was closely related but distinct (Potgieter et al., 1979). This conclusion was based on a lower antibody titer to StHV-1 in antisera raised against isolates of FHV-1 and CoHV-1. However, later studies using serum-neutralization were unable to differentiate the three viruses (Tantawi et al., 1983; Kaleta, 1990). Based on these studies, and the fact that most of the reported cases in raptors have been associated with documented or likely ingestion of pigeons, Kaleta (1990) suggested that they may actually be the same virus and that the virus is transmitted when raptors eat infected pigeons. Additional evidence supporting this hypothesis includes development of typical inclusion body hepatitis in recipient species after experimental transmission of virus isolates between owls, falcons, and doves, and similar restriction endonuclease patterns among the isolates. The initial transmission studies by Maré and Graham (1973) demonstrated classic inclusion body hepatitis in Great Horned Owls, an Eastern Screech Owl (*Otus asio*), and Ring-necked Doves (*Streptopelia risoria*) after inoculation with an isolate from a Prairie Falcon. Disease was also reproduced in American Kestrels (*Falco tinnunculus*) and Ring-necked Doves after inoculation with an isolate from a Great Horned Owl. Restriction endonuclease patterns for FHV-1 and CoHV-1, using the enzymes *Eco*RI, HindIII, and XbaI, are strikingly similar and are significantly different than the patterns for herpesvirus isolates from psittacines (Aini et al., 1993). Similar work comparing StHV-1 and CoHV-1 also demonstrated very similar restriction endonuclease patterns that were distinct from all other avian herpesviruses tested (Gunther et al., 1997).

In order to confirm herpesviral infection in falcons and owls with inclusion body hepatitis, and to further test the hypothesis that herpesviruses in pigeons, falcons, and owls are the same virus, we sequenced a fragment of the highly conserved DNA polymerase gene using degenerate PCR primers (VanDevanter et al., 1996). This sequence has been used extensively for mammalian and avian herpesviral identification and phylogenetic analysis (Ehlers et al., 1999; Pagamjav et al., 2005; Li et al., 2005). Tissue samples or virus isolates from cases of herpesviral inclusion body hepatitis submitted to the Washington Animal Disease Diagnostic Laboratory between 1991 and 2006 (Table 1), virus isolates obtained from the American Type Culture Collection (ATCC), and DNA polymerase gene sequences obtained from GenBank were used for analysis.

TABLE 1. Description of owls, falcons, and pigeons submitted to the Washington Animal Disease Diagnostic Laboratory between 1991 and 2006 with herpesviral inclusion body disease.

Animal ID	Species[a]	Date and location	Lesions	Other diagnoses	Sequence source
645	Rock Pigeon	1995 Washington	multisystemic necrosis with inclusions (including liver and spleen)	none	formalin fixed tissue
3315	Rock Pigeon	2002 Idaho	hepatic, intestinal, and cardiac necrosis	none	culture isolate
12049	Rock Pigeon	2006 Washington	hepatic, splenic, and crop necrosis with inclusions	none	formalin fixed tissue
469	Great Horned Owl	2000 Washington	hepatic and splenic necrosis with inclusions	*Mycobacterium avium* granulomas and mild pneumonia	culture isolate
9049	Great Horned Owl	1992 Washington	hepatic and splenic necrosis with inclusions	none	culture isolate
10027	Great Horned Owl	2004 Washington	hepatic, splenic, intestinal, and bone marrow necrosis with inclusions	none	fresh tissue
12351	Great Horned Owl	2004 Idaho	multisystemic necrosis with inclusions (including liver, spleen, and bone marrow)	trichomoniasis	fresh tissue
668	Gyrfalcon	1992 Washington	hepatic, splenic, and bone marrow necrosis with inclusions	none	culture isolate
806	Gyrfalcon/ Peregrine Falcon hybrid	1991 Idaho	hepatic, splenic, bone marrow, and adrenal necrosis with inclusions	none	culture isolate

[a] Rock Pigeon (*Columba livia*), Great Horned Owl (*Bubo virginianus*), Gyrfalcon (*Falco rusticolis*), Peregrine Falcon (*Falco peregrinus*).

Falcon, owl, and pigeon cases all originated from either Idaho or Washington. Clinical histories for the falcons, when known, were similar to previous reports of herpesviral inclusion body disease and included acute onset vomiting, anorexia, and depression. All falcons included in the study had a known history of feeding on pigeons. The owls were all wild birds and had no history other than severe depression in one bird (case 12351) that was observed shortly before death. Clinical histories for the pigeons, when available, included oral and ocular exudates and sudden death. Gross lesions in the falcons and owls were also consistent with previous reports and typically included pinpoint white foci throughout the liver and spleen of all birds and in the bone marrow of the falcons. Gross lesions in the pigeons were limited to ulceration of the oral cavity and upper respiratory tract. Microscopic evaluation of hematoxylin and eosin stained sections of formalin-fixed, paraffin-embedded tissues revealed that the white foci in the liver, spleen, and bone marrow consisted of areas of acute coagulative necrosis associated with a few macrophages and heterophils and comprised 30% to 75% of the organ parenchyma. Hepatocytes, macrophages, and occasionally other cell types around the margins of the areas of necrosis frequently had large, eosinophilic intranuclear inclusions that marginated the chromatin and were surrounded by a narrow, pale halo.

