

*Original Contribution*

# Expansion of an Exotic Species and Concomitant Disease Outbreaks: Pigeon Paramyxovirus in Free-Ranging Eurasian Collared Doves

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**Abstract:** Eurasian collared doves (*Streptopelia decaocto*) have expanded their range across the United States since their introduction several decades ago. Recent mortality events in Eurasian collared doves in Arizona and Montana, USA, during the winter of 2009–2010 were the result of pigeon paramyxovirus (PPMV), a novel disease agent. The first instance of mortality by this emerging infectious disease in this species occurred in Florida in 2001 with subsequent disease events in 2006 and 2008. Full diagnostic necropsies were performed on carcasses from the three states. PPMV was identified by RT-PCR and virus isolation and was sequenced to the V1b genotype of avian paramyxovirus-1 (APMV). Other APMVs are common in a variety of free-ranging birds, but concern is warranted because of the potential for commingling of this species with native birds, virus evolution, and threats to domestic poultry. Improved surveillance for wildlife mortality events and efforts to prevent introduction of non-native animals could reduce the threat of introducing new pathogens.

**Keywords:** pigeon paramyxovirus, Eurasian collared doves, Florida, Montana, exotic, Newcastle disease

## INTRODUCTION

Geographic expansion by an exotic species can introduce novel pathogens to susceptible hosts, such as the distribution of chytrid fungus due to movement of infected *Xenopus laevis* frogs (Weldon et al. 2004). Eurasian collared doves (ECDO, *S. decaocto*) have colonized the western USA and outbreaks of pigeon paramyxovirus (PPMV) in ECDO

were observed during 2009–2010, which could indicate an emerging infectious disease. Here, we will discuss the origins of PPMV and its relationship to other avian paramyxoviruses (APMV), the expansion of ECDO into the continental United States, mortality events in Florida, Arizona, and Montana USA, and potential ramifications of PPMV in both wild and domestic birds.

Panzootic outbreaks of PPMV in pigeons (Columbiformes) were first recognized in the Middle East where the virus was isolated in Iran in 1978 (Kaleta et al. 1985). In the 1980's, outbreaks of PPMV with low morbidity/mortality

were reported in feral pigeons in eastern Europe (Biancifiore and Fioroni 1983) then spread to pigeons and Eurasian collared doves in Italy (Terregino et al. 2003), and into racing and feral pigeons in western Europe (Ujvári et al. 2003). The disease was identified as a risk to domestic birds when an outbreak in pigeons in 1984 led to 22 cases of Newcastle disease in chickens in the United Kingdom through fecal contamination by infected pigeons (Aldous et al. 2004). Newcastle disease is a virulent APMV reportable to the World Organization for Animal Health (OIE, <http://www.oie.int/animal-health-in-the-world/oie-listed-diseases-2011>). In 2001, Canada had a similar outbreak of PPMV in commercial layer chickens due to feed contaminated by feces of PPMV-infected pigeons (Toro et al. 2005), and infected pigeons were also identified in Nova Scotia in 2001 (Daoust and McBurney 2001). Infections of PPMV in poultry were reported in France in 2010 and Sweden in 2011 with suspicions of wild birds entering poultry houses ([http://vla.defra.gov.uk/reports/docs/rep\\_survrep\\_qtlyw0111.pdf](http://vla.defra.gov.uk/reports/docs/rep_survrep_qtlyw0111.pdf)). Australia's first detection of PPMV occurred in 2011 in racing and show pigeons (ProMed, <http://www.promedmail.org/>, 9 Sept 2011). Currently, 236 species of free-ranging birds (pet, domestic, and wildlife) are known to be susceptible to APMV infection (Kommers et al. 2003), including apparently healthy ducks (Hinshaw et al. 1985), and PPMV is considered endemic in domestic pigeons in Europe (Kaleta 1992). In the United States between 2000 and 2007, one PPMV serotype 1 isolate was recovered from a mourning dove (*Zenaidura macroura*) in Texas out of more than 5,000 bird carcasses tested (Kim et al. 2008).

