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ABSTRACTS

Page 1 of 4

Pet Health

Effect of antioxidants on the proliferative response of canine lymphocytes in serum from dogs with vitamin E deficiency.

The in vitro effect of vitamin E and 3 other antioxidants-ethoxyquin, 2-mercaptoethanol, and ascorbic acid-on proliferation of canine lymphocytes was examined. Lymphocytes from 2 groups of dogs given a vitamin E-deficient diet or whelped from a bitch fed such a diet were cultured with pooled samples of serum from dogs fed a vitamin E-deficient diet or whelped from a bitch fed such a diet, or normal canine serum, and stimulated with phytohemagglutinin. Added vitamin E enhanced the responsiveness in serum from the dogs with vitamin E deficiency, but not in normal canine serum. A similar effect was noted with added ethoxyquin and 2-mercaptoethanol. Ascorbic acid had no effect on proliferation in either serum pool. These results indicated that depressed lymphocyte responsiveness seen with serum from vitamin E-deficient dogs may, at least in part, be due to a loss of antioxidant activity in this serum.

Am J Vet Res 1983 Jan;44(1):5-7

Nitric oxide modulates epithelial permeability in the feline small intestine.

The objective of this study was to assess whether inhibition of nitric oxide production leads to increased epithelial permeability in feline small intestine. Local intra-arterial infusion of the nitric oxide synthesis inhibitor NG-nitro-L-arginine-methyl ester (L-NAME; 0.025 $\mu\text{mol}\cdot\text{ml}^{-1}\cdot\text{min}^{-1}$) was performed in autoperfused segments of cat ileum for 90 min. An exogenous source of nitric oxide, sodium nitroprusside (SNP) was infused (0.025 $\mu\text{mol}\cdot\text{ml}^{-1}\cdot\text{min}^{-1}$) for the last 30 min of the 90-min L-NAME infusion. Epithelial permeability was quantitated by measuring blood-to-lumen clearance of ^{51}Cr -labeled EDTA throughout the experiment. An increase of approximately sixfold in mucosal permeability was observed within 30 min of L-NAME infusion and this effect was completely reversed by infusion of either SNP or L-arginine (0.125 $\mu\text{mol}\cdot\text{ml}^{-1}\cdot\text{min}^{-1}$). NG-nitro-D-arginine-methyl ester (D-NAME) had no effect on mucosal permeability. The increase in epithelial permeability was sufficiently large that rhodamine-dextran (mol wt = 17,200) clearance from interstitium to lumen was increased. Pretreatment with IB4, a monoclonal antibody directed against the leukocyte adhesive glycoprotein complex (CD11/CD18) did not prevent the L-NAME-induced increase in epithelial permeability. These data suggest that inhibition of nitric oxide production leads to a reversible circulating leukocyte-independent increase in epithelial permeability.

Am J Physiol 1992 Jun;262(6 Pt 1):G1138-42

Dietary beta-carotene absorption by blood plasma and leukocytes in domestic cats.

Three experiments were conducted to study the uptake of oral beta-carotene by blood plasma and leukocytes in domestic cats. In Experiment 1, mature female Tabby cats (12 mo old) were given once orally 0, 10, 20 or 50 mg of beta-carotene and blood taken at 0, 12, 24, 30, 36, 42, 48 and 72 h after dosing. Concentrations of plasma beta-carotene increased in a dose-dependent manner. Peak concentrations were observed at 12-24 h and declined gradually thereafter. The half-life of plasma beta-carotene was 12-30 h. In Experiment 2, cats were dosed daily for six consecutive days with 0, 1, 2, 5 or 10 mg beta-carotene. Blood was sampled once daily at 12 h after each feeding. Daily dosing of cats with beta-carotene for 6 d resulted in a dose-dependent increase in circulating beta-carotene. Experiment 3 was designed to study the uptake of beta-carotene by blood leukocytes. Cats were fed 0, 5 or 10 mg of beta-carotene daily for 14 d. Blood leukocytes were obtained on d 7 and 14 to determine beta-carotene content in whole lymphocytes and in subcellular fractions. Blood lymphocytes took up large amounts of beta-carotene by d 7 of feeding. Furthermore, beta-carotene accumulated mainly in the mitochondria (40-52%), with lower amounts accumulating in the microsomes (20-35%), cytosol (15-34%), and nuclei (1.5-6%). Therefore, domestic cats readily absorb beta-carotene across the intestinal mucosa and transfer the beta-carotene into peripheral blood leukocytes and their subcellular organelles. Beta-Carotene uptake kinetics show that some aspects of beta-carotene absorption and metabolism in cats are similar to those of humans.

J Nutr 2000 Sep;130(9):2322-5

Assessment of degree of oxidative stress and antioxidant concentrations in dogs with idiopathic dilated cardiomyopathy.

OBJECTIVE: To assess degree of oxidative stress and antioxidant concentrations in dogs with idiopathic dilated cardiomyopathy (IDCM). **DESIGN:** Prospective study. **ANIMALS:** 18 dogs with IDCM and 16 healthy control dogs. **PROCEDURE:** Concentrations of malondialdehyde (an indicator of oxidative stress); vitamins A, C, and E; glutathione peroxidase; and superoxide dismutase were measured. **RESULTS:** Glutathione peroxidase concentration was significantly increased in dogs with IDCM, compared with control dogs. Vitamin A and superoxide dismutase concentrations were not significantly different between groups. A negative correlation was found between disease severity and plasma vitamin E concentration. Disease severity was not correlated with concentrations of other antioxidants. Medications did not significantly affect oxidant or antioxidant concentrations. **CONCLUSIONS AND CLINICAL RELEVANCE:** The change in glutathione peroxidase concentration and the correlation between vitamin E concentration and disease severity suggest that the oxidant-antioxidant system may play a role in development of IDCM.