Areas of necrosis resulting in epithelial ulceration of the upper respiratory tract and digestive tract were also associated with similar intranuclear inclusion bodies in the pigeons and in one of the owls. When possible, tissue samples were frozen fresh and stored at −20 C or were immediately inoculated into chicken embryo fibroblast (CEF) cells or specific pathogen free (SPF) chicken embryos. All CEF cultures exhibited typical cytopathology and the SPF embryos all died of necrotizing inclusion body disease. Characteristic herpesviral particles were observed by electron microscopy in tissues or cell cultures from all of the cases.

For sequence analysis, viral DNA was extracted from either formalin-fixed tissues, fresh tissues, or cell culture monolayers infected with samples from the clinical cases or ATCC strains of virus. To extract nucleic acids from formalin-fixed tissues, five 10 μm sections were cut from the formalin-fixed paraffin-embedded tissues and incubated overnight at 60 C in 180 μl ATL Lysis buffer (Qiagen, Valencia, California, USA) and 4.4 mg/ml proteinase K. The top paraffin layer was removed by pipetting the melted wax and then by freezing at −20 C for 30 min and removing wax adhered to the tube walls. Nucleic acids were extracted from fresh tissue samples or cell cultures by homogenization and proteinase K digestion. The DNA was then purified from all samples using standard phenol-chloroform methods or by using the QIAmp DNA Mini-kit (Qiagen) according to the manufacturer's instructions. Consensus primer PCR, using three primers in the first reaction step and two in the second reaction, was performed as described (VanDevanter et al., 1996), with slight modifications described by Li et al. (2000). Amplification products were purified for cloning using the QIAquick gel extraction kit (Qiagen) and were cloned into pCR2.1 Topo Vector using the Topo TA Cloning Kit (Invitrogen, Carlsbad, California, USA) according to the manufacturer's instructions. Plasmid DNA was extracted using a QIAprep Spin Miniprep Kit (Qiagen). Insert presence and size was confirmed by PCR amplification and *Eco*RI restriction endonuclease digestion. Sequencing was performed by Amplicon Express (Pullman, Washington, USA). For analysis, the primer sequences were removed and the remaining 180 base pairs of the DNA polymerase gene were compared using ClustalW (Version 1.83, European Bioinformatics Institute, Cambridge, UK). A sequence alignment was generated that included consensus sequences from two to six clones from each of the clinical cases, ATCC isolates of CoHV-1 (VR-705) and FHV-1 (VR-709), and GenBank sequences of CoHV-1 from a pigeon (AF141890), an isolate from a Cooper's Hawk (*Accipiter cooperi*) with inclusion body disease (EF623994), an Indian Vulture (*Gyps indicus*) (AY571851), Gouldian Finches (*Erythrura gouldiae*) (AF520812), and an African Gray Parrot (*Psittacus erithacus erithacus*) (AY623124) (Fig. 1). Sequences from the pigeon cases (645, 3315, 12049), owl cases (469, 9049, 10027, 12351), and falcon cases (668, 806) have been submitted to GenBank as accession numbers, respectively: EF522960, EF522959, EF522958, EF522952, EF522953, EF522954, EF522955, EF522956, and EF522957.

All the sequences from the owls, falcons, the Cooper's Hawk, and the pigeons (including reference sequences) were identical, with the exception of single base differences in two of the pigeon sequences (cases 645 and 3315; Fig. 1). The few differences may be related to PCR errors, sequencing errors, or natural variation among different isolates. The sequences from pigeons, owls, and falcons were all considerably different from those of the other avian herpesviruses included in the study, with about 53% nucleotide identity with the finch sequence and 59% identity with the vulture and parrot sequences. There were also considerable differences between the isolates from the vulture, finch, and parrot. Based on these

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Pigeon-AF141890    GTCAACGGTCTGCTCCCGTGCCTGAACGTGGCCGCTACGGTGACGACCATCGGCCGGAAC  60
Pigeon-645         .........G.................................................  60
Pigeon-3315        .........G.................................................  60
Pigeon-12049       .........G.................................................  60
Pigeon-VR-705      .........G.................................................  60
Owl-469            .........G.................................................  60
Owl-9049           .........G.................................................  60
Owl-10027          .........G.................................................  60
Owl-12351          .........G.................................................  60
Falcon-668         .........G.................................................  60
Falcon-806         .........G.................................................  60
Falcon-VR-709      .........G.................................................  60
Hawk-EF623994      .........G.................................................  60
Vulture            AGT..T..C...T.G.....T...C.G..A..G..C.....T..C.....A..TA..G..  60
Finch              .G......CA....GT.C...A.AG.A..T..G..G..C..T...G....A...A..C.G  60
Parrot             A.G.....CA..A.G.....T..AG.G.....GT.C..T.....TG....A.....TG.A  60

