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Emergence and evolution of canine parvovirus (14-Aug-1999)

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Abstract

Canine parvovirus type-2 (CPV-2) emerged as a new virus of dogs in the late 1970's, and derived as a variant of a related virus of another host, possibly from the long known feline panleukopenia virus (FPV). We know today that the apparently different host range properties of CPV and FPV are determined by the capsid protein of the viruses, and that only few amino acids are responsible for critical genetic and antigenic properties. Various hypotheses about its sudden emergence had been put forward in the past, but it appears likely that CPV arose in a host different from the cat or dog, and that an other (wild) carnivore may have harbored the immediate ancestor of CPV.

The phylogenetic analysis of parvovirus DNA from various wild carnivore isolates supported that hypothesis. The only available fox isolate [from a farmed Arctic fox (*Alopex lagus*) from Finland] revealed a DNA sequence intermediate between the FPV and CPV viruses, although the corresponding amino acid sequence clearly defined the virus as an FPV type virus. A partial DNA sequence from a wild red fox (*Vulpes vulpes*) from Germany represented the first true intermediate sequence in terms of amino acid sequences. Serological survey in foxes revealed a sero-prevalence of parvoviruses in foxes of about 10-15%, making an inter-species transmission between domestic and wild carnivores feasible.

Analysis of further wild carnivore sequences, including two free-ranging red foxes and one captive Siberian tiger from Germany, five cheetahs from zoos in North America, 4 free-ranging cheetahs, one African wild cat, one bat-eared fox and one honey badger, all from Southern Africa did not reveal further intermediate sequences, but showed either FPV-like sequences (3 cheetahs, the African wild cat, the honey badger) or CPV-2b sequences (6 cheetahs, the bat-eared fox) and CPV-2a sequences (the Siberian tiger).

Soon after its first appearance CPV-2 was replaced during the next 3 years by antigenically variant viruses (CPV types-2a and -2b) which now coexist in dog populations world-wide. These new virus variants may be the result of an adaptation of the new virus to its host, but we also showed that the new virus types have a broader host range. Although the original CPV-2 did not replicate in cats, both types-2a and -2b replicated efficiently. Viruses isolated from cats with natural disease were antigenically indistinguishable from CPV type-2a and CPV type-2b viruses, and the capsid protein gene sequences of the feline isolates were essentially identical to corresponding CPV type-2b or type 2b viruses isolated from dogs.

The analysis of parvovirus sequences from large cats revealed a high percentage of CPV-2a and -2b viruses in clinically affected animals. This may reflect a higher susceptibility of large cats to these virus types, similar to the scenario that has been described for canine distemper virus. The history of our cases suggest that at least some infections were most likely due to dog to large cat transmission. This study may warrant a closer monitoring of parvovirus infections in large cats and may also add to the discussion to include CPV-2a or -2b components in feline vaccines.

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