DOWNLOAD PDF VITAMIN E, BIOCHEMICAL, HEMATOLOGICAL, AND CLINICAL ASPECTS

Chapter 1 : Library Resource Finder: Table of Contents for: Vitamin E, biochemical, hematological, a

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Page 30 Share Cite Suggested Citation: Vitamin Tolerance of Animals. The National Academies Press. These diseases include those prevented by vitamin E or certain synthetic antioxidants e. The role of vitamin E in human health is most apparent in conditions of poor enteric absorption of lipids, for example, biliary atresia, cystic fibrosis, and neonatal prematurity. Similar condi- tions of lipid malabsorption in animals, such as pancrea- titis or bile stasis, may be expected to impair the utilization of dietary vitamin E. How- ever, the metabolic basis for the nutritional interrela- tionships of vitamin E and selenium was not understood until Rotruck et al. Investigations of this interrelationship have led to the present understanding that vitamin E and selenium via glutathione peroxidase function as parts of a multicomponent antioxidant defense system. The different types of vitamin E-deficiency syn- dromes that are manifested in different animals have been taken to indicate that, in various species and organ systems, lesions in different aspects of the cellular aIlti 23 24 Vitamin Tolerance of Animals oxidant defense system may occur. Vitamin E is thought to be involved specifically in the protection against peroxidative deterioration of polyunsaturated phospho- lipids in cellular membranes. Lipid peroxidation initi- ated within the membrane is not presumed to be affected by the selenium-dependent glutathione perox- idase, which is present only in the cytosol and mitochon- drial matrix space. Lesions of this nature are thought to result in the deficiency syndromes described above that respond only to vitamin E or to fat-soluble synthetic antioxidants capable of entering membranes. Lesions that involve both the membrane and soluble compo- nents of cells are believed to result in the previously noted deficiency syndromes that respond to either vita- min E or selenium. Eight or more compounds in this category are found widely distributed in nature. Each of the free tocopherols is unstable to oxidizing conditions; hence, the vitamin E activity of foods and feedstuffs depends upon both the chemical form provided and the storage conditions of the product. In practice, the vitamin E contents of prac- tical feedstuffs is variable and not readily predictable. The form generally used for this purpose is the fully racemic form, all-rac-ol-tocopheryl acetate. This ester, which is not an antioxidant, is stable to oxidizing condi- tions. Vita- min E is absorbed as the free alcohol, tocopherol. Its enteric absorption, like that of other fat-soluble nutrients, therefore is dependent upon its micellar solubilization. Consequently, impairment of pancreatic function or bile production will result in im- paired absorption of vitamin E. The efficiency of ab- sorption of tocopherols is relatively low at 20 to 40 percent Gallo-Torres, a. Absorption is increased by medium-chain triglycerides and is decreased by high levels of linoleic acid. In mammals, absorbed tocopherol is transported by chylomicrons via the lymphatic circu- lation to the liver and subsequently to the general circu- lation in very low density lipoproteins VLDL. In birds and fish, absorbed lipids are conveyed via the portal vein to the liver. It is present in tissues as free tocopherol. The species have twice these levels in the liver and heart but only half the levels in the skeletal muscle. Although tocopherol is associ- ated with the lipid phase of cells, tissue tocopherol con- centrations do not relate directly to tissue lipid levels. The basis for the variation of tocopherol concentrations between tissues is poorly understood; nevertheless, all tissues show linear increases in tocopherol concentra- tions with increases in tocopherol intake. Tocopherol acts in the transfer of hydrogen for the reduction of free radicals within the cell. Other metabolites have been reported; these have been reviewed by Gallo-Torres b. Growth was depressed by 2, IU kg, however. At this growth-depressing level of intake, re- duced hematocrit, reticulocytosis, and increased pro 26 to. Vitamin K injections corrected the prothrombin times. The high level of vita- min E depressed bone calcification among chicks fed either a calcium- or vitamin D-deficient diet. Skeletal muscle mitochondria isolated from chicks fed the high level of vitamin E showed a 33 percent reduction in oxygen uptake. That level of vitamin E also depressed femur ash content by 16 months and

