1535 NUTRITION

EFFECT OF TRYPTOPHAN (trp) AND NICOTINAMIDE (Nam) INTAKES ON URINARY EXCRETION OF N^1-METHYLNICOTINAMIDE (NMe) IN THE RAT. J.I. Patterson* (SPON: M.K. Brush). Univ. of Wisconsin, Madison, WI 53706

Two experiments were performed using weanling rats fed graded levels of trp and Nam to determine the amount of dietary trp needed for the formation of 1 mg Nam. First, weanling rats were fed ad libitum for 10 days 12% casein diets containing 0, 10, 20, 50, 150 and 500 mg Nam/kg diet. Second, weanling rats were fed ad libitum for 12 to 16 days diets containing 15% amino acids and .04, .08, .12, .16, .2, .3, .5 and 1.0% trp with 0 or 20 mg Nam/kg diet. Food intake and weight gain were measured daily. During the last 3 days urine was collected and NMe measured. From 20 to 150 mg Nam/kg diet, NMe excretion was directly proportional to Nam intake. Therefore, at these levels of intake, 70.6% of dietary Nam was recovered as NMe. Rats fed 500 mg Nam/kg diet excreted less than the proportional amount of NMe. Above .16% trp, umoles NMe excreted/day by rats fed 0 or 20 mg Nam/kg diet increased linearly with increasing umoles trp intake (slopes=.106 and .112 respectively). Rats fed 1.0% trp diets excreted proportionately less NMe. Therefore, excluding dietary trp and Nam essential for growth, an additional 10 to 11 mg of dietary trp are needed by the growing rat for the formation of 1 mg of Nam regardless of whether Nam is in the diet. This conversion factor does not hold true at the highest levels of Nam and trp intakes since proportionately less NMe was excreted at these levels. (Supported in part by USPHS NIH grant HA 10748)

1536 NUTRITION

THE AVAILABILITY OF NIACIN IN FOODS. E.G.A. Carter* and K.J. Carpenter. Dept. of Nutritional Sciences, University of California, Berkeley, CA 94720

A bioassay using rats was developed to quantify the available niacin present in a range of foods. The same foods were analyzed chemically for 'total niacin' (i.e. after alkaline hydrolysis) and for free niacin (i.e. nicotinic acid plus nicotinamide extracted with aqueous ethanol). The 'total', 'available' and 'free' niacin values (mg/kg. air-dry food) respectively were: raw whole corn meal 19, 7, ND(none detected); baked whole corn meal 27, 8, ND; boiled whole corn, 19, 7, trace; tortilla, 13, 14, 13; raw sweet corn 51, 40, trace; steamed sweet corn 56, 46, 45; raw wheat 51, 16, ND; boiled wheat 57, 18, ND; boiled rice 71, 29, 12; boiled milo 45, 16, ND; autoclaved Phaseolus vulgaris beans 26, 31, 22; freeze-dried coffee 600, 420, 320; peanut flour 240, 100, ND; baked potato 81, 19, 23 and baked beef liver 310, 321, 280. As expected, the niacin in liver is both chemically 'free' and fully available. In the mature cereal grains, potatoes and peanuts, it is apparently less than 50% available. In the case of the grains and peanuts, the 'free' niacin values are virtually zero and so underestimate the availability. The bound niacin in sweet corn is more labile and both fully available and released by steaming. The complete availability of niacin in tortilla is confirmed. Beans seem to have their niacin fully available and this is also largely true for coffee.

1537 NUTRITION

EFFECT OF RIBOFLAVIN ON BRANCHED CHAIN AMINO ACID OXIDATION. Sang-Sun Lee*, Marjorie Caldwell*, and James Bergan, Food Science & Nutrition, Univ. of Rhode Island, Kingston, RI 02881

Catabolism of the branched chain amino acids (BCAA) involves transamination followed by dehydrogenation and decarboxylation. As the dehydrogenase is an FAD requiring enzyme, the effect of riboflavin (B_2) deficiency on BCAA oxidation was studied.

Growing rats received equal amounts of protein and other nutrients. Calories were fed ad libitum to a B_2 deficient group (5 ug B_2/day) and an ad libitum control (30 ug B_2/day). A third group was given adequate B_2 (30 ug) but calorie intake was restricted to that of the deficient animals.

Caloric intake and weight gain was severely depressed in the B_2 deficient animals and the pair-fed controls. Concentrations of B_2 in liver, muscle and erythrocytes were decreased in deficient animals compared to controls while the FAD induced Glutathionine Reductase activity (AC) was increased. Fasting increased the concentration of B_2 in liver in all groups. Erythrocyte and muscle values were not different but AC values decreased with fasting. Leucine oxidation was determined from the rate of $^{14}CO_2$ production following incubation of isolated diaphragms with L-leucine-U^{14}C. In the fed state, deficient animals produced less CO_2 than controls. Fasting increased CO_2 production in the deficient animals and in the ad libitum controls. Addition of FAD to the incubation media increased CO_2 production in all groups with the greatest effect in the deficient animals. Incorporation of ^{14}C-leucine into protein was not affected by B_2 intake but was decreased by fasting. (Supported in part by R.I. Agricultural Experiment Station.)