PPMV is a single-strand RNA virus in the family Paramyxoviridae. There are ten serotypes of APMV; PPMV is classified in the APMV-1 class, along with Newcastle disease virus (NDV). PPMV-1 is an antigenic variant of NDV with unique monoclonal antibody-binding profiles (Kommers et al. 2002; Ujvári et al. 2003) and now constitutes a distinct sublineage within this genome with two separate groups: VIb1 and VIb2 (Kim et al. 2008). PPMV-1 appears to have originated from multiple events of chicken-to-pigeon transmission of NDV genotype VI (Ujvári et al. 2003). PPMVs isolated from pigeons all had F-protein cleavage sites that would put them in the virulent pathotype ( $^{112}$ RRQKRF $^{117}$ ) to chickens (Dortmans et al. 2009). However, chickens inoculated with PPMV-1 had some neurological signs, but no obvious clinical disease and no mortality within 14 days (Kommers et al. 2002). Virulence increased in successive passages of PPMV-1 isolates, but did not cause mortality in poultry. Some depression and



**Fig. 1.** Map of the United States with Eurasian collared doves reported by state (dark). Data from [Feederwatch.org](http://Feederwatch.org) as of November 2011.

nervous signs: tremors, paralysis, and birds sitting on their hocks, were observed, and histological lesions were present in the heart, bursa, lymphoid organs, and brain (Kommers et al. 2001), which could potentially lead to developmental problems (Kommers et al. 2002). In this respect, PPMV-1 was like most other APMVs in which birds are inapparent carriers (Coffee et al. 2010).

Eurasian collared doves were introduced to New Providence Island, Bahamas in 1974 via release of fewer than 50 doves that increased to 10,000 over the next 13 years. Between the late 1970's to early 1980's, ECDO colonized southeastern Florida and the Florida Keys. The National Audubon Society's Christmas Bird Count identified ECDO in Key Largo in 1981, but the first published report did not appear until 1986 (Smith and Kale 1986) because of apparent misidentification as turtle doves (*S. roseogrisea*). Since that time, ECDO undertook rapid geographic range expansion across most of the country (Fig. 1).

## METHODS

### Field Investigation

In October and November 2009 in Buckeye, Arizona, two residential locations reported morbidity and mortality in ECDO. The first location operated a commercial operation with psitticines and galliformes on the property and became concerned when two to three dead ECDO were observed in a day. The nearby area trees and power lines were used for roosting by doves at night. Affected doves were reported as lethargic, ataxic, and reluctant to fly.

Mortality increased to an estimated 35 doves per day over a 4-week time-span and then declined. The second location, 16 km west, reported birds were falling out of trees and unable to fly. Up to six carcasses were recovered per day from this location over the course of 6 weeks. A total of eight doves, from both locations, were submitted for diagnostic testing from the Arizona Game and Fish Department.

Apparently ill and dead birds were observed in the towns of Belgrade and Three Forks (35 km apart), Montana beginning in December 2009 and January 2010, respectively, and ending in late January 2010. Belgrade had a small population of ~50 ECDO concentrated in neighborhoods while Three Forks had an estimated 100 ECDO. Affected birds were lethargic, sitting upright, and not moving when approached. Montana Fish, Wildlife and Parks submitted six ECDO for diagnostic testing the course of the event.

Mortality events involving ECDO had previously occurred in Florida in 2001, 2006, and 2008. In 2001, affected and dead ECDO were observed in Panhandle coastal areas of Santa Rosa, Okaloosa, and Bay counties, later extending south to the Tampa area and Florida Keys. Mortality began in late August or September, peaking in October, and an estimated 5,000 doves died. No other species were involved. Apparently healthy mourning doves were observed in the same area and were using bird feeders along with affected ECDOs. Doves were lethargic and allowed humans to approach. Death usually occurred within 1 to 2 days of clinical onset. No additional PPMV outbreaks were identified until late August 2006, when 20 “ring-necked” doves (potentially misidentified ECDO) were found dead or dying over a period of 2 to 3 months in Dade County. Birds were reported to be convulsing, and a toxin or paramyxovirus was suspected. In 2008 in Florida, an estimated 30–40 ECDO were found dead over a period of 3 to 4 weeks in late June into July; no other species were affected. Mortality was concentrated around bird feeders and bird baths around the Florida Keys.

