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Snapshot of Viral Infections in Wild Carnivores Reveals Ubiquity of Parvovirus and Susceptibility of Egyptian Mongoose to Feline Panleukopenia Virus

Margarida D. Duarte[1], Ana Margarida Henriques[1], Sílvia Carla Barros[1], Teresa Fagulha[1], Paula Mendonça[1], Paulo Carvalho[1], Madalena Monteiro[1], Miguel Fevereiro[1], Mafalda P. Basto[2], Luís Miguel Rosalino[2,3], Tânia Barros[4], Victor Bandeira[4], Carlos Fonseca[4], Mónica V. Cunha[1]*

1 INIAV, I.P.- Instituto Nacional de Investigação Agrária e Veterinária, Unidade Estratégica de Produção e Saúde Animal, Lisboa, Portugal, **2** Universidade de Lisboa, Centro de Biologia Ambiental, Faculdade de Ciências de Lisboa, Lisboa, Portugal, **3** Laboratório de Ecologia Isotópica/CENA, Universidade de São Paulo, São Paulo, Brasil, **4** Departamento de Biologia and CESAM, Universidade de Aveiro, Aveiro, Portugal

Abstract

The exposure of wild carnivores to viral pathogens, with emphasis on parvovirus (CPV/FPLV), was assessed based on the molecular screening of tissue samples from 128 hunted or accidentally road-killed animals collected in Portugal from 2008 to 2011, including Egyptian mongoose (*Herpestes ichneumon*, n = 99), red fox (*Vulpes vulpes*, n = 19), stone marten (*Martes foina*, n = 3), common genet (*Genetta genetta*, n = 3) and Eurasian badger (*Meles meles*, n = 4). A high prevalence of parvovirus DNA (63%) was detected among all surveyed species, particularly in mongooses (58%) and red foxes (79%), along with the presence of CPV/FPLV circulating antibodies that were identified in 90% of a subset of parvovirus-DNA positive samples. Most specimens were extensively autolysed, restricting macro and microscopic investigations for lesion evaluation. Whenever possible to examine, signs of active disease were not present, supporting the hypothesis that the parvovirus *vp2* gene fragments detected by real-time PCR possibly correspond to viral DNA reminiscent from previous infections. The molecular characterization of viruses, based on the analysis of the complete or partial sequence of the *vp2* gene, allowed typifying three viral strains of mongoose and four red fox's as feline panleukopenia virus (FPLV) and one stone marten's as newCPV-2b type. The genetic similarity found between the FPLV viruses from free-ranging and captive wild species originated in Portugal and publicly available comparable sequences, suggests a closer genetic relatedness among FPLV circulating in Portugal. Although the clinical and epidemiological significance of infection could not be established, this study evidences that exposure of sympatric wild carnivores to parvovirus is common and geographically widespread, potentially carrying a risk to susceptible populations at the wildlife-domestic interface and to threatened species, such as the wildcat (*Felis silvestris*) and the critically endangered Iberian lynx (*Lynx pardinus*).

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* E-mail: monica.cunha@iniav.pt

Introduction

Canine parvovirus (CPV) and feline panleukopenia virus (FPLV) are closely related viruses that have been included in the unique species *Feline panleukopenia virus* together with other antigenic and genetically related viruses, such as raccoon parvovirus (RPV), raccoon dog parvovirus (RDPV), blue fox parvovirus (BFPV) and mink enteritis virus (MEV) [1,2,3]. All together, these viruses infect a wide range of domestic and wild species of the order Carnivora [2]. VP2, the major structural protein of the viral capsid, determines the pathogenicity, tissue tropism and host ranges of this virus subgroup [4,5]. FPLV was originally identified in domestic cats [6] and later on other large felids, such as tigers, panthers, cheetahs and lions [2,7,8,9,10,11,12]. Canine parvovirus (CPV-2) was detected for the first time in 1978, possibly emerging from a FPLV like-virus [13]. This highly virulent virus rapidly became endemic in dogs throughout the world. Original CPV-2 strain did not infect cats [4], however it was replaced by new antigenic variants, designated CPV-2a, CPV-2b and CPV-2c that regained the ability to infect felids [3,5,14].

Depending on age and immunological status, the infection of young domestic carnivores and a few species of large felids can be sub-clinical, acute (characterized by leukopenia, fever, depression, dehydration, and diarrhoea), or cause sudden death [2,15]. However, in mustelids (otters, badgers, ferrets, martens and fishers) and viverrids (genets and civets), the pathogenicity of the disease caused by feline-like parvoviruses is still unclear. Reports refer mainly to serological or virological evidences rather than to clinical or anatomo-histological data (reviewed by [2]). MEV