865

ANTIBODIES TO NUCLEIC ACIDS IN JUVENILE ONSET DIABETES. Shih-Wen Huang, Noel K. Maclaren, Dept. of Ped. Univ. Md. Sch. Med., Baltimore, Maryland 21201.

The study on viral agent as a possible etiological factor in the development of juvenile onset diabetes (JDM) was undertaken by surveying serum antibodies to DNA and RNA from 80 children with JDM. 129 children with asthma and 40 normal individuals were selected for controls. The asthmatics were chosen for comparison because of their increased susceptibility to viral infection and the non-autoimmune nature of the disease. We found that JDM patients had significantly increased antibody titers to single stranded-DNA (61.3%), synthetic polyadenylic-polyuridylic acid (Poly A-U) (78.3%), synthetic polyinosinic-polycytidylic acid (Poly I-C) (62.5%), and to double stranded RNA of Statolon virus (51.3%) and Reovirus (27.3%) respectively, in contrast to asthmatics (15.5%; 34.9%; 3.9%; 20.2%; 2.3%) and to normal controls (2.5%; 7.5%; 5.0%; 5.0%; 5.0%). The difference among the groups are statistically significant ($P < 0.001$). Presence of SS-DNA antibody found in two third of cases of JDM further support the increased prevalence of autoimmune phenomena in that disease. Furthermore, the increased incidence of DS-RNA antibodies in JDM, found especially in cases of recent onset, provide evidence of active immune response against the underlying viral replications which may have led to beta cell injury in islets of pancreas. (Supported from NIH Grant (A1-19256)).

868

EFFECT OF DIET ON FAT CELL NUMBER, SIZE AND ENZYMES IN OBESE PEDIATRIC PATIENTS. David P. Katz, Russell

Merritt, Kim Timmers, Jerome Knittle, Robert Suskind
Dept. of Nutr. & Food Sci., MIT, Cambridge, MA, Nutr. Support Ser. Children's Hosp., Boston, MA and Mt. Sinai Sch. of Med., NYC.

Cell size, cell number and several lipogenic enzymes were measured from adipose tissue from 4 subjects (11-15 yrs, and $>200\%$ of ideal body wt) who were undergoing wt reduction. Subjects were placed on a 2600 cal diet (A) and 2 hypocaloric diets (880 cal), one high in fat (B), the other high in carbohydrate (C). Adipose tissue was obtained by needle aspiration from the buttock at the end of each dietary period. Fat cell number remained constant whereas fat cell size significantly decreased ($p < .05$) during wt reduction. Citrate cleavage enzyme (CCE) and phosphofructokinase (PFK) activities both increased during the reducing diets. Malic enzyme (ME) and α -glycerophosphate dehydrogenase (α GPD) were highest on diet C. Two-way analysis of variance and Scheffé testing indicate that CCE, PFK, and α GPD were highest on diet C ($p < .05$). Malic enzyme remained the same on diet B, but increased on diet C ($p < .01$). These data demonstrate that adipose tissue enzymes respond to diet with some activities increasing as cell size decreases.

Enzymes ¹	A	B	C
PFK	201±30 ²	320±16	477±22
CCE	119±2	155±2	182±4
ME	314±13	313±10	1430±31
α GPD	711±10	1246±18	2026±22

¹activity= μ moles/mg. protein ²Mean±S.E.M.

866

HEMOGLOBIN A_{1c} IN JUVENILE DIABETICS, AND IN CHILDREN ON HEMODIALYSIS OR PARENTERAL ALIMENTATION. George

Hug, David Langdon, Jeff Bosken and Mary Ryan. Dept. of Pediatrics, University of Cincinnati, Ohio 45229.

Glycosylated hemoglobin (HbA_{1c}) is thought to reflect blood sugar concentration existing during the lifetime of the erythrocyte. We measured HbA_{1c} by ion exchange chromatography (Bio-Rex® 70) in hemolysates of blood specimens obtained from volunteers (NL); from juvenile diabetics (JD): (A) controlled, attending outpatient clinic; (B) uncontrolled, hospitalized; from children on hemodialysis for renal failure (D) or on parenteral alimentation (PA) for: (1) less than 2 weeks; (2) more than 1 month. Results are as follows (in mean values, percent of total hemoglobin $\pm$ 1 S.D.; N = number of assays):

	HbA _{1a+b}	HbA _{1c}	HbA
NL(N:15)	2.41 $\pm$ 0.56	4.86 $\pm$ 0.64	92.73 $\pm$ 1.09
JD-A:(N:4)	3.03 $\pm$ 0.49	11.30 $\pm$ 2.26	85.60 $\pm$ 2.73
JD-B:(N:6)	3.67 $\pm$ 0.39	16.87 $\pm$ 1.96	79.44 $\pm$ 1.74
D(N:6)	2.96 $\pm$ 0.57	6.10 $\pm$ 1.60	90.92 $\pm$ 2.12
PA-1:(N:2)	2.52	4.86	92.62
PA-2:(N:4)	4.26 $\pm$ 2.20	10.91 $\pm$ 4.43	84.88 $\pm$ 6.39

These results indicate the possibility that (1) juvenile diabetics have markedly elevated HbA_{1c} (2) HbA_{1c} is higher in uncontrolled than in controlled juvenile diabetics (3) children on hemodialysis have slightly elevated HbA_{1c} (4) patients on long term parenteral alimentation have elevated HbA_{1c} similar to that of our ambulatory juvenile diabetics.