### Diagnostic Testing

The US Geological Survey—National Wildlife Health Center (NWHC), Madison, Wisconsin, USA collects information on wildlife mortality epizootics in the US in a nationwide database. Wildlife disease outbreaks are reported directly by state and federal wildlife management officials or other news sources. Diagnostic specimens from

Arizona (Cases 22835 and 22863) and Montana (Case 22893) were submitted to NWHC and other referral laboratories for diagnostic evaluation. Carcasses from Florida were examined by the University of Florida, College of Veterinary Medicine (Gainesville, FL) and Kissimmee Animal Diagnostic Laboratory (KADL, Kissimmee, FL). Refrigerated or frozen ECDO carcasses from reported die-offs were shipped by overnight courier. Diagnostic necropsies were performed, and tissues were processed using standard diagnostic laboratory methods for histology, parasitology, bacteriology, toxicology, and virology. A complete set of tissues from each bird was examined for histology, possibly including: brain, esophagus, proventriculus, ventriculus, intestine, heart, liver, kidney, spleen, pancreas, trachea, bursa of Fabricius, thymus, adrenal gland, kidney, skeletal muscle, peripheral nerve, eye, skin, and ovary. Tissues were fixed in 10% neutral buffered formalin before being embedded in formalin, sectioned at 5  $\mu$ m, and stained with hematoxylin and eosin. Liver and intestine were cultured for aerobic bacteria using standard methods (Meteyer et al. 1997). Toxicological testing was performed by gas chromatography/mass spectrometry at the Diagnostic Center for Population and Animal Health, Michigan State University, East Lansing, Michigan. Brain cholinesterase was determined by enzyme activity before and following incubation of the brain tissue at NWHC following the Ellman protocol (Nostrandt et al. 1993).

Virus isolation in embryonating eggs was conducted with fresh tissue or cloacal swabs according to the methods described by Alexander (1980). Allantoic fluid from each egg was tested for the presence of hemagglutinating viruses using chicken and turkey red blood cells. Hemagglutination-negative samples were passaged at least once more and re-tested before the original samples were considered negative. APMV-1 were identified from hemagglutination-positive samples by RT-PCR. Viral RNA extracted with the Applied Biosystems MagMax AI/ND 96 RNA extraction kit (Ambion, Austin, Texas, USA) according to manufacturer’s instructions. RT-PCR was performed using the US Department of Agriculture (USDA)-validated matrix and fusion gene primers according to Wise et al. (2004). Further characterization of APMVs was conducted at USDA—National Veterinary Services Laboratory (NVSL), Ames, Iowa, USA. The sequence of the fusion protein cleavage site was determined using Applied Biosystems BigDye sequencing method (Carlsbad, California, USA). Phylogenetic analysis was performed using the Neighbor-Joining method (Saitou and Nei 1987) as implemented in Mega 4.0

(Tamura et al. 2007). In brief, the sequences were aligned using ClustalW (Thompson et al. 1994), and Neighbor-Joining analysis was done using the amino acid substitution model with Poisson distribution and assuming uniform rates among sites. The percentage of replicate trees in which the associated taxa clustered together in the bootstrap test (1,000 replicates) is shown next to the branches (Felsenstein 1985). The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. Genetic group classification was as per Kim et al. (2008). We compared our PPMV genetic sequences from others derived from GenBank (<http://www.ncbi.nlm.nih.gov/genbank/>).

Immunohistochemical detection of NDV antigens was performed at Prairie Diagnostic Services, Inc. (Saskatoon, Saskatchewan, Canada), on fixed brain, kidney, liver, and spleen (NWHC case 22835, 1 bird) using an avidin-biotin complex technique adapted for an automated slide stainer as previously described (Kuiken et al. 1999) with a few modifications. In brief, heat-induced epitope retrieval was performed using sodium citrate buffer and nonspecific binding was blocked using a commercially available blocking kit (Avidin/Biotin Blocking Kit, Vector Laboratories Inc., Burlington, Ontario, Canada). The primary antibody (chicken polyclonal antiserum to NDV, Spafas, Preston, Connecticut, USA) was used at 1:4,000 and 1:8,000 dilutions and incubated overnight at 4°C. Binding of the primary antibody was detected using biotinylated goat anti-chicken immunoglobulins and an avidin-biotin immunoperoxidase complex reagent (Vector Labs Inc., Burlington, Ontario, Canada), with 3,3'-diaminobenzidine tetrahydrochloride (DAB) as the chromogen.

