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Evolution of canine parvovirus—A need for new vaccines?

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Abstract

Canine parvovirus (CPV) is a new virus, which is continuing to evolve, giving rise to new antigenic types and virus mutants that spread through the dog population. The most successful mutants, from an evolutionary perspective, appear to be selected by improved binding to the CPV receptor, the canine transferrin receptor, and by an extended host range, which for the newer antigenic types now includes both the dog and the cat. The new viruses also show antigenic differences that can be defined by binding of certain monoclonal antibodies; they also differ in their reactivity in virus neutralisation tests, using immune sera raised against the various antigenic types. These differences may influence the susceptibility of young animals to infection at the time when the level of maternally derived antibody decreases to the minimum protective titre. This minimum protective titre may vary depending on the infecting virus type. There is, however, a high degree of cross-protection between the virus types and the true relevance of the differences in neutralisation titer is currently not known.

Keywords: Canine parvovirus; Emergence; Evolution; Antigenic types; Protection; Vaccination

1. Emergence and evolution of canine parvovirus

Canine parvovirus (CPV) was first isolated in 1978 (Appel et al., 1979). Retrospective serological analysis has since revealed that the first positive sera were collected in 1976 (Koptopoulos et al., 1986). However, comprehensive independent studies on the rate of CPV molecular evolution has indicated that the virus must have emerged at least 10 years earlier (Shackelton et al., 2005; Hetzl et al., in preparation).

CPV is believed to have originated as a host range variant from feline panleukopenia virus (FPV). Various hypotheses for this emergence have previously been put forward; these include a direct mutation from FPV, a mutation from an FPV vaccine virus, and the adaptation to the new dog host via non-domestic carnivores, like mink and foxes (Truyen, 1999). In the author's opinion the latter appears the most likely scenario, as those animals were (and still are) often housed together on farms at a high animal density. Interestingly, this evolution required a consecutive substitution, and the parallel mutation of three key amino acids (Parrish et al., 1991; Truyen et al., 1995; Fig. 1).

Soon after its first recognition in the late 1970's the original virus type CPV-2 was replaced by a new virus type that could be distinguished using monoclonal antibodies. This new antigenic type was termed CPV-2a,

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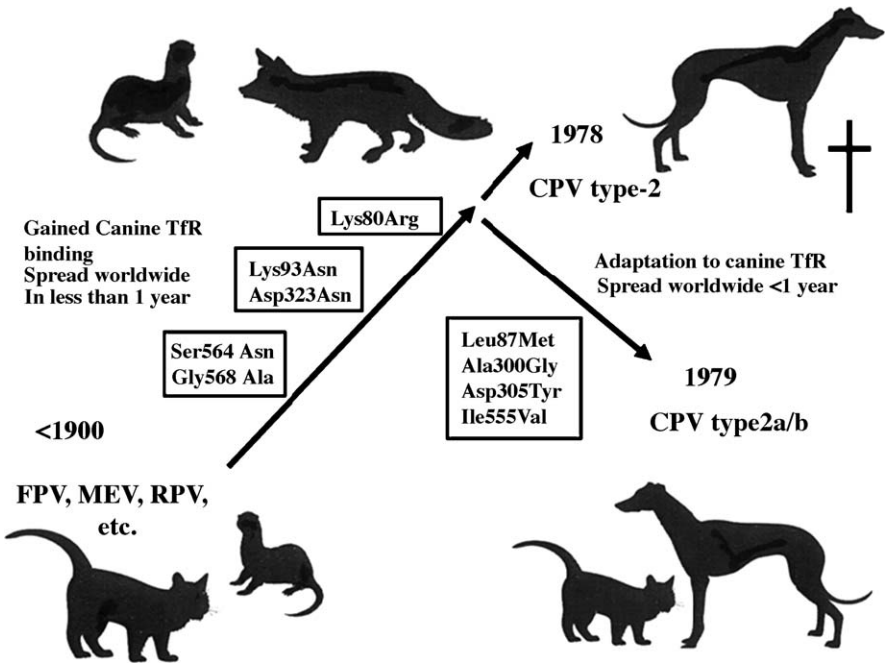

Fig. 1. Cartoon of the evolution of canine parvovirus as a host range mutant from feline panleukopenia virus (FPV), mink enteritis virus (MEV), or raccoon parvovirus (RPV). The original virus type CPV-2 went extinct, and was replaced by the new antigenic types CPV-2a and -2b. Key factors in this evolution were the gaining and adaptation to the canine tranferrin receptor (TfR) (modified from Truyen, 1999; Hueffer and Parrish, 2003).

and along with a virus type displaying another (single) mutation, termed CPV-2b, quickly spread worldwide (Parrish et al., 1985, 1988). Since then a number of further mutations have been described, some of them associated with antigenic differences. Unfortunately, the nomenclature of these viruses is inconsistent and confusing, so that various distinct "CPV-2c" viruses have been reported (Ikeda et al., 2002; Martella et al., 2004). A cartoon of the evolution of CPV is shown in Fig. 1, and the relevant amino acid differences between the various virus types and mutants are summarised in Table 1.

Interestingly, some mutations, such as S297A appear to be advantageous to the virus since this has spread and becomes established in the populations (Truyen, 1999).

Table 1
Evolutionary relevant amino acid substitutions of FPV, CPV-2, and the various antigenic types and mutants of CPV (one letter amino acid code)

	Amino acid VP2											
Virus	80	87	93	265	297	300	305	323	426	555	564	568
FPV	K	M	K	T	S	A	D	D	N	V	N	A
CPV-2	R	M	N	T	S	A	D	N	N	V	S	G
CPV-2a	R	L	N	T	S	G	Y	N	N	I	S	G
CPV-2b	R	L	N	T	S	G	Y	N	D	V	S	G
CPV-2c(a)	R	M	N	T	A	D	Y	N	N	V	S	G
CPV-2c(b)	R	L	N	T	A	D	Y	N	D	V	S	G
S297A	R	L	N	T	A	G	Y	N	N D	I	S	G
D426E	R	L	N	T	S A	G	Y	N	E	V	S	G
T265P	R	L	N	P	S	G	Y	N	D	V	S	G

Modfied from Ikeda et al. (2002) and Martella et al. (2004).