decreased prothrombin times at 12 months. Yang and Desai a,b found that high-level vita- min E treatment did not significantly affect liver vita- min A storage or urinary creatine or creatinine. This level of the vitamin signif- icantly reduced the relative weight of the adrenal gland but did not affect the relative weights of liver, kidney, spleen, or testes. Although the total protein concentra- tion of plasma was not significantly affected, the high level of vitamin E increased albumin concentrations and decreased globulin concentrations. The result was a 50 percent increase in the albumin: Alam and Alam found the same dietary level of vitamin E to produce no deleterious ef- fects on ash or mineral contents of developing rat teeth. Martin and Hurley studied the effects of exces- sive amounts of vitamin E during pregnancy and lacta- tion in the rat. They found that the placental transfer of vitamin E is inefficient; thus, the dietary exposure of the dams to vitamin E had minimal effects on the progeny before birth. The dams receiving the high level of vitamin E had enlarged livers and elevated plasma lipids. The results of clinical blood tests revealed no disturbances in the liver, kidney, muscle, thyroid gland, erythrocytes, leukocytes, coagulation pa- rameters, and blood glucose. Farrell and Bieri con- cluded that vitamin E in this range of intake produced no apparent toxic side effects. These effects were generally minor: Their results showed that vitamin E treatment did not significantly affect sub- jective evaluations of work performance, sexuality, gen- eral well-being, muscular weakness, or gastrointestinal disturbances. It also did not affect prothrombin times, total blood leukocyte counts, or serum creatine phos- phokinase activities. Vitamin E treatment did produce significant elevations in serum triglycerides in females. It significantly decreased serum concentrations of thy- roxine and triiodothyronine in females who were not using oral steroid contraceptive agents and in males. He showed that high levels of vitamin E do not affect coagulation mechanisms un- less animals are made mildly vitamin K deficient by the use of warfarin. In this case, high levels of vitamin E produce a profound coagulopathy. A double-blind study by Zipursky et al. Therefore, estimates of maximum tolerable levels in animals should be consid- ered tentative. For these species, the presumed upper safe levels of vitamin E are higher than the dietary levels by rather undefined increments. The studies by Yang and Desai a,b and Alam and Alam indicate that this level is not hazardous. In the absence of experimental data on hypervitaminosis E for other species, maximum tolerable levels of the vitamin can be inferred only by extrapolation from these esti- mates for rats and chicks. Vitamin E is a required nutrient for cell antioxidant protection by all animals. Hypervitaminosis E has been studied in rats, chicks, and humans. Effects of excess vitamin E on rat teeth. Alpha-tocopherol pretreatment increases adriamycin bone toxicity. Effect of different levels of dietary alpha tocopherol and linoleate on plasma and liver lipids in rats. Nutritional influence on cellular antioxidant de- fense systems. Assessment of vitamin E status in animals and man. Coagulation problems relating to vitamin E. Coagulapathy associated with vitamin E ingestion. On the existence of a hitherto unrecognized dietary factor essential for reproduction. The isolation from wheat-germ oil of an alcohol, cY-tocopherol, having the proper- ties of vitamin E. Megavitamin E supplementation in man. Evaluations of the health aspects of tocopherols and alpha-tocopheryl acetate as food ingredients. A Comprehensive Treatise, L. Blood transport and metabolism. Nutritional muscular dystrophy in the guinea pig and rabbit. Tocopherol excess in man: Creatinuria with prolonged ingestion. Influence of excess vitamin E on vitamin A toxicity in rats. The effect of alpha-to- copherol on the utilization of carotene by the rat. Syn- thesis of alpha-tocopherol vitamin E. Nutritional, Biochemical, and Clinical Aspects, L. Hypervita- minosis E in the chick. Effect of large amounts of vitamin E during pregnancy and lactation. Effects of deficiency in animals. Chemistry, Physiology, Pathology, Methods, Vol. An apparent rachitogenic effect of excessive vitamin E intakes in the chick. Effect of excessive dietary vitamin E in the chick. Cerebellar disorder in chicks, apparently of nutritional origin. Vitamin E and thrombophlebitis. Perspective on vitamin E therapy. Biochemical role as a component of glutathione peroxidase. Safety and tolerance of high-dose vitamin E administration in man: A review of the literature. Vitamin A absorption and metabolism in the chick: Effect of certain tocopherols and other antioxidants on the utilization of beta-carotene for vitamin A storage. Vitamin E as the biological lipid antioxidant. Study on the effect of megavitamin E supplementation in man. D,L-alpha- tocopheryl acetate vitamin E: A long-term toxicity and carcinoge- nicity study in rats.

