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PARTIAL PURIFICATION OF RABBIT SERUM ARYL ESTERASE (PARAOXONASE). J. K. Zimmerman and T. M. Brown, Dept. Biological Sci. and Dept. Entomology, Clemson University, Clemson, SC 29631.

Rabbit serum paraoxonase has been purified to the same specific activity as the most highly purified material reported (A.R. Main (1960) Biochem. J. 74: 10-20), but still is not homogeneous. The purification consists of a fractionation by PEG-4000 (14-25%), two DEAE Sepharose columns, and an Ultrogel AcA34 column. All purification steps were done at 4°, pH 7.0, and in MOPS buffer containing 2.5 mM Ca^{++} and 0.02% NaN_3. The calcium ion is necessary at all steps to maintain high recoveries while the azide prevents the rapid loss of activity reported by other investigators.

The fraction with the paraoxonase activity eluted from the Ultrogel AcA34 column with a partition coefficient of 0.35. SDS-PAGE patterns of this material showed a doublet of M_r's 40-45,000 and 47-54,000 which varied in intensity as the paraoxonase activity varied. SDS-PAGE bands from material eluting before and after the paraoxonase activity showed apparent molecular weights of 170-190,000 and 150-160,000 respectively. Analytical ultracentrifugation of the paraoxonase peak showed a sedimentation equilibrium weight average molecular weight of 190,000 daltons.

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DO DIETARY FATS CONTAIN PUFA ANTAGONIST(S)? Bela Szepesi and A. Kalla Kamara. Beltsville Human Nutrition Research Center, ARS, USDA, Beltsville, Md. 20705, and Howard University, Washington, D.C. 20559.

Rat liver glucose-6-phosphate dehydrogenase (G6PDH) activity is increased by starvation-refeeding to a level which "overshoots" ad libitum-fed values ("overshoot" - (OS)). A second starvation-refeeding produces an even higher G6PDH level ("induction increment" - (II)). Interposing a high-fat diet (containing a 1:1:1 mixture of beef tallow, lard, and corn oil): a) between starvation and refeeding the inducer diet abolishes the (OS); b) between the first and second starvation abolishes the (II). Ten years ago (Nutr. Rep. Int. 9: 15, 1974) raising the fat content (1:1:1 mixture) to 20% was sufficient to abolish both the (OS) and the (II). After an almost gradual change, in recent work, the (OS) can still be abolished, but not even a 40% fat diet can eliminate the (II). Ethyl esters of linoleic or linolenic acid (in the 1:1:1 mix) still can abolish both the (OS) and the (II), excluding a possible genetic drift toward selection in the Wistar rats for the absence of the control in question. We prepared lard and beef tallow in our own laboratory but diets prepared with these fats still could not abolish the (II). We cannot definitely rule out shipment or housing conditions of the rats, or the use of the AIN-76 salt mix as responsible factors, nor have we confirmed these. Another possibility being studied is that corn oil may contain PUFA antagonist(s).

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INFLUENCE OF ZINC ON DIARRHEA CAUSED BY A HEATED CASEIN-STARCH DIET IN THE RAT. Randall L. Hess and Dennis T. Gordon. Department of Food Science & Nutrition, University of Missouri, Columbia, MO 65211.

Maillard reaction products (MRP) have been reported to have anti-nutritional properties. We have observed that rats fed heated casein diets containing MRP developed a diarrhea that appeared to contain excess mucus and to depend on dietary Zn. To clarify these observations, rats were fed nutritionally complete diets containing unheated or heated casein-cornstarch-corn oil mixtures with Zn levels of 3 or 30 ppm. The diarrhea occurred within 3 days on heated diets but the onset was not affected by Zn status. Visual examination of colon contents showed poor pellet formation and adherence of fecal material to the colon wall. Measurements of mucus gel layer thickness of the colon showed no differences in treatments. Histological examination of cecum and colon showed no changes. Carbohydrate and protein analyses were used to estimate soluble glycoprotein content of colon tissue (CT) and colon fecal material (CFM). No differences were found in carbohydrate or protein content of CT. A 4-fold increase in the protein content of the CFM from animals fed heated diets was observed but there was no difference in carbohydrate content. In conclusion, Zn nutrition does not appear to be involved in diarrhea produced by MRP. Results suggest that the diarrhea was not caused by increased mucus secretion but incomplete protein digestion leading to osmolarity changes in the colon.

