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POSTPRANDIAL GLYCEMIA ON TWO SWEET CONFECTIONS WITH DIFFERENT CARBOHYDRATE PATTERNS. Carol S. Chuck*, James Scala, Gene A. Spiller and James H. Whittam*, Shaklee Research Center, Hayward, CA 94545

Postprandial glycemia differs after intake of different carbohydrates (CHO). Two confections were compared to a glucose solution for glycemic effect, all containing 60 g CHO. One treatment was a fruit-based bar (FB) containing raisins, 24% glucose (GLU), 20% fructose (FRU) and 2% sucrose (SUC); the other treatment was a chocolate-based bar (CB) with 49% SUC, 9% GLU and 2% FRU. 14 healthy human volunteers, age 25-51 (median age 30.5) years had blood drawn after overnight fasting at fasting and at 15, 30, 60, 90, 120 and 180 minutes (m) after consuming treatments with 600 ml water. All consumed GLU first, were then randomized 7 to FB and 7 to CB and the experiment repeated after a four week rest. The hyperglycemia caused by FB was lower than from GLU or CB after 15 m ($p<.05$) and lower than CB at 30 m ($p<.05$). FB hypoglycemia did not significantly differ from GLU except at 180 m, where FB caused significantly less ($p<.05$) hypoglycemia than GLU. CB hyperglycemia did not significantly differ from GLU. At 90 m CB caused greater hypoglycemia than GLU ($p<.05$) but similarly to FB, CB caused less hypoglycemia ($p<.05$) than GLU at 180 m. Products based on dry fruits and fructose appear to cause less hyperglycemia and hypoglycemia than chocolate-sucrose based products, except for a similar response at 180 m.

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BLOOD INSULIN, GLUCOSE, AND GASTRIC INHIBITORY POLYPEPTIDE LEVELS IN CARBOHYDRATE-SENSITIVE AND NORMAL MEN GIVEN A SUCROSE OR INVERT SUGAR TOLERANCE TEST. K. C. Ellwood*, O. E. Michaelis IV, J. Hallfrisch* and T. M. O'Dorisio*, Beltsville Human Nutrition Research Center, USDA, Beltsv[illegible] Md. 20705, and Ohio State University, Columbus, Ohio 432[illegible]

Twelve normal and 12 carbohydrate-sensitive men, 28-5[illegible] years old, were selected. Carbohydrate-sensitivity was b[illegible] on an abnormal insulin response to a sucrose load. The subjects were fed a diet consisting of 45% total carbohydrate, 40% fat, and 15% protein for 5 days prior to the tolerance test. In a crossover design, subjects were given 2 g/kg body weight of sucrose or invert sugar and responses to insulin, glucose, and gastric inhibitory polypeptide (GIP) were determined. Blood samples were taken at 0, 0.5, 1, 2, and 3 hours after being given the test loads. GIP and glucose were not significantly different between diets or between the carbohydrate-sensitive and normal men. At 1 hour, the insulin levels for the carbohydrate-sensitive men given sucrose were significantly higher than for the carbohydrate-sensitive men given invert sugar (disaccharide effect). Except for fasting values, the subjects that were carbohydrate-sensitive and given either sugar had greater insulin levels than the normal subjects. The study indicates that GIP was not different in unadapted human subjects following an invert sugar or sucrose load.

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IN VIVO INTRAGASTRIC HYDROLYSIS OF MILK BY A β-GALACTOSIDASE: A POTENTIAL APPROACH TO SYMPTOMATIC MILK INTOLERANCE IN PRIMARY LACTASE DEFICIENCY. Noel W. Solomons*, A-M Guerrero and Benjamin Torun. Institute of Nutrition of Central America and Panama, Guatemala City, and Massachusetts Institute of Technology, Cambridge, MA, 02139.

We studied indirectly the degree of effective in vivo hydrolysis when a commercial β-galactosidase (LactAid^R) was added at the time of drinking milk, using the excretion of breath H_2 as an index of the quantity of lactose escaping intestinal absorption. Subjects were screened with a 360 ml (12 oz) dose of intact whole cow's milk (ICM) (approx. 18 g of lactose). Those with a maximal increment in breath [H_2] >25 ppm were given the same dose of milk treated with either: 5 gtts of enzyme, 24 h earlier (to achieve >90% hydrolysis) (LHM); or 10 gtts, within 5 min of drinking (5MM). Of 11 subjects receiving all three treatments, 8 had a reduced H_2 production (7 - 86 %) with the 5MM as compared to the ICM. Older subjects showed a greater effect of 5MM than did younger individuals. The mean excess H_2 production in ppm·hr was 121 (ICM) 83 (LHM) and 9.5 (5MM), respectively. Adding 10 ml of antacid ($CaCO_3$) to 5MM in 6 subjects produced an additional decrement in H_2 in 5 as compared to 5MM alone. A food-grade β-galactosidase produced in vivo hydrolysis. Inferences from the age effect, and manipulation of pH suggests the stomach to be the site. Further tailoring of the enzymatic pH optimum might provide an even more effective modality to decrease milk intolerance in symptomatic lactase-deficient individuals.

