T-cell Dependent Antibody Responses in dogs administered lokivetmab (ZTS-00103289), a caninized, anti-canine IL-31 monoclonal antibody

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Interleukin 31 (IL-31), a cytokine involved in atopic dermatitis, affects interactions between T-cells, mast cells, eosinophils, and epithelial cells. Additional functions may include immunomodulation. This study evaluated whether inhibition of IL-31 signaling by lokivetmab impaired the T-cell dependent antibody response in dogs to Keyhole Limpet Hemocyanin (KLH), a model antigen. The study complied with GLP and local, national, and international animal welfare regulations. One-year-old laboratory Beagle dogs, immunologically naïve to KLH, were divided into four groups (n = 4M/4F per group). On Days 0 and 21, two groups received lokivetmab (10 mg/kg) and two groups received saline. On Days 5 and 26, one each of lokivetmab saline groups were immunized with 0.1 mg un-adjuvanted KLH; the remaining groups received 1 mg un-adjuvanted KLH. Anti-KLH IgG antibody titers were determined weekly from Days 5 to 42. Baseline pre-immunization anti-KLH titers in all groups (geometric mean, 95% confidence limits) were 100 (54.1, 184.1). All dogs in all groups developed anti-KLH titers. At 0.1 mg KLH immunization, Day 33 titers in lokivetmab-treated dogs were 55,393 (30,223, 101,526), while titers in saline-treated control dogs were 42,089 (8100, 218,700). At 1.0 mg KLH immunization, Day 33 titers in lokivetmab-treated dogs were 166,176 (90,667, 304,569), while titers in saline-treated control dogs were 190,638 (104,014, 349,402). Lokivetmab (10 mg/kg) did not impair the T-cell dependent antibody response to KLH at 0.1 mg or 1 mg doses of un-adjuvanted KLH in normal 1-year old Beagle dogs.

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