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Excretion of canine parvovirus type 2 (CPV-2) during gestation and lactation in bitches and puppies
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Canine parvovirus type 2 (CPV-2) is a frequent intestinal pathogen associated with high pup mortality. Transmission control by disinfection and patient isolation is of limited efficiency, raising questions about other possible contagion sources. The aim of our study was to determine whether vaccinated dams could excrete CPV2 from mating to end of lactation and so be a potential source of infection for their offspring. A total of 73 bitches (mean ± standard deviation: 4.4±1.9 years old; from 15 breeds) housed in the same kennel throughout the reproductive cycle were enrolled in the study. All were annually vaccinated (Nobivac DHPPi-Lepto vaccine; MSD, Beaucouzé, France). Forty-one dams were studied from mating to whelping (Week0-Week8), and 32 dams from whelping until weaning (W9-W16). Rectal swabs were collected every 14 days from dams during gestation (n=41), and every 7 days from lactating dams (n = 32) and their 3-8 week old pups (n=135). Rectal swabs were tested by real time PCR for CPV-2 genomic DNA (capsid) [1]. Using this method, the quantification threshold has been previously established as 2x10^5 copies/g feces and the viral load associated with clinical parvovirosis is 5x10^8 copies/g feces [1]. The number of copies per gram of feces per dog were analyzed through logistic regression and mixed linear models (R, R Foundation for Statistical Computing, Auckland, NZ). Of dams sampled during pregnancy, CPV2 went above the quantification threshold only in one sample vs 64% of dams tested during lactation. During lactation, excreted viral loads were higher at W14 (5x10^8/g feces, p=0.001), W15 (8x10^8/g feces, p<0.001) and W16 (10^9/g feces, p<0.001) than earlier in lactation (<10^6 copies/g feces; W9-12). None of the bitches, including those excreting viral loads above the threshold for clinical parvovirosis, expressed any clinical sign. In 28% of the cases, the dam excreted before her puppies. Viral loads excreted by puppies were not correlated with those excreted by dams. While the percentage of pups with >5x10^8 CPV2 copies/g feces increased from 2-76% per litter from W10 to W16, the overall mortality was only 3% (4/134) and 14% of the puppies experienced at least one episode of diarrhea. This study indicates that vaccinated dams may contribute to CPV2 circulation. Their role in its persistence in the kennel (between the end of lactation until next whelping) remains to be explored. The role of systemic and local immunity in the control of viral excretion and into the control of clinical expression would also be interesting to evaluate, both in dams and in puppies (quality of the passive immune transfer).