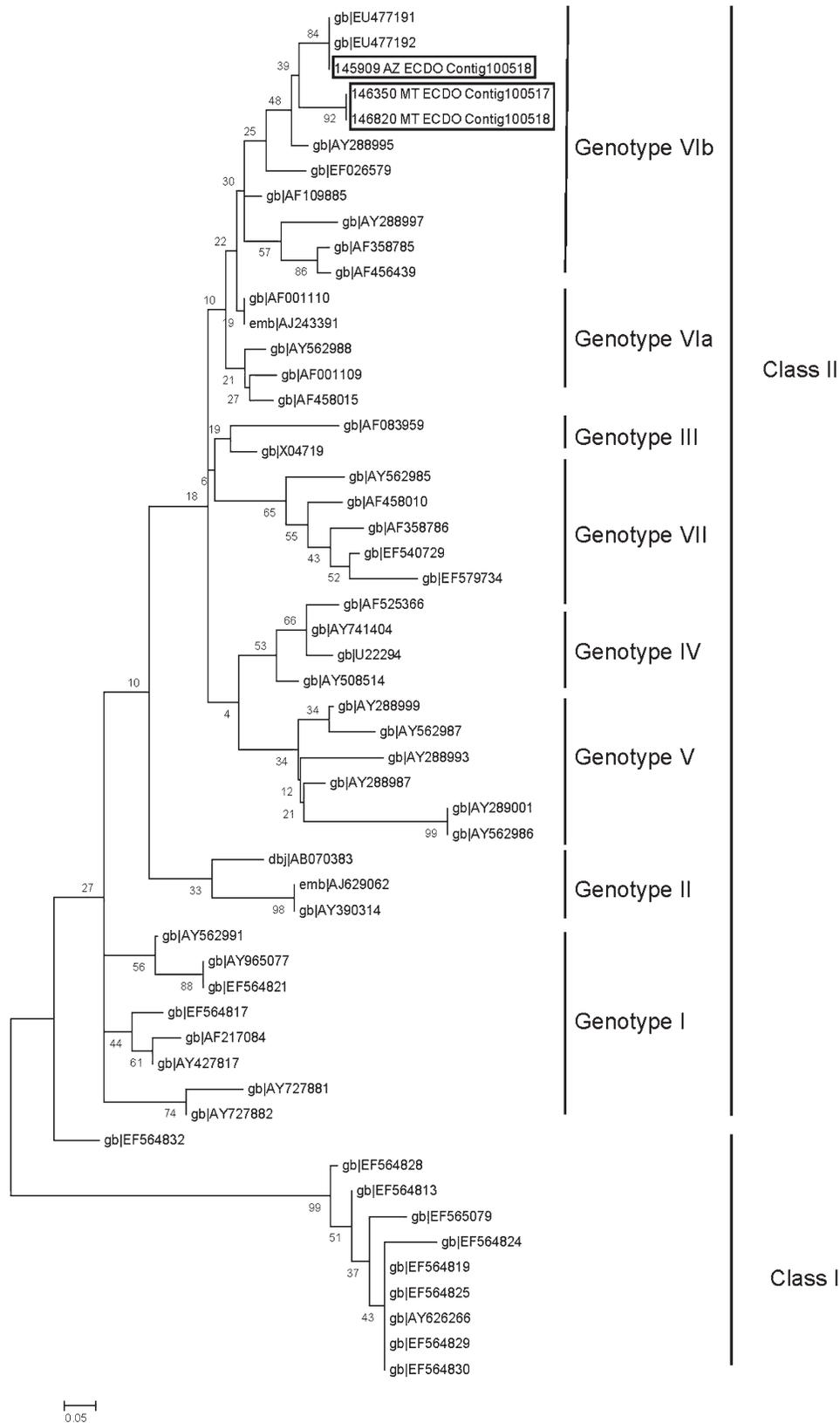
## RESULTS

Three of the eight carcasses (4 male, 4 female; 4 adult, 4 immature) from Arizona examined had extensive crushing trauma with multiple fractures of the keel; all were emaciated. Mild renal urate deposits were visible grossly in several birds. Two birds had diffuse or multifocal plaques in esophagi and crops, which we presume to be infection with *Trichomonas* spp.; culture was precluded because the birds had been frozen. Histologically, each bird had nephrosis with mild to moderate interstitial lymphocytic inflammation similar to PPMV infections in pigeons (Barton et al. 1992). One dove had severe vascular necrosis and vasculitis in the pericardial sac and coronary fat. There were no other