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Chapter 2 : - NLM Catalog Result

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Data having different superscript letter within the same row were statistically different from each other. Discussion The present study investigated the protective effects of resveratrol on hematological and biochemical changes in rats induced by sodium fluoride. Similar to our studies, others have shown decreases in WBC and PLT counts [35], neutrophil ratio [20], and RBC count [13 , 33 , 36] in animals treated with fluoride. In the present study, it was observed that fluoride-induced hematological changes were ameliorated in the group that received fluoride and resveratrol. It has been reported that fluoride toxicity causes hematopoietic progenitor cells injury in humans [6] and mice [38]. Decreased WBC, PLT, and neutrophil counts observed in the present study were probably due to harmful effects of fluoride on bone marrow and hematopoietic organs. In the present study, in which no anemia was observed, decreased RBC count may have been associated with a decreased rate of erythrogenesis due to the negative effect of fluoride on erythropoiesis or to shortened life span of erythrocytes and membrane degeneration by means of fluoride causing erythrocytes to change into echinocytes [39]. Unlike Juan et al. Considering the anti-inflammatory characteristic of resveratrol [41], the observed decrease in WBC was expected. One of the most important findings of this study was that some fluoride-induced hematological changes were improved in the group that received resveratrol with fluoride. These results suggest a beneficial effect of resveratrol treatment against fluoride-induced changes in bone marrow and hematopoietic progenitor cells. Some biochemical parameters, such as ALT activity, which is correlated with hepatic necrosis in rats [43] and indicates alteration in hepatic functions, were higher in the group that received fluoride as compared to the control group, despite the absence of differences in plasma ALP activity, urea, albumin, calcium, and inorganic phosphorus levels. The present study observed statistically insignificant increases in AST activity in the fluoride-treated group. In contrast to the present study, Xiong et al. Although there was no significant alteration in ALT or AST enzyme activity in the group that received resveratrol with fluoride, as compared to the control group and fluoride group, it was observed that both enzyme activities in this group showed a downward tendency. The liver, which has an active metabolism, is extremely sensitive to fluoride toxicity [46]. The insignificant decrease in AST and ALT enzyme activity in the group that received resveratrol with fluoride suggested that resveratrol had a protective effect on the liver. An insignificant decrease in calcium level was recorded in the fluoride-treated group in this study. Phosphorus enters the cell as inorganic phosphorus via a secondary active transport mechanism [47]. Furthermore, Anderson et al. In this study, the reduced inorganic phosphorus level was ameliorated by resveratrol. Conclusions In conclusion, two remarkable results have emerged from this study. First, fluoride exposure caused changes in hematological and biochemical parameters, which were not in perfect agreement with other studies. These discrepancies are thought to be related to the dose and duration of fluoride exposure, as well as to differences in animal species and individual differences. Second, resveratrol reduced some harmful effects induced by fluoride treatment. Conflict of Interests The authors declare that there is no conflict of interests regarding the publication of this paper. The fluoride problem in the groundwater of Sri Lanka-environmental management and health. International Journal of Environmental Studies. Fluorosis management programme in India. Genetic toxicity of fluoride. Environmental and molecular mutagenesis. Neurotoxicity of sodium fluoride in rats. Distribution and excretion of radiofluoride in the human. Proceedings of the Society for Experimental Biology and Medicine. The influence of sodium fluoride on the clonogeneclty of human hematopoietic progenitor cells: Environmental epidemiology of fluorine and its effects on health. Soil and Environmental Science. Evaluation of hematological changes in population exposed to fluoride. Toxicological and Environmental Chemistry. Effects of sodium fluoride on locomotor behavior and a few biochemical parameters in rats. Environmental Toxicology and Pharmacology.