Pigeon-AF141890    ATGCTGCTCGCCGTGCGCGATTACATACACCGGCGGTGG-GCGAGCTGGGACGCTCTGAT  119
Pigeon-645         ...........................................-..................  119
Pigeon-3315        ...........................................-......C...........  119
Pigeon-12049       ...........................................-..................  119
Pigeon-VR-705      ...........................................-..................  119
Owl-469            ...........................................-..................  119
Owl-9049           ...........................................-..................  119
Owl-10027          ...........................................-..................  119
Owl-12351          ...........................................-..................  119
Falcon-668         ...........................................-..................  119
Falcon-806         ...........................................-..................  119
Falcon-VR-709      ...........................................-..................  119
Hawk-EF623994      ...........................................-..................  119
Vulture            .....C..G..GAC......C..TG.C...GA.AA....-..T.CGCCC..ACT.T....  119
Finch              .....TT.GAG.AC.AAACGG......G.GGA.GAA...C.A..CTG.C......TCCGC  120
Parrot             .........AAAACCAAAC........G.AGA.AAT...C....ATACTC.A..CTCCGG  120

Pigeon-AF141890    TAAGGAATTCCCGCAGCTGGACGGGCACGCGAAGGCGGGGGAGGACTACTCCGTGTCGGTT  180
Pigeon-645         ...........................................................  180
Pigeon-3315        ...........................................................  180
Pigeon-12049       ...........................................................  180
Pigeon-VR-705      ...........................................................  180
Owl-469            ...........................................................  180
Owl-9049           ...........................................................  180
Owl-10027          ...........................................................  180
Owl-12351          ...........................................................  180
Falcon-668         ...........................................................  180
Falcon-806         ...........................................................  180
Falcon-VR-709      ...........................................................  180
Hawk-EF623994      ...........................................................  180
Vulture            GTC..CG.....AG.TAC..C..A.T.TCT..TT.GC..TAC.C.T...GAG.CAAAAA.C  180
Finch              G.GC.-C...G.---CG.AAT...T.C...-.GAAA.C.T-C.-GG.GG..TA.CAAC..C  174
Parrot             G..C.GT..TT.---C.C.TT...A.G.TT..G.AT.T.C-C.C.A.....T.....A..A  177
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FIGURE 1. Alignment of partial sequences for avian herpesviral DNA polymerase gene. The sequences given for the Rock Pigeons are those from cases 12049, 3315, 645, ATCC VR-705; the single GenBank sequence for CoHV-1 (AF141890) from a pigeon; and an isolate from a Cooper's Hawk listed as CoHV-1 (EF623994). The sequences given for the owls are from cases 469, 9049, 10027, and 12351. The sequences given for the falcons are from cases 668, 806, and ATCC VR-709. The sequences from the Indian Vulture, Gouldian Finch, and African Gray Parrot are from GenBank accessions AY571851, AF520812, and AY623124, respectively. The GenBank pigeon sequence (AF141890) is provided as the basis for comparison. Dots indicate nucleotides with identity to the pigeon sequence; letters indicate nucleotides that are different from the pigeon sequence.

sequencing results, we conclude that: 1) previously and currently described pigeon, falcon, and owl herpesviruses are the same virus; 2) this virus is distinct from other known avian herpesviruses; 3) most or all cases of herpesviral inclusion body disease in owls and falcons is due to infection of these aberrant hosts with CoHV-1; and 4) feeding of pigeons (unless known to be free of CoHV-1) to falcons and owls should be avoided.

A herpesvirus isolated from tracheal swabs collected from healthy eagle nestlings and an isolate from two eagles with lesions consistent with herpesviral inclusion body disease have been reported (Docherty et al., 1983; Ramis et al., 1994). Unfortunately, no restriction endonuclease or sequence data is available and the identity of these viruses and their relationship to other avian herpesviruses is unknown. However, a recent sequence submission to GenBank (EF623994) of an isolate from a Cooper's Hawk with inclusion body disease suggests that accipitrids may also be susceptible to CoHV-1-induced disease. No other herpesviruses have been reported from members of the Falconidae or Strigidae families, and thus, there are no currently known naturally occurring herpesviruses of falcons or owls. We therefore propose that, in the absence of proof to the contrary, the herpesviruses in owls and falcons that cause inclusion body hepatitis be referred to as columbid herpesvirus 1.

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