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A.L. KINMONTH\*, R. ANGUS\*, AND J.D. BAUM. University Department of Paediatrics, John Radcliffe Hospital Oxford, England. Blood glucose control of diabetic

children eating unrefined (U) or refined (R), high carbohydrate diets.

Increasing the proportion of carbohydrate in diabetic diets may improve diabetic control in adults, but it is unclear whether this is due to the amount or type of carbohydrate eaten. A randomised crossover outpatient study was performed on 6 diabetic children to compare the effect of eating unrefined or refined high carbohydrate diets (50-55% C) from acceptable food sources, on blood glucose control. The unrefined diet used wholefoods such as beans and wholemeal bread (mean fibre:energy ratio 3:100), and the refined diet used processed foods such as white bread (mean fibre:energy ratio 1:100). The diets were individualised and were isocaloric for carbohydrate, fat, and protein. Diabetic control was assessed by daily urinalysis for glucose, home blood glucose monitoring (Ames;Eyetone) and fortnightly estimations of glycosylated haemoglobin. After 6 weeks on each diet, morning metabolic profiles, and 24-hour capillary blood and urinary glucose measurements were taken at home. The levels of blood glucose and b-hydroxybutyrate from the morning profiles, and the capillary blood and urinary glucose measurements from the 24-hour profiles were all lower on the unrefined diet. Mean blood glucose levels (mmol/L±SEM) were: fasting 6.5±1.3 (U), 8.5±2.3 (R), peak (post breakfast) 13±2.6 (U), 17.3±1.6 (R), midday 6.2±0.7 (U), 12.14 ± 1.2\* (R). Mean 24-hour urinary glucose was; 14.4±5 gm (U) 53.8±21.7 gm (R)\*. The use of whole foods high in dietary fibre can improve glycaemic control of diabetic children on liberal carbohydrate diets. \*p = 0.025.

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Increased hemoglobin A1c in mothers of overweight babies.

Hb A1c levels in diabetics are well correlated with their successful control. These levels were studied on women who had normal oral glucose tolerance tests (OGTT), but had delivered overweight (> 4500g) babies, and were compared to normal controls and to diabetic women controlled with oral antidiabetic agents or insulin. In total 117 women were studied. Women who had delivered newborns with normal weight had a mean Hb A1c of 8.01±0.19% on their 7th post partum day. Women who had delivered overweight babies had Hb A1c of 10.37±0.43% (p<0.001), although their OGTT were normal. Their insulin levels were higher than normal and there was a delay in achieving basal values. Diabetic non pregnant women successfully treated with diet only, had Hb A1c levels of 10.75±0.62%; treated with oral drugs only 10.83±0.63%; pregnant women treated with insulin 13.31±1.27%. The determinations of Hb A1c in mothers with overweight babies may be a sensitive parameter for the early discovery of carbohydrate intolerance.

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CHANGES OF TAURINE METABOLISM IN ACUTE UREMIA Michalk, D.,\* Essich, H.J.,\* and Lutz, P. University Children's Hospital, Heidelberg, FRG

Taurine (Tau) is known to be important for the regulation of ion fluxes in excitable tissues, especially in the heart muscle, where it is the most abundant amino acid. Neuromuscular disturbances are common in uremia. In order to investigate the role of Tau in uremia the concentration of Tau was determined in plasma, liver, brain, skeletal and heart muscle of 38 rats 12, 24 and 48 hours after bilateral nephrectomy (NX) and compared with sham operated (SH) and normal animals. In NX rats mean plasma creatinine rose linearly from 0.44 to 7.39 mg/dl. Tau was found to be elevated in plasma with a maximum level of 0.41 vs. 0.23 μmol/ml at 24 hrs, and in liver, in which the highest level (7.2 vs. 2.0 μmol/g wet wt) was observed 48 hrs. post NX. Mean Tau concentration was unchanged in brain (5.3 μmol/g wet wt) and skeletal muscle (15.1 μmol/g wet wt), but was lowered in the heart (20.9 vs. 26.0 μmol/g wet wt). The decrease of heart Tau content was significantly correlated to the degree of uremia (p<0.001) and was not due to changes in heart weight, which was identical in NX and SH rats. In conclusion, the elevation of plasma and liver Tau concentration in acute uremia is not followed by similar changes in muscle and brain. The apparent efflux from the heart might be involved in the development of uremic heart disease.

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K.O. RAIVIO and M.A. BECKER†, Children's Hospital, University of Helsinki, Finland, and VA Hospital, San Diego, Ca. Mechanism of purine overproduction in genetic defects of purine metabolism.