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LACTOSE TOLERANCE IN ASCARIS INFECTED PRE-SCHOOL CHILDREN. E.Carrera,* D.W.T. Crompton** and M.C. Nesheim*. *Cornell University, Ithaca, N.Y., **University of Cambridge, England.

Recent studies by Forsum et al. (Parasitology, 83: Part II 1981) found that intestinal lactase activity and lactose tolerance were depressed in pigs infected with Ascaris suum. To determine the significance of this observation in children, lactose tolerance tests were carried out in Panama, on twenty children, 3-6 years of age infected with Ascaris lumbricoides and twenty-one controls of comparable age, sex and nutritional status. Mothers reported that 67% of children later found to be infected had a history of milk intolerance, while only 5% of control children had a similar history. Following a lactose load, 65% of infected children presented with abdominal discomfort and/or diarrhea, whereas only 24% of controls had similar symptoms. The mean rise in blood glucose from infected children 40 minutes after a lactose load was about half of that of the controls, although the differences were not statistically significant. After deworming, lactose tolerance improved in previously infected children ($p < 0.08$) to a level similar to the one initially obtained from the controls. There were no abdominal symptoms reported during this second lactose tolerance test. These results indicate that infection with Ascaris lumbricoides appears to impair a pre-school child's ability to digest lactose.

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EFFECT OF DIETARY FIBER AND MEAL PATTERN ON MODULATION OF HMG-CoA REDUCTASE (HMGR) ACTIVITY. Michael J. Kelley*, James N. Thomas*, Victoria J.K. Liu* and Jon A. Story, Dept. Foods and Nutrition, Purdue Univ., W. Lafayette, IN 47907.

Catalytic activity of HMGR can be modulated in vitro by phosphorylation/dephosphorylation in response to glucagon/insulin, respectively. We assessed the ability of alteration of glucose and insulin levels in vivo by meal pattern or dietary fiber to alter the active fraction of HMGR. Meal-fed rats killed just prior to the meal, displayed comparable levels of expressed (R_A) and total (R_T) activities of hepatic microsomal HMGR when fed stock (64.2 and 329.9 pmol/min/mg; pU/mg) or semipurified (SP) diets (48.3 and 303.2 PU/mg). Animals killed at the midpoint of the dark cycle under ad lib feeding conditions exhibited different R_A and R_T activities (stock:101.8 and 1088 pU/mg; SP:55.5 and 580.8 pU/mg). In a second experiment, animals were fed a SP diet containing 15% cellulose (CEL), CEL + 2% cholesterol (CH), or 15% oat bran (OB) for 2 h. Animals were killed at 0,7.5,15,30,60 min,2 and 4 h after onset of feeding. Plasma glucose levels increased but returned to fasting levels by 15 min while insulin remained elevated through 4 h. R_A increased slightly from 0 to 4 h as did R_T in CEL-fed animals. In OB-fed rats, R_A rose slightly byt R_T steadily increased to a level 4 times the original value. CEL + CH-fed animals had depressed R_A and R_T at all time points. Thus diet pattern and dietary fiber can alter R_A and R_T but the ratio, R_A/R_T, may remain unchanged. (Supported in part by the Showalter Trust.)

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MODIFICATION OF CHOLESTEROL REGRESSION IN RATS BY PECTIN AND LIGNIN. James N. Thomas*, Michael J. Kelley*, Marilyn S. Petro* and Jon A. Story, Dept. Foods and Nutrition, Purdue Univ., W. Lafayette, IN 47907

Male, Wistar rats were fed a semipurified diet containing 1% cholesterol(CH) for 4 wk which resulted in an accumulation of liver CH(31.5mg/g vs. 3.3mg/g for CH-free). Animals were then divided into 3 groups and fed CH-free diets with 5% cellulose, lignin(L), or pectin(P) and groups of animals killed after 1,3,7,14 and 21 d. L- and P-fed animals had the lowest liver CH levels at 21d, the changes being mainly in the esterified fraction. Daily fecal bile acid excretion was greatest in the L-fed group during the first week but P-fed animals increased their excretion and maintained a high level of excretion through 21d. Concentration of fecal bile acids was slightly reduced in the P-fed animals. Incorporation of newly synthesized and previously synthesized CH into bile acids (measured by incorporation of [5-^{3}H] mevalonate and [4-^{14}C]CH) indicated little difference in ^{14}C but higher ^{3}H specific activities from P-fed animals. These data indicate that esterified CH is a major substrate for excretion of CH as bile acids. The predominance of chenodeoxycholic acid and its metabolites in the feces during these periods of increased excretion suggests a possible preference for synthesis of these bile acids when esterified CH is used as a substrate. (Supported in part by the Indiana Heart Association and the Showalter Trust).

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