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# ALEUTIAN MINK DISEASE PARVOVIRUS IN WILD RIPARIAN CARNIVORES IN SPAIN

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ABSTRACT: Serious declines in populations of native European mink (*Mustela lutreola*) have occurred in Europe. One responsible factor may be infectious diseases introduced by exotic American mink (*Mustela vison*). In order to investigate a possible role for Aleutian mink disease parvovirus (ADV), we surveyed native riparian carnivores and feral American mink. When serum samples from 12 free-ranging European and 16 feral American mink were tested, antibodies to ADV were detected from three of nine European mink. ADV DNA was detected by polymerase chain reaction in whole cell DNA from four of seven carcasses; two American mink, one European mink and a Eurasian otter (*Lutra lutra*). Lesions typical of Aleutian disease were present in one of the American mink. A portion of the ADV VP2 capsid gene was sequenced and the results suggested that two sequence types of ADV were circulating in Spain, and that the Spanish ADVs differed from other described isolates from North America and Europe. Future conservation and restoration efforts should include measures to avoid introduction or spread of ADV infection to native animals.

*Key words:* Aleutian mink disease parvovirus, *Lutra lutra, Mustela lutreola, Mustela putorius, Mustela vison,* polymerase chain reaction, survey.

## INTRODUCTION

The European mink (*Mustela lutreola*) is one of the most threatened terrestrial mammals in the world (Schreiber et al., 1989; Camby, 1990; Saint-Girons, 1991). Although these animals were once widely distributed throughout Europe (Youngman, 1982), their distribution is now restricted to a small fraction of their historic range. The remaining populational nuclei, which are isolated and distant from each other, are situated in eastern Europe, southern France, and northern Spain.

Several factors have been implicated in the dramatic decline of this mustelid throughout Europe (Camby, 1990; Saint-Girons, 1991; Rozhnov, 1993; Maran and Henttonen, 1995; Palazón and Ruiz-Olmo, 1997). These include the loss of habitat, excessive hunting and trapping, pollution, and interspecies competition by the larger American mink (*Mustela vison*). Nevertheless, according to currently available data, none of these factors alone can explain the decline of the native species (Maran and Henttonen, 1995).

With regard to the introduction of the American mink, both interspecies competition (Maran, 1989; Maran et al., 1998) and the possible introduction of exotic diseases have been cited as possible causes for declines (Henttonen and Tolonen, 1983; Henttonen, 1992). American mink have been raised on commercial European mink farms since the start of the twentieth century. Both accidental escape and deliberate release of these animals has led to substantial feral American mink populations throughout Europe (Dunstone, 1993). The American mink have negatively affected the European mink as a result of competition (Sidorovich, 1992; Maran et

al., 1998), although in many areas the decline of native mink was evident prior to the arrival of the exotic species.

A similar situation exists in Spain. Since the first demonstration of American mink as a wild species in the Iberian Peninsula in 1978 (Ruiz-Olmo et al., 1997), the number of existing wild American mink populations in Spain has expanded widely (Ruiz-Olmo et al., 1997). Because the present range of the European mink is so limited, the presence of American mink farms and the abundance of these animals in the wild pose definite risks to continued survival of the native species.

One risk that the exotic American mink poses is the introduction of infectious diseases or strains of infectious agents that are particularly deleterious to the European mink. Among these infections, Aleutian mink disease parvovirus (ADV) is of particular interest because of its high prevalence on fur farms and because of its characteristics. ADV is highly persistent in the environment (Hansen, 1985). Disease may be spread by asymptomatic carriers, and transmission can be both horizontally, by urine, feces, saliva and vertically (Kenyon et al., 1963; Gorham et al., 1964, 1976).

In American mink, ADV can cause different syndromes, depending on host factors, such as age and genotype, and the virulence of the viral strain (Bloom et al., 1994). It produces acute, usually fatal, interstitial pneumonia in seronegative neonatal American mink (Alexandersen et al., 1994; Bloom et al., 1994). The classic form of Aleutian disease is characterized by persistent viral infection, the development of plasmacytosis, hypergammaglobulinemia, and immune complex-mediated glomerulonephritis and arteritis in adult mink (Porter, 1986; Bloom et al., 1994). In addition, some animals can have an inapparent infection (An et al., 1978; Hadlow et al., 1985) while others can clear the infection (Hadlow et al., 1985), although it is uncertain if this is a consequence of host factors or the particular isolate of infecting virus (Fox et al., 1999). In addition to direct mortality, ADV infections can lead to population declines by causing a decrease in fertility and spontaneous abortions (Padgett et al., 1967; Haagsma, 1969; Gorham et al., 1976; Hansen and Lund, 1997). In brief, there are a number of ways in which ADV could lead to population declines.