A number of defects of purine metabolism are associated with hyperuricemia and/or other indicators of increased purine synthesis, whereas in another group of mutations purine production in vivo is normal. In the former group, elevated purine synthesis has been correlated with increased intracellular phosphoribosyl-pyrophosphate (PRPP) concentrations in hypoxanthine phosphoribosyltransferase (HPRT) deficiency (Lesch-Nyhan syndrome) and PRPP synthetase overactivity, whereas in purine nucleoside phosphorylase (PNP) deficiency normal PRPP concentrations have been reported despite increased purine synthesis. We have re-evaluated PRPP metabolism in PNP-deficient fibroblasts and demonstrated that the rate of PRPP synthesis is normal but the steady-state concentration is significantly elevated. The association of increased purine synthesis with increased PRPP levels was confirmed in HPRT deficiency and PRPP synthetase overactivity, and normal values for both parameters were found in adenine phosphoribosyltransferase and adenosine deaminase deficiencies. All the mutants studied thus conform to the rule that elevated PRPP levels are correlated with increased purine synthesis, and a causal relationship is suggested. Supported by the Academy of Finland, Sigrid Jusélius Foundation, NIH (18197), and VA Medical Research Service.

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A. BEN MANSOUR\*, and J.F. DESJEUX. Groupe de Recherches sur le Diabète et la Nutrition chez l'Enfant, INSERM U. 83. CHU VILLEMIN. 10, avenue de Verdun, 75010-PARIS-France. EFFECTS OF MALNUTRITION, AGE, AND CHOLERA TOXIN ON GLUCOSE-STIMULATED WATER ABSORPTION.

Oral administration of glucose-containing solutions reduces net stool output in patients with cholera. Since malnutrition is frequently associated with diarrhea, we examined the influence of malnutrition on intestinal response to glucose in experimental cholera. Water fluxes (Jv) were determined by weighing intestinal fluid at different times in isolated ligated loops of jejunum. The effects on Jv of glucose and mannitol (30mM + Ringer) were compared in loops with and without (C) cholera toxin (CT), in 20 adult control rats, 20 adult rats with protein-caloric malnutrition (PCM), 10 adult rats on a protein-free diet (APM), and 10 growing rats on a protein-free diet (YPM). Glucose stimulated water absorption (expressed in μl/10 min cm<sup>2</sup>) equally in control loops and in CT loops in all four groups studied. The C and CT values for Jv were respectively 36.2 ± 7.5 and 39.6 ± 11.9 in control rats, 64.4 ± 8.3 and 73.8 ± 10 in PCM, 37.1 ± 8.1 and 41 ± 5.2 in APM, and 18.9 ± 3.5 and 18.1 ± 2.6 in YPM. Similarly, CT did not modify the fluxes of Na, Cl, HCO<sub>3</sub> and glucose. The relationship between Jv, JNa and Jglucose remained unchanged. The effect of glucose on water fluxes in intestine was therefore not influenced by cholera toxin, but greatly depended on the type of malnutrition and on the age of the rats. In addition, the present results suggest that the intestinal secretion induced by cholera toxin is independent of the intestinal absorption stimulated by glucose.

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UTILIZATION OF OLEIC ACID AND D-BETA-OH-BUTYRATE (D-BOHB) AS ENERGY SUBSTRATES BY BRAIN CELL CULTURES OF NEWBORN MICE AT DIFFERENT

GLUCOSE CONCENTRATIONS. E. BOSSI\*, H.P. SIEGRIST\*, K. ZUPPINGER; U. WIESMANN; N. HERSCHKOWITZ. Department of Pediatrics, University of Berne, Switzerland. The objective of this study was to prove the metabolization of the D-BOHB-molecule and to show the hitherto unknown role of Oleic acid as cerebral energy fuels. 1 μCi of either D-BOHB-3-<sup>14</sup>C or Oleic acid-UL-<sup>14</sup>C were added at different glucose concentrations to dissociated brain cell cultures of newborn mice after 15 days of growth. Both substrates are increasingly metabolized to <sup>14</sup>CO<sub>2</sub> with incubation time. At glucose concentrations below 0.2 mM, however, significantly (p<0.01) more <sup>14</sup>CO<sub>2</sub> is produced from Oleic acid than at higher concentrations, whereas the same quantity of <sup>14</sup>CO<sub>2</sub> can be collected from D-BOHB independently of the glucose concentration:

gluc. conc. (mM)	0.03 - 0.2	0.6 - 1.2	1.7 - 2.9	3.0 - 4.7
CO <sub>2</sub> from D-BOHB (%)	100.3 ± 7.4	103.8 ± 7.4	99.2 ± 9.7	110.2 ± 16
CO <sub>2</sub> from Oleic ac.	100 ± 10.2	76 ± 8.9	80 ± 3.4	71 ± 5.7

(100% = <sup>14</sup>CO<sub>2</sub>-production as DPM/mg prot./4 hrs. incubation at the lowest glucose concentration; ± 1SD). BOHB is steadily metabolized by neonatal mouse brain cells in culture; Oleic acid can increasingly be utilized as energy fuel when glucose availability diminishes. The quantitative significance of the two substrates for brain oxidative metabolism remains to be elucidated.