significant lesions. No bacteria or *Salmonella* spp. were identified in cultures. West Nile virus isolation attempts were negative. Toxicological analyses of crop and stomach contents were negative for poisoning by avicides and strychnine, and liver tissue was negative for toxins by gas chromatography/mass spectrometry. Brain cholinesterase activity was normal in three samples indicating that the birds had not been poisoned by organophosphate or carbamate pesticides. One bird was positive by RT-PCR for the avian influenza matrix protein, but no viable virus was recovered. At NWHC, testing of tracheal (7 of 8 positive) and cloacal (8 of 8 positive) swabs gave a mixture of weak and strong responses to APMV by RT-PCR following virus isolation. An APMV was isolated from specific-pathogen-free chicken embryos inoculated with samples from tissues: brain (6 of 8 positive), intestine (1 of 1 positive), and kidney/spleen pooled (1 of 1 positive). At NVSL, PPMV-1 was confirmed by differential reactions to specific monoclonal antibodies. This isolate was further confirmed by sequencing at NWHC. The cause of death for the eight ECDO was PPMV-1.

From six carcasses submitted by Montana Fish, Wildlife and Parks, three (3 male; 1 adult, 2 immature) received complete examinations. Body condition was poor to fair based on fat deposition and pectoral muscle mass. On gross examination, some birds had faint urate deposits in the kidneys. The crop was filled with a mixed bird seed. One bird had bite wounds to the head. Histologically, there was mild necrosis of the liver, fibrinous necrosis of the spleen, nephrosis, degenerate lymphocytes in the bursa of Fabricius, and no evidence of encephalitis. Brain cholinesterase activity was normal for two tested samples. No birds had evidence of other viral, bacterial, or parasitic infections, including *Trichomonas* spp. Of the three birds examined, virus was first identified by RT-PCR in tracheal (2 of 2 positive) and cloacal (2 of 2 positive) swabs, and isolation confirmed PPMV-1 in multiple organs: pinfeathers (1 of 2), brain (1 of 3), and kidney (3 of 3). The NVSL confirmed this isolate as PPMV-1 in one of the six case accessions. The cause of illness and death in the three ECDO examined was infection with PPMV-1.

Genetic sequencing identified isolates in the Genotype VIb (Fig. 2). Nucleotide sequence accession numbers of the three ECDO isolates reported here have the following GenBank accession numbers: 145909 AZ ECDO Contig100518, 146350 MT ECDO Contig100517, and 146820 MT ECDO Contig100518. The accession numbers of previously published sequences used in the analyses are in



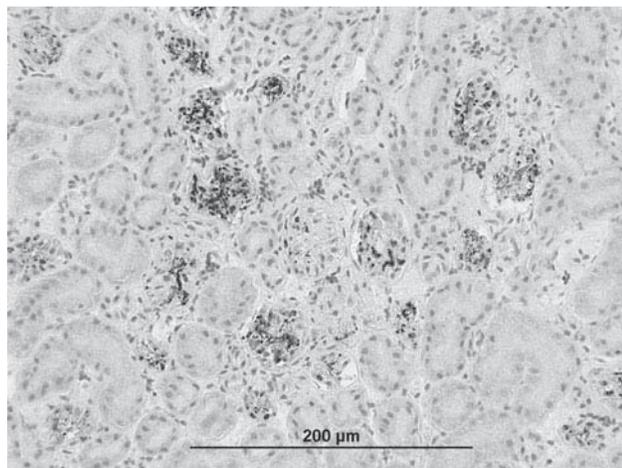
**Fig. 2.** Phylogenetic grouping of APMV-1 by class and lineage performed using Neighbor-Joining method (Saitou and Nei 1987). The tree is drawn to scale and branch lengths are in the same units as evolutionary distances. Arizona and Montana sequences are shown in boxes. Other sequences derived from GenBank (<http://www.ncbi.nlm.nih.gov/genbank/>).

Fig. 2. Immunohistochemistry had positive staining for NDV antigens in the nuclei and cytoplasm in the renal tubules of the kidney (Fig. 3) and blood vessels of the spleen for one bird from AZ (NWHC case 22835).

Testing of ECDO mortality events in Florida in 2001 was conducted at the University of Florida, and results were confirmed by NVSL as PPMV-1, not pathogenic to chickens. The birds had overall good body condition, but gross lesions included hepatic hemorrhage and pale small spleens. Histological examination revealed severe lymphoid depletion, large vacuoles in the mononuclear cells, and periportal inflammation and hemorrhage in the liver. Some birds were also infected with *Trichomonas*.

Two “turtle” doves (no formal identification performed) examined by KADL in 2006 were positive for PPMV-1 in brain tissue by virus isolation in cell culture, but neither had lesions consistent with APMV infection. The NVSL confirmed PPMV-1 by APMV-1 PCR (tracheal and cloacal swabs) and virus isolation (cloacal swabs). An unidentified dove from adjacent Broward County was positive for PPMV-1 by virus isolation, PCR, and negative-stained electron microscopic examination of cell culture, but PCR testing of tracheal and cloacal swabs were negative.