In spite of the fact that ADV infections are so widespread on mink ranches and in ferret populations worldwide (Hansen, 1985; Welchman et al., 1993), information concerning the impact of ADV on related species is limited. It has been suggested that a number of mustelids, including short-tailed weasel (*Mustela erminea*), fishers (*Martes pennanti*), marten (*Martes americana*), and otters (*Lutra canadensis*) can be infected with ADV and serological evidence for infection in striped skunk (*Mephitis mephitis*), raccoons (*Procyon lotor*), wild American mink and foxes (*Vulpes vulpes*) has been detected (Ingram and Cho, 1974; Kenyon et al., 1978; Alexandersen et al., 1985). Furthermore, a role for American raccoons in the transmission of virus has been suggested (Oie et al., 1996) and viral DNA sequences have been identified in feral American raccoons and striped skunks (Oie et al., 1996). To our knowledge, data does not exist on the prevalence of ADV in wild, riparian carnivores in the Paleartic region, although in one study its presence was suspected in a wild Eurasian otter (*Lutra lutra*) (Wells et al., 1989). Therefore, the knowledge of the prevalence and effects of this disease in a threatened species like the European mink, and in other riparian carnivores such as the Eurasian otter and the European Polecat (*Mustela putorius*), may be of importance for their conservation.

## MATERIALS AND METHODS

Serum samples were collected from 12 European mink (nine adults and three subadults) trapped at La Rioja (seven), Alava (two), Burgos (two) and Soria (one) and 16 American free-ranging mink (11 adults and five subadults) trapped at Segovia (nine), Alava (three),

Madrid (one), Coruña (one), Girona (one) and Castellón (one). The animals were captured between November 1997 and November 1999 in $20 \times 20 \times 60$ cm wire cage traps (home-made), and injected intramuscularly with an anesthetic combination of Ketamine hydrochloride (Imalgène 1000, Merial, Lyon, France) with xylazine hydrochloride (Rompún, Bayer AG, Leverkusen, Germany) or medetomidine hydrochloride (Domtor, Orion Corporation, Espoo, Finland). Blood samples were collected by jugular venipuncture or by toenail cutting into heparinized and non-heparinized capillary tubes. After the animals had recovered from anesthesia, they were released. Serological evidence of ADV infection was evaluated by counter immune electrophoresis (CEP) using a commercial test antigen (United Vaccines, Inc. Madison, Wisconsin, USA) (Bloom et al., 1975; Oie et al., 1996). The presence of elevated serum gamma globulins was assayed by serum protein electrophoresis (Bloom et al., 1975). Technical considerations (small volume of serum, broken capillary tubes, serum hemolysis) made it impossible to perform CEP endpoint determinations by testing serial dilutions or to do both CEP and serum protein electrophoresis in every case. Nevertheless, the presence of a positive CEP reaction under the conditions we have employed is considered diagnostic of ADV infection (Bloom et al., 1975; Oie et al., 1996). No samples of blood or tissue from live captured animals were available for DNA studies.

Carcasses from one adult Eurasian otter from Lugo, one adult European mink from Alava and five American mink (four adults and one subadult) from Madrid (two), Burgos (one), Segovia (one) and A Coruña (one) were available for study. Serological analysis had previously been completed on two of the American mink. Organ samples from kidney, lungs, spleen, mesenteric lymph node, liver, heart, intestine and muscle were submitted for pathological study to determine if lesions characteristic of ADV infection were present.