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INCREASED CELL NUMBER IN ESOPHAGUS OF ZN-DEFICIENT RAT. J. Apgar, U. S. Plant, Soil, & Nutrition Laboratory, USDA-ARS, Ithaca, NY 14853.

Skin lesions are common to Zn deficiency in many species. Sensitivity of epidermal cells to low Zn intake is particularly marked in rat esophagus; parakeratosis appeared within a week in both young and adult animals (Apgar, Fed. Proc. 41:283, 1982). Mucosal weight also increased in -Zn rats; submucosal weight was not affected. To determine whether increased mucosal weight was due to failure of cells to slough or to increased growth of basal (dividing) cells, cells from the mucosal layer of -Zn rats and of pair weight controls were separated into basal and differentiated fractions by centrifugation on a Percoll gradient. Cell number was determined by count or DNA analysis. In 4 experiments an average of 2.2 times as many cells of each type were recovered from -Zn esophagi. Within the differentiated cell fraction the percentage of cornified envelopes, characteristic of fully differentiated epidermal cells, was similar in -Zn and +Zn rats. Differentiation therefore appeared to be less affected than growth. Increased basal cells confirm earlier indications of growth stimulation in these cells when growth of other cell types is retarded. Identification of factor(s) responsible for stimulation of growth in -Zn rats or inhibition of growth in +Zn rats would not only be of considerable interest but might be useful in assessing Zn status.

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I-1407

Preservation of acute phase response in murine zinc deficiency. N. W. Solomons, E. Udomkesmalee* and K. McAdam*. Massachusetts Inst. of Technology, Cambridge, MA 02139 and Tufts Univ. Sch. of Med., Boston, MA 02111.

Uptake of zinc(Zn) by the liver after an acute inflammatory stimulus appears to correlate with increased production of the acute phase proteins, one of which is serum amyloid A (SAA). The mediator for SAA synthesis has been identified within leukocyte endogenous mediator (LEM). Experiments were conducted to determine whether adequate Zn nutriture is required at the macrophage production of LEM or at the hepatic synthesis of SAA or both. C3H mice were fed Zn-deficient(ZD) diet (<1ppm) for 8 wks. They were triplet matched to the ad libitum (AL) and pair-fed (PF) controls on an adequate Zn diet (60ppm). Serum Zn concentrations for AL, PF and ZD mice were 153, 138 & 55 ug/dl respectively. Following an i.p. injection of E.coli endotoxin, there were no significant differences of SAA production(ugAA/ml) among the AL(705±61), PF(879±71) and ZD(787±70) mice. Furthermore, ZD mice showed a similar SAA response to standard LEM as seen in AL and PF mice both in vivo and in the hepatocyte culture system (in vitro). LEM produced by endotoxin-activated macrophages of ZD mice was as active as those of AL and PF mice in stimulating SAA production in the normal endotoxin nonresponder mice in vivo and in vitro. These data suggest that the acute phase response represented by the synthesis of SAA is preserved in murine Zn deficiency.

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EFFECTS OF MARGINAL DIETARY ZINC DEPRIVATION ON THE DEVELOPMENT OF RHESUS MONKEY INFANTS. Mari S. Golub, M. Eric Gershwin, Lucille S. Hurley, Anthony Cheung and Andrew G. Hendrickx. Departments of Internal Medicine and Nutrition and CPRC, University of California, Davis, CA 95616.

Our laboratory has been studying marginal deprivation of zinc during gestation in pregnant rhesus monkeys. We have extended these observations to the growth, behavior, nutritional status and immune function during the first year of life in rhesus monkey infants marginally deprived of zinc (4 µg/g zinc diet) from conception. The purified diet contained biotin-enriched sprayed egg white as the protein source. Plasma zinc concentrations were 10-25% lower in deprived infants than in controls fed a 100 µg/g diet. Other group differences included: intrauterine growth retardation and postnatal catch up growth; slowed growth from 9-12 months; alopecia and dermatitis; hypogeusia; delayed skeletal maturation and defective mineralization; hypochromic microcytic anemia in the neonatal period; depressed response of peripheral lymphocytes to T- and B-cell mitogens; depressed PMN chemotaxis and phagocytosis; hypoactivity, lethargy and impaired discrimination reversal performance. The appearance and severity of some of these deficiencies was dependent on sex and developmental stage. These and other data suggest that childhood growth, immune and behavioral disorders can originate in selective marginal dietary zinc deprivation. Supported by HD 14388 and RR00169.