J Am Vet Med Assoc 1999 Sep 1;215(5):644-6

Dietary lutein stimulates immune response in the canine.

The possible immuno-modulatory action of dietary lutein in dogs is not known. Female Beagle dogs (17-18-month old; 11.4±0.4kg body weight) were supplemented daily with 0, 5, 10 or 20mg lutein for 12 weeks. Delayed-type hypersensitivity (DTH) response to saline, phytohemagglutinin (PHA) and a polyvalent vaccine was assessed on Weeks 0, 6 and 12. Blood was sampled on Weeks 0, 2, 4, 8 and 12 to assess (1) lymphocyte proliferative response to PHA, concanavalin A (Con A), and pokeweed mitogen (PWM), (2) changes in peripheral blood mononuclear cell (PBMC) populations, (3) interleukin-2 (IL-2) production and (4) IgG and IgM production. After the completion of 12-week study, we continued to collect the blood weekly up to 17 weeks to evaluate the changes in immunoglobulin production upon first and second antigenic challenges on Weeks 13 and 15. Plasma lutein+zeaxanthin was undetectable in unsupplemented dogs but concentrations increased ($P<0.05$) rapidly on Week 2 in lutein-supplemented dogs. Thereafter, concentrations generally continued to increase in dose-dependent manner, albeit at a much slower rate. Dogs fed lutein had heightened DTH response to PHA and vaccine by Week 6. Dietary lutein increased ($P<0.05$) lymphocyte proliferative response to all three mitogens and increased the percentages of cells expressing CD5, CD4, CD8 and major histocompatibility complex class II (MHC II) molecules. The production of IgG increased ($P<0.05$) in lutein-fed dogs after the second antigenic challenge. Lutein did not influence the expression of CD21 lymphocyte marker, plasma IgM or IL-2 production. Therefore, dietary lutein stimulated both cell-mediated and humoral immune responses in the domestic canine.

Vet Immunol Immunopathol 2000 May 23;74(3-4):315-27

The riboflavin requirement of adult dogs at maintenance is greater than previous estimates.

A study was conducted to determine the riboflavin requirement of adult dogs at maintenance. Twenty adult mixed breed dogs were fed a semipurified meal with one of five riboflavin concentrations: Diet 1, 1.7 mg/kg; Diet 2, 2.7 mg/kg; Diet 3, 3.7 mg/kg; Diet 4, 4.7 mg/kg; and Diet 5, 5.7 mg/kg. The erythrocyte glutathione reductase activity coefficient (EGRAC) was used to determine biochemical riboflavin deficiency. Dogs fed Diet 1 had a greater ($P < 0.05$) EGRAC (1.24) on d 56 of the trial compared with that of dogs fed Diet 5 (1.11), indicating marginal riboflavin deficiency in dogs fed Diet 1. On d 84 the mean EGRAC for dogs fed Diet 1 (1.36) was different from EGRAC obtained for dogs fed the other diets (1.19, $P < 0.05$). The difference in mean EGRAC was still present on d 112 (1.59 vs. 1.27; $P < 0.01$). There was no difference in d 112 mean EGRAC for dogs fed Diets 2, 3, 4 and 5 ($P < 0.05$). The broken line requirement estimate for the adult dog at maintenance was determined to be 66.8 microgram riboflavin x kg body wt⁻¹ x d⁻¹ using the d 112 EGRAC as the basis for assessing biochemical riboflavin deficiency.

J Nutr 1996 Apr;126(4):984-8

Changes in the defense against free radicals in the liver and plasma of the dog during hypoxia and/or halothane anaesthesia.

Defenses against free radicals were evaluated in the dog under different conditions of ventilation. Changes in the levels of reduced glutathione (GSH), alpha-tocopherol (vitamin E), ascorbic acid (vitamin C) and the lipid peroxidation end-products, estimated as malondialdehyde (MDA) and the activity of superoxide dismutase (SOD), were studied in serial liver biopsies from dogs ventilated with either oxygen, halothane and oxygen, hypoxic gas mixture of 8% oxygen and 92% nitrogen or halothane under hypoxic conditions. Simultaneous determination of GSH, vitamin E and MDA were carried out in the plasma. The results showed time-dependent depletion of GSH and vitamin E in liver and plasma and vitamin C in the liver. This was accompanied by a simultaneous increase in the levels of MDA. The magnitude of the change was in the following order: halothane and hypoxia > hypoxia > halothane and oxygen > oxygen. The greatest depletion was observed for vitamin E and the least for vitamin C. The rise in the level of MDA in plasma was much higher than in the liver tissue. Hypoxia resulted in inhibition of liver SOD activity. It seems that increased production of free radicals under hypoxic conditions may have overwhelmed the anti-oxidant defenses in the liver. In addition, the much higher level of MDA in plasma, as compared to liver tissue, may indicate that MDA could have originated in tissues or organs other than the liver and leaked into the blood, indicating possible damage in other locations in the body.

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