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Effects of sodium fluoride exposure on some biochemical parameters in mice: Food and Chemical Toxicology. Toxicity of fluoride to diabetic rats. Role of vitamin E in combination with methionine and L-carnosine against sodium fluoride-induced hematological, biochemical, DNA damage, histological and immunohistochemical changes in pancreas of albino rats. Effect of subacute dosage of fluoride on male mice. Karadeniz A, Altintas L. Effects of Panax ginseng on fluoride-induced haematological pattern changes in mice. Cumulative effect of fluoride on hematological indices of mice, Mus Norvegicus albinus. Haematological and biochemical studies on fluoride toxicity in sheep. Erciyes University Journal of Health Science. Journal of Advance Researches in Biological Science. Beneficial effect of tamarind ingestion on fluoride toxicity in dogs. Effect of industrial fluorosis on haemogram of camels. Effect of endemic fluorosis on hematological parameters. Biological Trace Element Research. Mitigation by black tea extract of sodium fluoride induced histopathological changes in brain of mice. An LC-MS method for analyzing total resveratrol in grape juice, cranberry juice, and in wine. Journal of Agricultural and Food Chemistry. Neuroprotective effects of resveratrol against traumatic brain injury in immature rats. Neuroprotection by resveratrol against traumatic brain injury in rats. Molecular and Cellular Biochemistry. Resveratrol protects against methotrexate-induced hepatic injury in rats. Journal of Pharmacy and Pharmaceutical Sciences. Resveratrol ameliorates methotrexate-induced hepatotoxicity in rats via inhibition of lipid peroxidation. Human and Experimental Toxicology. Resveratrol attenuates cisplatin-induced nephrotoxicity in rats. Gentamicin-induced nephrotoxicity in rats ameliorated and healing effects of resveratrol. Biological and Pharmaceutical Bulletin. Resveratrol treatment protects against doxorubicin-induced cardiotoxicity by alleviating oxidative damage. Evaluation of propolis effects on some biochemical parameters in rats treated with sodium fluoride. Pesticide Biochemistry and Physiology. Fluorosis and its hematological effects. Toxicology and Industrial Health. Effect of resveratrol on antioxidant enzyme activities in the brain of healthy rat. Vitamin E supplementation protects oxidative stress during arsenic and fluoride antagonism in male mice. Drug and Chemical Toxicology. Arpita S, Bidyut B. Effect of fluoride toxicity on some clinical, biochemical and physiological aspects of albino rats. International Journal of Research in Chemistry and Environment. Toxic effects of deltamethrin and fluoride on hematological parameters in rats. In vivo effects of sodium fluoride on bone marrow transplantation in lethally irradiated mice. The daily oral administration of high doses of trans-resveratrol to rats for 28 days is not harmful. Anti-inflammatory effects of resveratrol in lung epithelial cells: The American Journal of Physiology: Lung Cellular and Molecular Physiology. The protective effect of resveratrol in experimentally induced non-sterile clean wound inflammation in rats. Kafkas Universitesi Veteriner Fakultesi Dergisi. Veterinary Hematology and Clinical Chemistry. Effects of sodium fluoride on hepatic toxicity in adult mice and their suckling pups. Dose-effect relationship between drinking water fluoride levels and damage to liver and kidney functions in children. Shashi A, Thapar SP. Histopathology of fluoride-induced hepatotoxicity in rabbits. Molecular mechanisms of fluoride toxicity. Parallels with its inhibition of the sarcoplasmic reticulum CaATPase. Journal of Biological Chemistry. Effects of cortisol and fluoride on ion-transporting ATPase activities in cultured osteoblastlike cells.

Chapter 3 : Biochemical aspects of neurological disease | Clinical Gate

MEMBRANE EFFECTS OF VITAMIN E DEFICIENCY: BIOENERGETIC AND SURFACE CHARGE DENSITY STUDIES OF SKELETAL MUSCLE AND LIVER MITOCHONDRIA (pages). Alexandre T. Quintanilha, Lester Packer, Joanna M. Szyszlo Davies, Toinette L. Racanelli and Kelvin J. A. Davies.