The cause of death for ECDO from the Florida Keys in 2008 was attributed to enteric parasitism (roundworms and *Eimeria* sp.), in combination with PPMV-1. Additional testing eliminated virulent ND, *Salmonella* spp., and toxicosis. Tracheal and cloacal swabs from all three birds were positive for paramyxovirus matrix on PCR, and the isolated virus was characterized with monoclonal antibodies as



**Fig. 3.** Immunohistochemistry staining of renal tubules of the kidney ( $\times 40$ ) by avidin–biotin complex technique with chicken polyclonal antiserum to NDV (Prairie Diagnostic Services, Inc., Saskatchewan, Canada).

PPMV-1. Confirmation at USDA-NVSL identified APMV-1 viral RNA, but the amino acid sequence at the fusion cleavage site indicated that it was not virulent APMV-1.

## DISCUSSION

Although it is possible that an unidentified disease agent may have been responsible for the disease events in Arizona, Montana, and Florida, we consistently identified the presence of PPMV in the absence of other primary pathogens. Immunohistochemistry further confirmed PPMV identified in virus culture, PCR and sequencing, and histological findings by detection of viral antigens in cells of renal tubules and spleen. PPMV-1 previously had not caused mortality in western states in ECDO, and these events were separated by  $> 1,700$  km. Therefore, these are presumed to be separate incidents with either ECDO acting as a low prevalence reservoir for the disease that typically does not result in mortality or isolated spillover from other carrier species, such as domestic pigeons. Because the AZ and MT events occurred during winter months, it is possible that environmental conditions or stress may have contributed to these disease events. The geographic range of this species now covers most of the country (Fig. 1) and has the potential to intermingle with many other types of birds. The mortality outbreaks in AZ and MT were in relatively rural areas where birds were unlikely to encounter high densities of pigeons. An unknown carrier is improbable as Kim et al. (2008) demonstrated that PPMV is not widespread in wild birds in the US. The relatively recent introduction of ECDO to the United States combined with a disease that is not commonly identified as a source of mortality in wild birds may have allowed previous inconspicuous outbreaks to be overlooked. The occurrence of outbreaks of genetically similar viruses (Fig. 2) in Arizona and Montana suggest that ECDO may serve as a source of introduction for PPMV virus disease in the United States. Based on the definition of an emerging disease as an infectious disease that is increasing in geographical range, host range, or prevalence (Dobson and Foufopoulos 2001), PPMV in ECDO is an emerging disease in the US.

The potential for species intermingling and transmission of novel viruses is of great concern for APMV-1 because the same virus may behave differently in various hosts (Toro et al. 2005). The majority of research related to PPMV was driven by concern over the potential spread to chickens (Kommers et al. 2002), and it is not routinely

considered in the diseases of wild birds despite speculation that PPMV is endemic and circulating in the US in feral pigeons (Kim et al. 2008). Our identification indicates PPMV in ECDO is the same sublineage VI (Fig. 2) as previously identified viruses in pigeons (Miller et al. 2009). Serosurveys and mortality sampling of wild birds indicated a low prevalence of PPMV in ECDO (Toro et al. 2005). The few isolates identified showed differences between pigeon strains in Rhode Island and those isolated from ECDO in Texas (Kim et al. 2008). Fortunately, changes in virulence are under negative or neutral selection so rapid alterations of the virus are unlikely (Miller et al. 2009). The rate of nucleotide change is 1% per decade for most NDV strains so slow adaptation of the virus to new hosts may happen without manifestation of disease (Ujvári et al. 2003); most infections go undetected until they are firmly established and an outbreak results. The first PPMV outbreak reported in Florida occurred two decades after ECDO invaded the Florida Keys and now three decades later PPMV has been detected in western states. Despite the slow mutation rate, PPMV has the potential to infect other species or recombine with other APMV strains to create a new genotype with variable infectivity and pathogenicity related to the host shift. It should not be assumed that PPMV is confined to a single species because ECDO are known to frequent bird feeders where they associate with a number of other species. There is a need to examine the pathogen host community and look for ecological factors that will determine emergence of disease in a new host species. These include spatial proximity of the reservoir host to other species, host immunocompetence, and pathogen evolution.

