The effect of ammonia on canine polymorphonuclear cell oxidative burst and the implications in hepatic encephalopathy

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OBJECTIVES

To assess the effect that escalating concentrations of ammonia have on the oxidative burst generated by polymorphonuclear cells (PMNCs).

METHODS

Surplus whole blood was incubated with escalating concentrations of ammonia ranging from 0 to 250 μM , in 25 μM increments, for 90 minutes.

A commercial kit was used to evaluate the oxidative burst formed by both the cells stimulated with *E coli* and those without stimulation.

The PMNC generated oxidative burst was quantified based upon the oxidation of one of the reagents, dihydrorhodamine 123, to the fluorescent rhodamine 123.

The red cells were lysed and centrifuged to obtain the PMN cell pellet.

This was then resuspended and evaluated using flow cytometry and subsequently analysed and compared.

The pH of the ammonia treated blood was evaluated to ascertain whether this was significantly different and may have had an effect.

RESULTS

When evaluating the spontaneous burst, in the absence of stimulation, there was a significant increase observed at 125 μM ($p < 0.05$), which increased further at 150 μM ($p < 0.01$) and again above 175 μM ($p < 0.001$).

There was no significant difference from baseline in those cells stimulated with *E coli* at the various ammonia concentrations ($p = 0.953$).

The pH was not significantly different between the blood incubated at different concentrations of ammonia ($p = 0.2$).

STATEMENT (CONCLUSIONS)

Increasing ammonia exposure resulted in an increase in the spontaneous oxidative burst, resulting in an aberrant inflammatory response, which can precipitate hepatic encephalopathy and play a role in promoting a pro-inflammatory state which can worsen liver disease.