TECHNICAL UPDATE: CANINE PARVOVIRUS 2C IN AUSTRALIA

What is the new canine parvovirus and what do I need to do?

A recent study published by researchers from Adelaide University reported the detection for the first time in Australia of a variant of canine parvovirus (CPV) – canine parvovirus 2c (CPV-2c)[1]. As this is the first report of this particular variant in Australia, it has generated some interest in both the veterinary and lay press, as well as concern among veterinarians and pet owners as to what this means for the health of their patients and pets. Whilst this virus may be new to Australia, it has actually been present overseas since the turn of the century. Due to this extensive experience, we may reasonably draw conclusions about its possible effects in Australia. There is also a significant body of scientific literature regarding CPV-2c from which we can learn.

Boehringer Ingelheim hopes the following information will help address concerns regarding diagnosis of, and vaccination against canine parvovirus in light of the recent detection of CPV-2c in Australia.

What is CPV-2c? - Unscrambling the canine parvovirus alphabet!

Before addressing its implications, we should first consider what CPV-2c is. To help with this, an understanding of the evolution of canine parvovirus is beneficial – fortunately the history of this virus in dogs is relatively short! For vets and pet owners, it is hard to remember a time before canine parvovirus, however such a time did exist in the not too distant past. In fact, canine parvovirus as we know it today has only been with us for approximately 40 years, emerging and rapidly spreading in the dog population from the late 1970s. This new virus was named canine parvovirus 2, to differentiate it from canine parvovirus 1, also known as minute virus of canines, which had been identified around a decade earlier. Unlike CPV-1, CPV-2 was highly virulent in dogs of all ages, and rapidly and unmistakably made its presence felt. By the early 1980s, CPV-2 was replaced by an antigenic variant, CPV-2a, the latter differing by 5 amino acids from the original type[2]. This new antigenic variant out-competed the original virus, such that CPV-2 is now considered extinct in the wild. A second variant, CPV-2b, differing by 2 amino acids from CPV-2a, was first identified in the USA in 1984, while the latest variant, CPV-2c, differing by a single amino acid from CPV-2b, was first identified in Italy in 2000[2]. Since that time, CPV-2c has been documented throughout Europe, North America, South America, and parts of Asia, becoming the predominant type in some of these locations.

What does this mean for diagnosing dogs with CPV infections?

The clinical signs of canine parvovirus infection are the same regardless of the infecting type – i.e. it is not possible to differentiate between types based on clinical signs alone[3]. The classical signs of vomiting, anorexia, lethargy, and haemorrhagic diarrhoea may be seen, however there can be a marked variation in the clinical response, ranging from subclinical infections to mild clinical signs, right through to acute, fatal disease. The difference in the clinical picture seen in infected dogs is likely attributable to the host's immune response rather than a difference in the pathogen itself. Whilst the later antigenic types (2a, 2b, and 2c) have a shorter incubation period and may be associated with a more rapid disease progression than the original CPV-2, there is no strong evidence that there are variations in virulence between the more recent types. This has been the experience overseas, and, from the limited information available, there is no evidence thus far in Australia to suggest that the CPV-2c viruses are behaving any differently in regards to the clinical signs they cause.

Although it has often been said that vets can smell parvovirus, clinical signs, including smell, are insufficient for a definitive diagnosis. Signalment and history can provide supporting information (e.g. a young unvaccinated pup increases the likelihood of CPV infection), however confirmation of the diagnosis requires the detection of the organism or its genetic material in faeces from infected dogs. Most commonly this is performed using point-of-care kits which detect viral antigen in the faeces. These tests have high specificity, but relatively low sensitivity[3]. False negatives are not uncommon due to the limited period in which high titres of faecal antigen are shed, the intermittent nature of shedding, and through interference by faecal antibodies.

Studies overseas have shown that there is no significant difference in the ability of faecal antigen tests to detect the different CPV variants, with a study by Decaro et al. (2010) showing positive tests in 80.4% of dogs with CPV-2a, 78.0% in dogs with CPV-2b, and 77.0% in CPV-2c infections[4]. In the recently published Australian study, two of the three dogs infected tested positive with in clinic antigen tests, in line with previously reported data on their sensitivity.

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That a single dog tested negative in this paper is not surprising given the natural history of CPV infections, and should not be taken as proof that current diagnostic tests are inadequate for the detection of CPV-2c. For dogs testing repeatedly negative with faecal antigen testing, but for which a high index of suspicion remains, faecal PCR testing can be performed through commercial laboratories. PCR can detect lower quantities of virus (viral DNA to be precise), are unaffected by the presence of faecal antibodies, and remain positive for a longer period post infection than point-of-care antigen tests.

Vaccination against CPV-2c

The panzootic spread of CPV-2 in the late 1970s was associated with high morbidity and mortality in canine populations around the world. This was not surprising given it involved the introduction of a completely new virus into an immunonaïve population. Following its emergence, the first step in the control of CPV-2 was achieved through the use of heterologous vaccines (modified live or inactivated feline panleukopaenia/mink enteritis virus vaccines). Homologous vaccines, initially inactivated and then later modified live soon followed, and have proven to provide excellent protection against the existing strains to date. Improvements in vaccine technology, utilising high titre, low passage vaccines, have allowed vaccines to overcome higher titres of maternal antibodies, meaning pups can be protected earlier, allowing all important socialisation to occur.

Current parvovirus vaccines in Australia and elsewhere use either the original CPV-2 or newer CPV-2b antigenic types. Challenge studies have been reported with both types and have demonstrated cross protection against all existing CPV antigenic variants (2a, 2b, and 2c)[5, 6]. This position is supported by the current WSAVA guidelines which state *"All genotypes are antigenically related; challenge studies have shown that vaccination of dogs with current CPV vaccines containing either CPV-2 or CPV-2b will provide protective immunity against all the other variants, including CPV-2c"*[7]. Based on these studies, it would be expected that currently registered vaccines in Australia will provide adequate protection against CPV-2c.

The challenge studies referred to above are, by their very nature, limited in size; for ethical reasons rightly using only the minimum number of animals required to demonstrate statistical significance. What about the real world? We are fortunate that there is significant field experience with the use of CPV-2 and -2b based vaccines in CPV-2c endemic countries since 2000. In these countries there have not been wholesale episodes of vaccine failures in adult dogs. Occasional vaccine failures in adult dogs are reported in the literature, some of which have been attributed to newer types, however that these are still published as individual case reports speak to their rarity. This is not to say that vaccine failures can't occur. They can, and they do, albeit rarely. Invariably they occur in young dogs and are due to interference by maternal antibodies, and not due to a new and novel vaccine resistant virus. If there were a new vaccine resistant virus out there, vaccinated dogs of all ages would be getting sick, not just those who had a primary puppy course and no others. The viruses reported in the recent study by Adelaide University were identified in 2015. Since that time, there have been no reported mass outbreaks of CPV associated disease in Australia in vaccinated dogs (pups or adults), suggesting that current vaccines provide adequate protection against CPV-2c in Australia.

What do I need to do?

So getting back to the question posed at the beginning – what should a veterinarian do differently in response to this latest finding? The answer in most cases will be 'very little'. The clinical signs to look out for and the testing modalities for diagnosis do not change. Be aware that false negatives will occur, but the data suggests these are no more common with CPV-2c than any other variant. Remember PCR testing can be used in suspicious cases testing repeatedly negative with antigen testing. As for vaccination, it remains arguably the single most important thing we do for our patients. Vaccination against canine parvovirus has historically been extremely effective, and there is no data, from clinical studies or extensive overseas experience, to suggest that the presence of CPV-2c will impact upon this.

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