The broad maintenance of APMV in various species and opportunities for ECDO to commingle with other species could facilitate emergence of this new pathogen in free-ranging species (Fenton and Pedersen 2005). Transmission events may be rare, but single spillover occurrences could have dire consequences for naïve species. Ecological barriers, such as geographic separation, have been removed by range expansion of exotic ECDO to the majority of the US. In this situation, it is important to know where to direct management activities. If ECDO are reservoirs that maintain the virus and transmissions occur from repeated exposures, actions targeting native species may not be effective. Rather, actions that prevent between-species transmission will be most beneficial, including possible eradication of the pathogen by applying control measures to the host.

The majority of emerging pathogens are viruses or bacteria that have crossed species barriers as a result of

anthropogenic activities (Dobson and Foufopoulos 2001). While it is possible these PPMV outbreaks were the first to be reported and the event was caught in the early stages of establishment, reporting biases often exist when the outbreaks are small (<20 birds) or not fully investigated. Previous mortalities in ECDO may have had more importance placed on co-infections (i.e., *Trichomonas* spp. or other parasitism) and overlooked PPMV infection. The lack of a centralized wildlife health reporting protocol has hindered the flow of information about the occurrence of diseases across a range of species and habitats. Anthropogenic factors that assisted these outbreaks include introduction of exotic species (e.g., ECDO released in the Bahamas), feeding stations (e.g., birdfeeders), and possibly the presence of feral species (e.g., feral pigeons, parrots).

Non-native species disrupt and threaten natural ecosystems in many ways, one of which is by acting as reservoirs for disease. While there have been no detections of PPMV spillover to native species yet, it will be a difficult disease to manage if that occurs. Therefore, it is essential to take exotic disease risks to wildlife seriously and work to prevent introductions of hosts and pathogens.

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## REFERENCES

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- Aldous EW, Feller CM, Mynn JK, Alexander DJ (2004) A molecular epidemiological investigation of isolates of the variant avian paramyxovirus type 1 virus (PPMV-1) responsible for the 1978 to present panzootic in pigeons. *Avian Pathology* 33:258–269
- Alexander DJ (1980) Newcastle disease. In: *A Laboratory Manual for the Isolation and Identification of Avian Pathogens*, Purchase HG, Arp LH, Domermuth CH, Pearson JE (editors), Kennett Square, PA: American Association of Avian Pathologists, pp 114–120