1538 NUTRITION

ASCORBIC ACID METABOLISM AND BODY POOL SIZE IN THE MONKEY. J.A. Tillotson*, R.J. O'Connor*, and E.L. McGown* (SPON: J.H. Skala). Letterman Army Institute of Research, Presidio of San Francisco, CA 94129

Monkeys (Macacca fasicularis) were maintained on constant intakes (low, medium, high) of ascorbic acid (AA) and fed ^{14}C-l-AA to study urinary AA metabolites and body pool sizes. To reduce stress, the monkeys had been familiarized to personnel and experimental routine, including restraint in primate chairs. Degradation of AA and its metabolites was minimized by freezing the urine as it was voided and by separating the ^{14}C metabolites within 24 hr by cation exchange chromatography. Four peaks of radioactivity were consistently obtained: peak 1 contained AA and weakly acidic compounds; peak 2, a minor unidentified compound; peak 3, a stable, but unidentified metabolite; and peak 4 was primarily oxalate. The percentage distribution of ^{14}C in each peak was dependent upon the level of AA intake and it remained constant through a minimum of 25 days after the last labelled dose or until the AA supplement was changed. The ^{14}C in peak 1 varied from 20-30% in monkeys fed 0.5 mg AA/kg body weight/day to 70-80% in monkeys fed >20 mg AA/kg body weight/day. The oxalate fraction contained from 50% to 6% of the total urinary ^{14}C in the monkeys fed the low to high levels of supplements. Body pool sizes were calculated from the logarithmic decay of specific activity of urinary AA as measured in peak 1 over a period of 10 days. Estimates of body pools ranged from 9.5 to 76 mg AA/kg in animals maintained on 0.5 mg to >20 mg/kg body weight/day.

1539 NUTRITION

THE KINETICS OF VITAMIN C IN HUMAN BLOOD PLASMA FROM VITAMIN C SUPPLEMENTS. B. P. Poovaiah*, J. A. Rider*, J. Scala, Ralph K. Davis Medical Center, S. F., CA, 94114

A human crossover study was used to evaluate the appearance of Vitamin C in plasma and urine from 1000 mg dosage. The dose was provided in five 200 mg doses at one hour intervals; a single dose from a tablet and a single dose time preparation designed to release the ascorbic acid over a five hour period. The results indicate that blood levels are maintained most effectively by small doses at regular intervals or by sustained release preparations. Urinary excretion of Vitamin C begins within one hour after ingestion. It is possible to conclude from these experiments that optimum bioavailability can be achieved by several means, including both sustained release and the use of small rapidly absorbed doses.

1540 PHYSIOLOGY

INTERACTIONS BETWEEN FOLIC ACID AND ASCORBIC ACID IN THE GUINEA PIG. C.M. Lewis*, E.L. McGown*, M.G. Rusnak*, and H.E. Sauberlich. Letterman Army Institute of Research, Presidio of San Francisco, CA 94129

Scorbutic patients have been reported to have a megaloblastic anemia which may or may not respond to folic acid (FA) treatment. To investigate possible interactions between ascorbic acid (AA) and FA metabolism, guinea pigs were fed a semipurified diet containing adequate or low FA. After 3 weeks each group was subdivided into 2 groups which were placed on diets adequate or deficient in AA. When signs of deficiency appeared in the deficient groups, the animals were exsanguinated and tissue samples (liver, kidney, adrenal, spleen, intestinal mucosa) were removed for FA and AA analysis. Adrenal folate levels were unaffected by folic acid deficiency. In contrast, FA deficiency caused significantly lower FA content in all other tissues. AA deficiency caused significantly lower AA content in all tissues of AA deficient animals, but had no generalized effect on tissue folate levels at either adequate or low FA intake. AA deficiency did exacerbate the lowered white blood cell count resulting from FA deficiency, but AA deficiency alone did not affect hematologic parameters. FA deficiency significantly lowered adrenal AA content in animals receiving either level of AA. These data suggest that hematologic effects of AA deficiency are not due to a generalized effect of AA on FA levels in tissues. They also suggest there may be a specific AA/FA interaction in the adrenal gland.

MARCH 1, 1980 • VOLUME 39, NUMBER 3

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Federation Proceedings

Abstracts

64th Annual Meeting
Anaheim, California
April 13–18, 1980

FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY

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