

VV114 - MICROSCOPIC LESIONS AFTER CHALLENGE WITH TWO BRAZILIAN AVIAN INFECTIOUS BRONCHITIS VARIANTS

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Avian Infectious Bronchitis (IBV) is one of the greatest health challenges on poultry industry. The emergence of new variants has been frequent recently, therefore studies reporting pathogenesis are important to characterize the most representative IBV strains. The aim of this study was to evaluate microscopic lesions in SPF chickens challenged with two Brazilian IBV field isolates previously genotyped as IBV variants. Sixty two one-day old SPF chickens were divided into three groups and housed in positive pressure isolators. Groups A and B were challenged at 28 days of life with 104.0IED50 of 3735 and 3736 IBV variants, respectively. Group C was kept as mock infected negative control group. Nine birds from each group were euthanized at 1 day-post infection (dpi) and at 5 dpi, remaining six birds at 8 dpi. All birds were necropsied and trachea, sinuses, Harderian gland, lung, kidney and gonads were collected, fixed in buffered formalin and processed by routine histology. At 1 dpi acute tracheitis was observed in few birds of groups A and B. Exudation of mucus and heterophils; degeneration, necrosis and sloughing of epithelial cells and heterophilic infiltrate in the mucosa were predominant lesions. At 5 dpi, all birds of groups A and B had chronic tracheitis, with complete ciliary loss, lymphocytes and plasma cells infiltrating the mucosa and epithelial hyperplasia. In sinuses, fifty percent of the birds from groups A and B had mild chronic inflammation, the same trend was observed at 8 dpi in this tissue. At 8 dpi tracheal mucosa was regenerating, showing glandular degeneration, lymphocytic infiltration and mild epithelial hyperplasia. Chronic interstitial nephritis was observed in few birds of groups A and B at 8 dpi. Mild lymphocytic infiltration was observed in the Harderian gland of all birds of challenged groups. No lesions were observed in the tissues of group C and in lungs and gonads of all groups. Significant positive correlation (P

< 0.0001) was reported between scores of microscopic lesions and ciliary activity (gold standard method) on tracheal samples at 5dpi, by Pearson test. No significant microscopic differences were found between these two IBV variants tested. The results indicate that, besides the genotypic variation, lesions produced by the IBV variants here studied were similar to those described for standard IBV strains with respiratory tropism, producing main lesions in the upper respiratory tract and, renal damage in few birds. FINANCIAL SUPPORT: PROJETO EMBRAPA 03.12.03.012.0.00

VV119 - OCCURRENCE OF CANINE HERPESVIRUS INFECTION IN DOGS OF BREEDING KENNELS IN TROPICAL CLIMATE ZONE

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The canine herpesvirus (CHV) is a cause of abortion in dogs, but can be detected in healthy animals. This virus is associated to different clinical abnormalities as death of newborns and lesions in respiratory and reproductive tracts in adult dogs. There is no vaccine against CHV in Brazil due to scarcity of studies confirming the importance of virus in breeding kennels. Thus, this study aimed to correlate the abortion incidence with percentage of positive dogs to CHV at Ribeirão Preto region. Adult (1 to 5 years-old) dogs (39 males and 209 females) from different breeds (Schnauzer, Pug, Poodle, Yorkshire Terrier, Lhasa Apso, Shih-Tzu, Maltese, German Spitz and French Bulldog) from 9 commercial kennels were used. Blood samples were obtained by jugular vein puncture and placed into vials containing 10% EDTA. The DNA extractions were made with Wizard® Genomic Purification Kit (Promega, Madison, WI, USA). The extracted DNA was subjected to qPCR using primers directed to the thymidine kinase CHV3 (CGTGGTGAATTAAGCCTAA) and CHV4 (ATGCTATTGGGTGTCTATC), and GoTaq™ qPCR master mix kit (Promega, Madison, WI, USA) in the ABI 7300 (Life Technologies™, SP, Brazil) equipment. As a negative control reaction, nuclease-free water was used. The reaction conditions for amplification of the gene fragment for thymidine kinase CHV were initial denaturation at 95°C/2 min, followed by 40 cycles of