Samples of spleen, mesenteric lymph node, liver and kidney from each animal were pooled and whole cell DNA was isolated (Oie et al., 1996). Polymerase chain reaction (PCR) was done on an aliquot of DNA from each animal as previously noted (Oie et al., 1996; Bloom et al., 1997). The ADV DNA product from positive PCR reactions was sequenced; this segment contains the hypervariable region of the VP2 capsid protein gene (Oie et al., 1996). Because the overall length of VP2 for the Spanish ADV samples was not known, it was not possible to assign residue numbers that correspond to the other sequences. Therefore, the first amino acid is numbered "1." The predicted amino acid sequences were compared with that published for other American and European ADV isolates (Oie et al., 1996; Schuierer et al., 1997), as well as an isolate of ADV derived from ferrets (ADV-Ferret) (Saifuddin and Fox, 1996). We have denoted the Spanish ADV as ADV-ES. The GENBANK Submission numbers for the ADV-ES sequences are AF 205380, AF 205381, AF 205382.

## RESULTS

Counterimmunoelectrophoresis (CEP) was performed on 14 of the 16 serum samples obtained from American mink. None of these sera gave a positive reaction for antibody to ADV. In addition, 15 of 16 animals had gamma globulin levels <20% of the total serum proteins ($\bar{x}$ = 6%, range = 3–15%).

One American mink, captured near Segovia in August, 1999, had a gamma globulin level of 27%. This value is consistent with progressive ADV infection (Bloom et al., 1975; Oie et al., 1996); however, there was insufficient serum to perform CEP on this animal. Consequently, it was not possible to state with certainty if this animal was infected with ADV.

CEP also was performed on nine of 12 serum samples obtained from free-ranging European mink. Three of these samples were positive for antibody to ADV (one adult and two subadult) trapped at La Rioja (two) and Burgos (one).

None of 11 European mink serum samples performed had gamma globulin levels suggestive of progressive ADV infection ($\bar{x}$ = 13%, range = 6–20%). The ADV positive mink all had levels <13%.

Carcasses from seven animals were available for study: five American mink, one European mink and one Eurasian otter. Two of the American mink had previously tested negative for ADV by CEP. Microscopic lesions consistent with AD were observed only in a solitary feral American mink (Colas MV2), road-killed near Madrid in September, 1998. This animal had glomerulonephritis and plasma cell proliferation with infitration into liver, spleen,

kidney and mesenteric lymph node. None of the other animals had pathological evidence of AD.

ADV sequences were detected by PCR in whole cell DNA from four animals: two American mink (ADV-ES(MV1) and ADV-ES(Colas MV2)), including the one with microscopic changes consistent with AD, one European mink (ADV-ES(Andres ML1)) and a Eurasian otter (ADV-ES(LL6))(Oie et al., 1996). Thus, four of the animals were infected with ADV but only a single mink had microscopic changes of typical disease.

In order to compare the sequence of the VP2 capsid gene of the Spanish ADV (ADV-ES) with the corresponding segment of other ADV isolates, PCR products were purified and DNA sequenced. The amplified portion of the gene contains sequence encoding the hypervariable region of the VP2 capsid protein (Bloom et al., 1988). Although the hypervariable region sequence does not correlate with virulence or pathogenicity, it is a useful tool in identifying different isolates of ADV (Oie et al., 1996; Saifuddin and Fox, 1996; Bloom et al., 1998; Fox et al., 1999). Sequence information was obtained for virus from three animals: a European mink (Andres ML1), an American mink (Colas MV2), and the Eurasian otter (LL6); the Colas MV2 mink had lesions characteristic of ADV on pathological study. Comparison of the predicted amino acid sequences suggested that the Eurasian otter (LL6) and the American mink (Colas MV2) were infected with the same virus. However, the sequence of the ADV-ES obtained from the European mink (Andres ML1) was different (Fig. 1).

The ADV-ES sequences also were compared with the corresponding sequences from a number of American ADV isolates (ADV-G, ADV-TR, ADV-Utah, ADV-Pullman), a Danish isolate (ADV-DK (zk8)), and ADV-Ferret (Gottschalk et al., 1991; Oie et al., 1996; Saifuddin and Fox, 1996). The ADV-SL3 isolate from Germany has a sequence identical to ADV-G in this study (Schuierer et al., 1997). None of the Spanish sequences were identical to those reported for other ADV isolates. However, the LL6 and the Colas MV2 ADV-ES sequences shared a—QXQLEWTGT—motif present in the hypervariable region of the Danish isolate of ADV and most pathogenic American isolates (Oie et al., 1996). The Andres ML1 shared only four residues in the hypervariable region, and thus appeared distantly related to any of the described isolates. None of these ADV appeared similar to the ADV-Ferret sequence (Saifuddin and Fox, 1996).

