Vaccination against canine parvoviral enteritis in healthy dogs

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HUMAN and animal immunisation reduces the mortality from, and prevalence of, vaccine-preventable infections. Even though vaccines are generally considered safe, in the past there has been strong opposition coming from several lobby groups against the use and the implementation of vaccination (Jeffrey 2005).

Vaccinations may sometimes be misused (for a variety of reasons) particularly when a risk analysis of the vaccine’s benefit is not performed. For example, a vaccination may be unnecessary if an animal already has antibodies against the infectious disease, but usually vets do not know the immune status of their patients.

This situation is discussed in the paper by Riedl and others (2015) summarised on page 597 of this issue of Veterinary Record. The authors gathered field parvoviral immunity status in a German dog population and, based on their results argue that, antibody status against canine parvovirus (CPV) should be determined instead of periodic ‘blind’ vaccination to ensure reliable protection without unnecessary vaccination in adults animals. Such an approach could prove useful in reducing the adverse effects of vaccination, such as a hypersensibility and immunosuppression in dog, as well as improving public opinion. However, misinformed dog owners could potentially avoid having their dogs vaccinated leading to low antibody coverage rates, below those required to achieve herd immunity.

Riedl and others found that a number of dogs showed a high titre of antibodies against CPV without a history of previous vaccinations. They therefore highlight that some dog’s ‘natural booster’ enhances the production of CPV antibodies without the need for vaccination. It is well know that wild and vaccinal strains of CPV are able to survive for several years (Decaro and Buonavoglia 2012) and the possibility of reinfection is high. Therefore, the titre
of antibodies in dogs that are vaccinated yearly does not necessarily lead to a beneficial booster effect, as Bernstein and others (2003) demonstrated in people.

Riedl and others (2015) also report that body weight might affect vaccination response. They found that heavier dogs (over 30 kg) did not express an increase in titre vaccination when compared to dogs that weighed 10 kg or less (Kennedy and others 2007). Recently a study showed that a reduced immune response to a candidate HIV-1 vaccine was observed among overweight, uninfected people (Kennedy and others 2007). Recently a study showed that a reduced immune response to a candidate HIV-1 vaccine was observed among overweight, uninfected people (Kennedy and others 2007). Obesity is considered a chronic inflammatory state that can lead to impairments in innate and adaptive immune functions (Eliakim and others 2006).

Excess peripheral adiposity can lead to inadequate vaccine delivery intramuscularly or subcutaneously reducing antigen uptake and presentation to the immune system (Zuckermann 2000). However, more research is needed (particularly in the veterinary field) to demonstrate a clear association between obesity and an impaired immune response (Gowda and others 2015). Also, the dogs studied in Riedl and others study varied in size, but the bigger dogs were not necessarily obese so this needs further consideration too.

Another interesting finding was the complete lack of antibodies in very few cases considering most of the dogs shared an incomplete and/or incorrect vaccination schedule. Even if the lack of antibodies does not mean a lack of immunity, as the cell-mediated immunity was not evaluated, it is important to mention that a correct vaccination schedule (especially in puppies) is a milestone for a complete immunological coverage in dogs.

In conclusion, Riedl and others have helped to introduce a new concept: to evaluate the immunity of a dog before its first and subsequent vaccinations against the CPV infection. In my opinion this practice would be a feasible method for all infectious diseases as long as the aetiological agent is genetically stable. For example, with cat calicivirus infections, serological data in predicting protection are limited because antibodies to the calicivirus strain used in laboratory tests might not necessarily protect cats against the strains they will subsequently be exposed to in the field (Radford and others 2009). Further research is necessary, but a different and additional way of managing vaccination protocols has been explored here.

References


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