Dog dementia risk increases with each added year of life

MVC, August 26th 2022

For dogs older than ten years, each extra year of life increases the relative risk of developing the neurodegenerative condition Canine Cognitive Dysfunction (CCD) by more than 50% and the risk of developing CCD is almost 6.5 times greater in inactive dogs compared to those who are very active. The new findings, published in Scientific Reports, suggest that lifespan estimates could inform veterinarians whether to screen dogs for CCD.As with humans, cognitive function declines as dogs age and animals affected by CCD may show signs such as memory deficits, loss of spatial awareness, altered social interactions, and sleep disruption. Previous estimates of CCD rates in dogs have varied from 28% in 11- to 12-year-old dogs to 68% in 15- to 16-year-old dogs.The study suggests that when considering age alone among dogs aged more than ten years, the odds of being diagnosed with CCD increased by 68% for each additional year of age. When controlling for other factors such as health problems, sterilisation, activity levels, and breed type, the odds of a dog developing CCD increased by 52% for each additional year of life. Researchers also note that for dogs of the same breed, age, and health and sterilization status, the odds of CCD were 6.47 times higher in dogs whose owners reported were not active compared to those whose owners reported were very active.

Hypocobalaminaemia in cats and dogs

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Cobalamin, or vitamin B12, is a key water-soluble vitamin responsible for multiple functions in the body. As an essential enzyme co-factor, it is involved with cellular energy production, DNA synthesis, nervous system function, amino acid and lipid metabolism, and in erythrocytosis.Hypocobalaminaemia is becoming increasingly recognised in small animal medicine, as it can be caused by a multitude of diseases.The specific diseases include exocrine pancreatic insufficiency (EPI), dysbiosis, chronic enteropathies, intestinal neoplasia and inherited disorders of cobalamin absorption. Breeds in which these inherited disorders can be seen include the border collie, giant schnauzer, Shar Pei and beagle. It is, therefore, recommended to monitor serum cobalamin concentration in patients with EPI and chronic gastrointestinal disease.Patients with hypocobalaminaemia often do not respond to treatment of their underlying GI disorder if low cobalamin concentrations are not corrected.The clinical signs of hypocobalaminaemia in dogs and cats can be vague and non-specific, including: inappetence/anorexia, lethargy, weight loss, gastrointestinal signs (vomiting, diarrhoea), poor immune function, failure to thrive, poor body condition score, anaemia, hypoglycaemia. Prevalence of hypocobalaminaemia: Various studies have investigated the prevalence of low B12 concentrations in different disease processes. EPI: 82% of dogs5, 100% of cats. Chronic inflammatory enteropathy: 19% to 38% of dogs, increasing to 43% to 75% of dogs with protein losing enteropathy (PLE), Feline gastrointestinal disease: 61% of cats, Intestinal lymphoma: 40% to 71% of dogs. Furthermore, hypocobalaminaemia has been shown to be a negative prognostic indicator in dogs with EPI and chronic enteropathies.

STUDY

Health impact assessment of pet cats caused by organohalogen contaminants by serum metabolomics and thyroid hormone analysis (Science of The Total Environment, July 2022)

Summary: Exposure to persistent organic pollutants in pet cats lowers blood thyroid hormone levels and causes chronic oxidative stress, a new study finds.

Abstract: Companion animals are in close contact with the human surroundings, and there is growing concern about the effects of harmful substances on the health of pet cats. In this study, we investigated the potential health effects of organohalogen compounds (OHCs) on thyroid hormone (TH) homeostasis and metabolomics in Japanese pet cats. There was a significant negative correlation between concentrations of several contaminants, such as polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), hydroxylated PCBs (OH-PCBs), hydroxylated PBDEs (OH-PBDEs), and THs in cat serum samples. These results suggested that exposure to OHCs causes a decrease in serum TH levels in pet cats. In this metabolomics study, each exposure level of parent compounds (PCBs and PBDEs) and their hydroxylated compounds (OH-PCBs and OH-PBDEs) were associated with their own unique primary metabolic pathways, suggesting that parent and phenolic compounds exhibit different mechanisms of action and biological effects. PCBs were associated with many metabolic pathways, including glutathione and purine metabolism, and the effects were replicated in in-vivo cat PCB administration studies. These results demonstrated that OHC exposure causes chronic oxidative stress in pet cats. PBDEs were positively associated with alanine, aspartate, and glutamate metabolism. Due to the chronic exposure of cats to mixtures of these contaminants, the combination of their respective metabolic pathways may have a synergistic effect.

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