Serum melatonin concentrations in dogs with congenital portosystemic shunts

Hepatic encephalopathy (HE) is a syndrome of neurologic dysfunction present in several liver diseases and an important contributor to patient morbidity. Canine HE is most commonly reported in association with congenial portosystemic shunting (cPSS). Although not completely characterised, several factors are known to influence the pathogenesis of HE, namely increased ammonia, inflammation and manganese, as well as alkalosis, hyponatraemia and hypokalaemia. Melatonin is a hormone secreted by the pineal gland in the brain and by the enterochromaffin-like cells in the gastric mucosa, with actions linked with regulation of the circadian rhythm, enterocyte integrity, and free oxygen radical scavenging, amongst others. The majority of melatonin is metabolised in the liver and recently, melatonin levels were noted to be significantly increased in human alcohol-induced liver cirrhosis, correlating with both disease severity and with the severity of HE, hence hypothesised as a potential contributor to the development of the latter. To the authors' knowledge, melatonin homeostasis in canine liver disease is currently unknown. The aim of this study was to investigate whether melatonin concentrations could be altered in dogs with cPSS, with the hypothesis that higher levels would be present when compared to healthy controls.Medical records were retrospectively reviewed for inclusion into two cohorts: dogs with a confirmed diagnosis of cPSS (n = 24) and healthy dogs examined through wellbeing appointments (n = 15). A canine competitive enzyme-linked immunosorbent assay was used to measure serum melatonin prospectively. 100uL of sample was used from an archive of surplus samples retained after clinical diagnostic purposes. Informed owner consent for surplus retention and use for research had been obtained at the time of clinical sampling. Melatonin concentrations in each group were assessed for normality with the Anderson-Darling test. Both groups were not normally distributed, therefore data was described as median (minimum - maximum ranges) and differences between groups compared with the Mann-Whitney U test. Statistical significance level was set at $P < 0.05$.The concentrations of melatonin in the cPSS group (25 pg/mL [18.5 – 244.9 pg/mL]) did not differ significantly ($P = 0.7839$) from the healthy controls (27.2 pg/mL [19.8 – 161.5 pg/mL]).This study suggests that serum melatonin is not increased in dogs with cPSS and is unlikely to play a role in HE pathogenesis.