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Impact of L-arginine (a nitric oxide synthesis precursor) and N-ω-nitro-L-arginine-methyl-ester (L-NAME) (a non-selective nitric oxide synthase inhibitor) chronic, subchronic and acute toxicity-induced lesions on ascites-pulmonary Hypertension Syndrome Development in Broiler Chickens

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**Experiment A.** Chronic toxicity. 140 one day-old male broiler chicks divided in four groups. Every other day: intraperitoneal (ip) L-NAME (LN, 10 mg/kg BW); L-arginine (LA, 100 mg/kg BW), L-arginine and a L-NAME combination (100 and 10 mg/kg BW, respectively), and physiological saline (0.90% w/v; 0.5 mL/kg BW). Seven birds were euthanized weekly during their first five weeks of life.

Experiment B. Subchronic toxicity. Equal doses of the same substances: ip every eight hours into 38 day-old birds over 36 hours.

**Experiment C.** Acute toxicity. Thirty day-old birds: one ip dose of various concentrations of L-NAME (50, 100 and 150 mg/kg BW); L-arginine (100 mg/kg BW) and saline (0.5 ml/kg BW). All the birds were euthanized after six hours.

**The end of experiment A:** the gross finding of ascites-pulmonary hypertension syndrome (PHS) confirmed vasoconstrictory effect of L-NAME in five birds (LN). Towards the end of experiment A; in experiment C: histopathological findings (myocardial/ pulmonary oedema/LN/; congestion/haemorrhages/LA/) the most prominent (higher LN-doses/100 and 150 mg/kg BW/). Irreversible myocardiolysis and hepatocellulolysis: confirmed in all three experiments (LN). Focal myocardial degeneration solely: in the L-NAME/L-arginine simultaneously treated group.

Haematological and blood chemistry values; stress index value; and relative organ weights agreed with well-known literature data on PHS. Experiments A and C. The severity of LN-provoked hypoxic changes and plentiful haemorrhages (LA) - dose and time dependent. Mild changes in L-NAME/L-arginine group confirmed a protective role of LA.

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## Modulatory effects of equithrive joint on oxidative stress and inflammatory biomarkers in aged lame horses

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This study was aimed to evaluate themodulatory effects of Equithrive joint supplement on oxidativestress and inflammatory biomarkers in aged lame horses. A total of 16 horses of both sexes, weighing between 350 and 450 kg were used. Eight of the horses were administered with equithrive joint for four weeks and eight others served as untreated (controls), and were given only Saccharomyces cerevisiae yeast strain used as carrier in the supplement. Blood samples were collected from each horse before supplementation (week 0) and at first, second, third and fourth week of the experiment. Activities of oxidative biomarkers (catalase, glutathione peroxidase, malondialdehyde and superoxide dismutase) and concentration of the inflammatory biomarker, tumor necrosis factor-alpha were determined by standard methods. Equithrive joint administration increased (P<0.05) the activities of catalase and superoxide dismutase, while glutathione peroxidase activity, malondialdehyde and tumour necrosis factor alpha concentrations reduced (P<0.05) in the treated horses, compared with the controls (untreated). The result indicated that Equithrive joint is a potent antioxidant and anti-inflammatory agent, which may be of value in reducing inflammatory mediators; thereby improving locomotion in aged lame horses.

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