- Barton JT, Bickford AA, Cooper GL, Charlton BR, Cardona CJ (1992) Avian paramyxovirus type 1 infections in racing pigeons in California: clinical signs, pathology, and serology. *Avian Diseases* 36:463–468
- Biancifiori F, Fioroni A (1983) An occurrence of Newcastle disease in pigeons: virological and serological studies on the isolates. *Comparative Immunology, Microbiology and Infectious Diseases* 6:247–252
- Coffee LL, Hanson BA, Luttrell MP, Swayne DE, Senne DA, Goekjian VH, et al. (2010) Avian paramyxoviruses in shorebirds and gulls. *Journal of Wildlife Diseases* 45:481–487
- Daoust PY, McBurney S (2001) Newcastle disease in pigeons, Prince Edward Island. *Canadian Cooperative Wildlife Health Centre Newsletter* 8:8–9
- Dobson A, Foufopoulos J (2001) Emerging infectious pathogens of wildlife. *Philosophical Transactions of the Royal Society* 356:1001–1012
- Dortmans JCFM, Koch G, Rottier PJM, Peeters BPH (2009) Virulence of pigeon paramyxovirus type 1 does not always correlate with the cleavability of its fusion protein. *Journal of General Virology* 90:2746–2750 [Online Feb 14, 2010] . doi:10.1099/vir.0.014118-0
- Felsenstein J (1985) Confidence limits on phylogenies: an approach using the bootstrap. *Evolution* 39:783–791
- Fenton A, Pedersen AB (2005) Community epidemiology framework for classifying disease threats. *Emerging Infectious Diseases* 11:1815–1821
- Hinshaw VS, Wood JM, Webster RG, Deibel R, Turner B (1985) Circulation of influenza viruses and paramyxoviruses in waterfowl originating from two different areas of North America. *Bulletin of the World Health Organization* 63:711–719
- Kaleta EF (1992) Paramyxoviruses in free-living and captive birds—a brief account. In: *Workshop on Avian Paramyxoviruses*, Kaleta EF, Heffels-Remann U (editors), Germany: Rauischholzhausen, pp 262–271
- Kaleta EF, Alexander DJ, Russell PH (1985) The first isolation of the avian PMV-1 virus responsible for the current panzootic in pigeons? *Avian Pathology* 14:553–557
- Kim LM, King DJ, Guzman H, Tesh RB, Travassos da Rosa ABA, Bueno R Jr, et al. (2008) Biological and phylogenetic characterization of pigeon paramyxovirus serotype 1 circulating in wild North American pigeons and doves. *Journal of Clinical Microbiology* 46:3303–3310 [Online Feb 12, 2010] . doi:10.1128/JCM.00644-08
- Kommers GD, King DJ, Seal BS, Brown CC (2001) Virulence of pigeon-origin Newcastle disease virus isolates for domestic chickens. *Avian Diseases* 45:906–921
- Kommers GD, King DJ, Seal BS, Carmichael KP, Brown CC (2002) Pathogenesis of six pigeon-origin isolates of Newcastle disease virus for domestic chickens. *Veterinary Pathology* 39:353–362
- Kommers GD, King DJ, Seal BS, Brown CC (2003) Pathogenesis of chicken-passaged Newcastle disease virus isolated from chickens and wild and exotic birds. *Avian Diseases* 47:319–329
- Kuiken T, Wobeser G, Leighton FA, Haines DM, Chelack B, Bogdan J, Hassard L, Heckert RA, Riva J (1999) Pathology of Newcastle disease in double-crested cormorants from Saskatchewan, with comparison of diagnostic methods. *Journal of Wildlife Diseases* 35:8–23
- Meteyer CU, Docherty DE, Glaser LC, Franson JC, Senne DA, Duncan R (1997) Diagnostic findings in the 1992 epornitic of neurotropic velogenic Newcastle disease in double-crested cormorants from the upper Midwest United States. *Avian Diseases* 41:171–180
- Miller PJ, Kim LM, Ip HS, Afonso CL (2009) Evolutionary dynamics of Newcastle disease virus. *Virology* 391:64–72 [Online Feb 12, 2010] . doi:10.1016/j.virol.2009.05.033
- Nostrandt AC, Duncan JA, Padilla S (1993) A modified spectrophotometric method appropriate for measuring cholinesterase activity in tissue from carbaryl-treated animals. *Toxicological Science* 21:196–203
- Saitou N, Nei M (1987) The neighbor-joining method: a new method for reconstructing phylogenetic trees. *Molecular Biology and Evolution* 4:406–425
- Smith PW, Kale HW (1986) Eurasian Collared Doves collected in Florida. *Florida Field Naturalist* 14:104–107
- Tamura K, Dudley J, Nei M, Kumar S (2007) MEGA4: molecular evolutionary genetics analysis (MEGA) software version 4.0. *Molecular Biology and Evolution* 24:1596–1599
- Terregino CG, Cattoli CG, Gorssele B, Bertoli E, Tisato E, Capua I (2003) Characterization of Newcastle disease virus isolates obtained from Eurasian collared doves (*Streptopelia decaocto*) in Italy. *Avian Pathology* 32:63–68
- Thompson JD, Higgins DG, Gibson TJ (1994) CLUSTAL W: improving the sensitivity of progressive multiple sequence alignment through sequence weighting, position-specific gap penalties and weight matrix choice. *Nucleic Acids Research* 22:4673–4680
- Toro HF, Hoerr J, Farmer K, Dykstra CC, Roberts SR, Perdue M (2005) Avian paramyxovirus: association with common avian pathogens in chickens and serologic survey in wild birds. *Avian Diseases* 49:92–98
- Ujvári DE, Wehmann E, Kaleta EF, Werner O, Savić V, Nagy E, Czifra G, et al. (2003) Phylogenetic analysis reveals extensive evolution of avian paramyxovirus type 1 strains of pigeons (*Columba livia*) and suggests multiple species transmission. *Virus Research* 96:63–73
- Weldon C, du Preez LH, Hyatt AD, Muller R, Speare R (2004) Origin of the amphibian chytrid fungus. *Emerging Infectious Diseases* 10:2100–2105
- Wise MG, Suarez DL, Seal BS, Pedersen JC, Senne DA, King DJ, Kapczynski DR, Spackman E (2004) Development of a real-time reverse-transcription PCR for detection of Newcastle disease virus RNA in clinical samples. *Journal of Clinical Microbiology* 42:329–338