869

AN EVALUATION OF ZINC STATUS IN PATIENTS WITH ANOREXIA NERVOSA. Barbara S. Kirschner, Regina Casper,

Robert A. Jacob, Harold H. Sandstead and John M. Davis (Spons. by S. Spector) Univ. of Chicago, Dept. of Pediatrics Chicago, IL; Illinois State Psychiatric Institute, Chicago, IL; and the USDA Human Nutrition Laboratory, Grand Forks, ND.

Characteristics of zinc deficiency include growth retardation delayed sexual maturation, alopecia and hypogeusia. Zinc deficient rats have hypovitaminosis A, thought to be caused by impaired synthesis of retinol-binding protein (RBP), a zinc-metalloprotein. Since these findings are also features of A.N., zinc status was assessed in 26 hospitalized patients (26 female, 1 male) ages 12 to 32, who had lost $>25\%$ of their body weight in the absence of other discernible medical causes. Standardized taste discrimination tests indicated no dysgeusia (taste perversions) although reduced taste acuity was present in 6 of 13 patients tested.

	Anorexia Nervosa (N=26)	Controls (N=34)	P Value
Fasting a.m.			
plasma zinc (μ g/dl)	73.9 $\pm$ 48.3	83.3 $\pm$ 9.0	$p < 0.02$
Hair zinc (μ g/gm)	201.1 $\pm$ 46.3	186.5 $\pm$ 38.1	N.S.
Serum carotene (μ g/dl)	218.1 $\pm$ 20.0	132.2 $\pm$ 39.3	$p < 0.05$
Serum vitamin A (μ g/dl)	46.9 $\pm$ 10.3	50.3 $\pm$ 9.9	N.S.
Serum RBP (μ g/ml)	53.3 $\pm$ 14.0	53.1 $\pm$ 13.8	N.S.

Plasma and hair zinc levels did not correlate with taste perception or serum carotene, vitamin A or RBP, nor did they reflect the amount of weight loss or the duration of the symptoms, including amenorrhea.

867

ESTIMATION OF GLUCOSE PRODUCTION IN THE HUMAN NEWBORN USING SIMULTANEOUS ¹³C-AND D-LABELLED GLUCOSE. Satish

C. Kalhan, Dennis M. Bier, Samuel M. Savin and Peter A. J. Adam. Pediatric Metabolism Div., Case Western Reserve Univ. at Cleveland Metropolitan General Hosp. and Washington Univ. School of Medicine, St. Louis.

In order to compare two methods of quantifying systemic glucose production (SGP), 5 newborn infants age 2 h-3 days were infused simultaneously with glucose-1-¹³C (G1¹³C) and glucose-6-⁶D₂ (G6D₂) tracers. The older infants were studied 6 h after a meal. G1¹³C was infused at 6 μ g/Kg min according to the prime constant rate infusion technique and ¹³C enrichment of C-1 of glucose measured by enzymatic decarboxylation and mass spectrometry. G6D₂ was infused at 60 μ g/Kg min and deuterium enrichment of glucose measured by gas chromatography-mass spectrometry. SGP was calculated from the tracer dilution assuming steady state kinetics, from 30 min for ¹³C and from 120 min for D. Although ¹³C was expected to randomize away from C-1, recycling occurred and was estimated from the difference in rate of SGP quantified by dilution of the two tracers (mean $\pm$ S.D.)

Plasma Glucose mg/100 ml	¹³ C 30-90'	¹³ C 120-180'	D 120-180'
54.8 $\pm$ 10.1	4.12 $\pm$ 0.58	3.93 $\pm$ 0.31	4.65 $\pm$ 0.39

SGP ranged from 3.5 to 5.1 mg/Kg min. Recycling of glucose C-1 was 11-15% of SGP and it did not change with the age of infant. Conclusion: 1) SGP is established in the human newborn by 2 hours of age. 2) The two stable tracers used give similar estimates of SGP.

870

PLASMA BICARBONATE LEVELS AND TISSUE LEAD DISTRIBUTION

Joseph A. Kochen, Albert Einstein Coll. Med., Montefiore Hosp. and Med. Ctr., Dept. Peds., The Bronx, NY.

Among factors known to enhance the toxicity of lead (Pb) are dehydration and acidosis. In this study the effects of reduction of plasma bicarbonate (HCO₃) concentration on the uptake of plasma Pb by red blood cells (rbc) and other tissues was investigated. Depletion of HCO₃ in human plasma was accomplished with comparable results by ultrafiltration, dialysis or CO₂ absorption with alkali. Following standardization of plasma pH, the uptake of Pb²¹⁰ by added rbc was measured. Depletion of plasma HCO₃ to levels below 10 mEq/L resulted in a consistent decrease in the transfer of Pb from plasma to rbc. At 10 minutes, 38.0 $\pm$ 12.6% (9) of Pb²¹⁰ in HCO₃ - depleted plasma was taken up by rbc, as compared to 75.4 $\pm$ 7.9% (20) from control plasma ($p < .001$). Addition of NaHCO₃ or replenishment of plasma HCO₃ by gassing with CO₂ resulted in an increased Pb uptake by rbc to control levels. In the anesthetized rat, acidosis and reduction of plasma HCO₃ by I.P. injection of HCl resulted in an increased uptake of intramuscular Pb²¹⁰ by plasma (+118%, $p < .01$), liver (+97%, $p < .05$) and heart (+63%, $p < .01$) and decreased Pb uptake by kidney (-63%, $p < .05$). The change in distribution of blood and tissue Pb following the reduction of plasma HCO₃ is similar to that observed when iron salts are added to plasma. It is of particular interest that both iron and HCO₃ are specifically bound by transferrin. These findings suggest an interaction between Pb and HCO₃ which may be mediated by transferrin and which affects the tissue distribution of Pb.