Diagnosing vitamin A deficiency and toxicity Evaluating persons with intestinal malabsorption of lipids Evaluating individuals with motor and sensory neuropathies for vitamin E deficiency Monitoring vitamin E status of premature infants requiring oxygenation Clinical Information Discusses physiology, pathophysiology, and general clinical aspects, as they relate to a laboratory test Vitamin A: The level of vitamin A in the plasma or serum is a reflection of the quantities of vitamin A and carotene ingested and absorbed by the intestine carotene is converted to vitamin A by intestine absorptive cells and hepatocytes. Vitamin A plays an essential role in the function of the retina adaptation to dim light , is necessary for growth and differentiation of epithelial tissue, and is required for growth of bone, reproduction, and embryonic development. Together with certain carotenoids, vitamin A enhances immune function, reducing the consequences of some infectious diseases. Degenerative changes in eyes and skin are commonly observed in vitamin A deficiency. Poor adaptation of vision to darkness night blindness is an early symptom that may be followed by degenerative changes in the retina. In developing countries, vitamin A deficiency is the principal preventable cause of blindness. Severe or prolonged deficiency leads to dry eye xerophthalmia that can result in corneal ulcers, scarring, and blindness. Another important consequence of inadequate intake is acquired immunodeficiency disease, with an increased incidence of death related to infectious diseases. In patients with HIV, vitamin A deficiency is associated with increased disease progression and mortality. Vitamin A in excess can be toxic. In particular, chronic vitamin A intoxication is a concern in normal adults who ingest more than 15 mg per day, and in children who ingest more than 6 mg per day of vitamin A over a period of several months. Manifestations are various and include dry skin, cheilosis, glossitis, vomiting, alopecia, bone demineralization and pain, hypercalcemia, lymph node enlargement, hyperlipidemia, amenorrhea, and features of pseudotumor cerebri with increased intracranial pressure and papilledema. Liver fibrosis with portal hypertension and bone demineralization may also result. Congenital malformations, like spontaneous abortions, craniofacial abnormalities, and valvular heart disease have been described in pregnant women taking vitamin A in excess. Consequently, in pregnancy, the daily dose of vitamin A should not exceed 3 mg. Vitamin E contributes to the normal maintenance of biomembranes, the vascular system, and the nervous systems, and provides antioxidant protection for vitamin A. The current understanding of the specific actions of vitamin E is very incomplete. The tocopherols vitamin E and related fat-soluble compounds function as antioxidants and free-radical scavengers, protecting the integrity of unsaturated lipids in the biomembranes of all cells and preserving retinol from oxidative destruction. Vitamin E is known to promote the formation of prostacyclin in endothelial cells and to inhibit the formation of thromboxanes in thrombocytes, thereby minimizing the aggregation of thrombocytes at the surface of the endothelium. Those influences on thrombocyte aggregation may be of significance in relation to risks for coronary atherosclerosis and thrombosis. Deficiency of vitamin E in children leads to reversible motor and sensory neuropathies; this problem also has been suspected in adults. Premature infants who require an oxygen-enriched atmosphere are at increased risk for bronchopulmonary dysplasia and retrolental fibroplasia. Supplementation with vitamin E has been shown to lessen the severity of, and may even prevent, those problems. In addition, low blood levels of vitamin E may be associated with abetalipoproteinemia, presumably as a result of a lack of the ability to form very low-density lipoproteins and chylomicrons in the intestinal absorptive cells of affected persons. Vitamin E toxicity has not been established clearly. Chronically excessive ingestion has been suspected as a cause of thrombophlebitis, although this has not been definitively verified. Deficiencies of vitamins A and E may arise from poor nutrition or from intestinal malabsorption. Persons at risk, especially children, include

those with bowel disease, pancreatic disease, chronic cholestasis, celiac disease, cystic fibrosis, and intestinal lymphangiectasia. Infantile cholangiopathies that may lead to malabsorption of vitamins A and E include intrahepatic dysplasia and rubella-related embryopathy Reference Values.

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Chapter 5 : VAE - Clinical: Vitamin A and Vitamin E, Serum

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Chapter 7 : 3 Vitamin E | Vitamin Tolerance of Animals | The National Academies Press

Vitamin E is a generic term denoting eight different isomers among which alpha -tocopherol is the most important and most active. Vitamin E metabolism is closely linked to lipids during intestinal.

Chapter 8 : Bertram Lubin: used books, rare books and new books @ blog.quintoapp.com

In addition, low blood levels of vitamin E may be associated with abetalipoproteinemia, presumably as a result of a lack of the ability to form very low-density lipoproteins and chylomicrons in the intestinal absorptive cells of affected persons.