## DISCUSSION

In this study we have identified the presence of ADV in free-ranging, riparian carnivores in Spain. It was not possible to define a role for ADV infections in the population declines of the European mink. However, our results suggested that ADV infections are present among free-ranging riparian carnivores in Spain. Consequently, continued investigations are warranted on both European and American mink, Eurasian otters, and also related species like the European polecat (*Mustela putorius*). Additional surveys on both live captured and dead animals will provide a more accurate assessment of ADV infection in the wild populations at risk.

Neither of the two different sequences types of ADV in Spain appeared identical to other isolates reported in Europe or in North America (Fig. 1) (Gottschalk et al., 1991; Oie et al., 1996; Saifuddin and Fox, 1996; Schuierer et al., 1997). This finding suggested that the Spanish ADV did not derive from a previously described isolate. We plan to obtain ADV samples from other areas of Europe and make comparisons of the DNA sequences. This information will enable us to assess the spectrum of ADV in Europe and perhaps to derive a phylogenetic tree.

Recent studies have shown that the pathogenicity of ADV is in part determined by virally encoded determinants in the capsid protein between amino acids

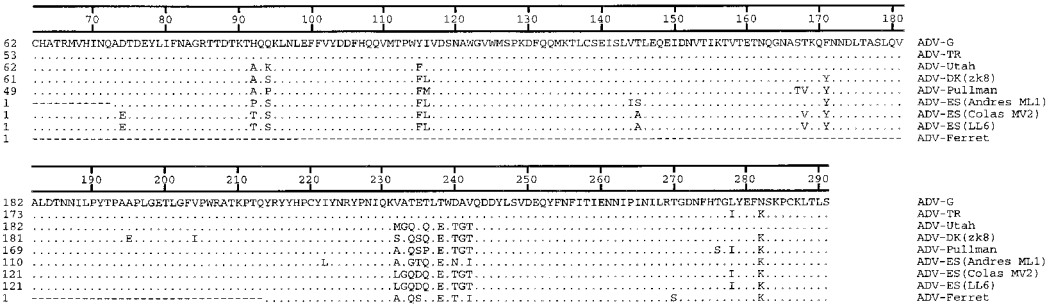


FIGURE 1. Comparison of Spanish Aleutian mink disease parvovirus (ADV) VP2 capsid gene sequences with American and European ADV isolates. The translated amino acid sequences from ADV-ES (Colas MV2), ADV-ES (Andres ML1), and ADV-ES (LL6), were aligned with the corresponding VP2 regions from ADV-G, ADV-TR, ADV-Utah, ADV-Pullman, ADV-DK (zk8) and ADV-Ferret using the Megalign module of DNASTAR (DNASTAR, Inc., Madison, Wisconsin). The residue numbers above the alignment refer to the ADV-G VP2 coordinates and hypervariable region comprises ADV-G VP2 residues 231 to 242.

341 and 590 of VP2 (Bloom et al., 1998; Fox et al., 1999). The limited sequence obtained for the two Spanish viruses did not include this region of VP2 (Fig. 1). Consequently, it was not possible to speculate upon their pathogenicity based on the DNA sequence. However, the animal infected with ADV-ES (Colas MV2) had pathology consistent with progressive Aleutian disease, suggesting that this virus had pathogenic potential.

Adult mink can be persistently infected with certain types of ADV, develop antiviral antibody, and still not develop evidence of progressive disease (An et al., 1978; Hadlow et al., 1984; Bloom et al., 1988; Fox et al., 1999). It is unknown if subtle infections like this lead to reduced reproductive success, interstitial pneumonitis in mink kits, or could cause population declines.

The origin of the ADV infecting the animals is uncertain. Since ADV is common on commercial mink farms, diligence is required to insure that infectious virus does not enter the environment from these operations (Hansen, 1985). Therefore, it is imperative to institute adequate ADV sanitation programs on fur farms and also to establish measures to prevent animals from escaping. In addition, procedures should be developed to disinfect animal remains after pelting.

Finally, a major goal of conservation programs is the restoration and reintroduction of threatened native species. This will necessitate use of captive breeding or translocation. In order to avoid contamination of these facilities and subsequent introduction or spread of infection, strict protocols will have to be developed and monitored.

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