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LOW SERUM 3,3',5' TRIIODOTHYRONINE (REVERSE T₃) CONCENTRATION IN BURNED CHILDREN AND ITS RELATIONSHIP TO NUTRITIONAL STATE. Craig L. Kien, William J. Vanjonack, and Hans H. Bode. Harvard Medical School, Shriners Burns Institute, Dept. of Pediatrics, Boston and MIT, Dept. of Nutrition, Cambridge, Mass.

We studied the correlation between protein turnover and serum thyroid hormone levels in 10 children, age 1-16 yrs., with severe burn injuries. Whole body protein synthesis (S) and breakdown (C) were determined using an (¹⁵N) glycine infusion technique. Patients were maintained in good nutritional status as evidenced by a net anabolism (S>C) in 14 of 21 studies. There was no change in mean levels of serum 3,3',5' triiodothyronine (T₃), a decrease in serum thyroxine (T₄) and a suppression of serum reverse T₃ levels.

	Burned ($\bar{m} \pm S.D.$)	Controls	p Value
Age (yr.)	9.2 \pm 4.5 (n=10)	11.8 \pm 3.9 (n=11)	p >.05
T ₄ (ug%)	6.4 \pm 1.6 (n=10)	8.3 \pm (n=12)	p <.005
T ₃ (ng%)	152 \pm 71 (n=10)	146 \pm 23 (n=14)	p >.05
rT ₃ (ng%)	15.9 \pm 17.7 (n=10)	50.8 \pm 14.1 (n=14)	p <.0005
rT ₃ /T ₄	2.2 \pm 2.1 (n=10)	5.8 \pm 1.6 (n=11)	p <.005

The difference between protein synthesis and breakdown (S minus C) was negatively correlated with the rT₃/T₃ ratio (r=-.046, p .05). Thus, a decrease in N balance was associated with a rise in rT₃/T₃ ratio, similar to observations made in humans during fasting or severe stress. These studies suggest that the rT₃/T₃ ratio may be useful clinically as an indicator of relative dietary energy deficiency.

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COMBINED GI ALLERGY AND IMMUNOGLOBULIN DEFICIENCY: ROLE OF CROMOLYN. Samuel Kocoshis and Joyce Gryboski Yale U. Sch. of Med., Yale-New Haven Hosp., Dept. of Ped., New Haven, Ct.

Ninety-eight infants fulfilled criteria for GI allergy after disaccharide tolerance tests and small bowel biopsy when indicated. Of 62 allergic to cow's milk, 58 developed soy and 24 gluten allergies. Of 10 initially allergic to soy, 10 were later allergic to milk and 7 to gluten. 20 children under 3 developed allergies after gastroenteritis: 20 to milk, 16 to soy and 4 to gluten. Nearly 30% of primary milk and 105 of soy allergic infants and 20% postgastroenteritis allergic infants had low or absent serum IgA (mean 8 mg%). Twelve per cent of milk allergic patients and 5% of those with secondary allergies had serum IgG less than 300 mg%. Two-thirds of the IgA deficient and all but 1 IgG deficient infant had normal levels by 15 months. Oral Cromolyn (DSCG) or placebo was used in a 90 day double blind crossover in 14: 10 responded to drug, 3 to placebo (p<01) and 1 attained tolerance on placebo. Discontinuation of drug resulted in exacerbation by 2 days in 4, by 14-21 days in 5 and 2 have been asymptomatic for 6 months. There were no hepatic, renal or hematologic impairments.

Food allergies in infants are frequently multiple and often are associated with immune deficiency. DSCG is an efficacious modality of treatment in children who require severe dietary restrictions.

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USE OF ORAL NUTRITIONAL SUPPLEMENTS TO REVERSE GROWTH RETARDATION IN CHILDREN WITH CROHN'S DISEASE.

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Growth retardation occurs in approximately 20% of children and adolescents with Crohn's disease (CD). Total parenteral and peripheral intravenous alimentation have reversed this growth retardation, but these methods necessitate prolonged hospitalization. Six consecutive prepubertal patients with CD, all <3rd percentile in height, had caloric intakes 50-73% of the RDA for height age. The effect of oral nutrition combined with medical therapy upon growth velocity was studied in these 6 pts. Low residue formula supplements were added to the diet to provide 80-100 kcal/kg/day and at least 1.5 gm protein/kg/day. Medications were sulfasalazine alone (3 pts) or sulfasalazine and Prednisone (3 pts). Prednisone was given daily for 6-16 wks, then changed to an alternate day regimen. Before therapy, the growth velocities of 5 of the 6 pts were 1.1-1.9 cm/yr (>2 SD below normal); one had a falling growth velocity but remained in the low normal range. Three of these pts were completely growth arrested for >1 yr. After therapy, growth velocities increased to 6.5 \pm 2.0 cm/yr in 5 pts, which is within the normal range. One pt grew only 1.8 cm/yr which subsequently increased to 5.5 cm/yr after 4 wks of peripheral intravenous alimentation. These results indicate that oral calorie/protein supplementation in conjunction with medical therapy is an effective method for reversing growth retardation in prepubertal CD patients.

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JEJUNOILEAL GRADIENT OF SUCRASE ACTIVITY IS PROGRAMMED IN FETAL RATS IN UTERO. O. Koldovsky,

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Sucrase activity is absent in the small intestine of fetal rats, but it increases during the weaning period. At the same time, its jejunoileal gradient, known to exist in most adult mammals, appears. Mechanisms leading to the expression of biochemical specialization of individual sections of the small intestine are not clear.

Fragments of fetal rat jejunum and ileum (21-day old, CDF strain Charles River Farm) implanted under the kidney capsula of adult rats (Gastroent. 64:292 and J. End. 74:145) were assayed after 4 and 6 weeks for sucrase activity and compared with small intestine developing in situ.

POSTCON- CEPTIONAL DAYS	IN SITU		IMPLANTS	
	Jejunum (N)	Ileum	Jejunum (N)	Ileum
49	4.3 \pm 0.2*(15)	0.3 \pm 0.04	6.4 \pm 0.8 (11)	3.5 \pm 0.5
66	4.3 \pm 0.4 (7)	0.4 \pm 0.01	10.4 \pm 0.8 (7)	5.0 \pm 0.9

* μ moles sucrose split per 60 min/mg prot \pm S.E.M.

Results demonstrate that expression of jejunoileal gradient of sucrase activity is not dependent on direct contact of food; this factor as well as gastric, duodenal and pancreatic secretion might be responsible for its magnitude. (Support: NIH grant #AM HD 14531).

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FECAL BILE ACIDS IN GREEN DIARRHEA. Samuel Kocoshis, Cameron Ghent, Joseph Bloomer and Joyce Gryboski, Yale U. Sch. of Med., Yale-New Haven Hosp., Depts. of Ped. and Med., New Haven, Ct.

Fecal bile acids were measured by gas chromatography in 39 ambulatory children (age 3 mos.-4 yrs.) with chronic diarrhea of 2-18 mos. duration. Ten normals (age 2 mos.-4 yrs.) were controls. Clinical response to cholestyramine or Pepto Bismol was compared in those with brown or green diarrheal stools. Within each category, bile acid levels were unrelated to age, sex, diet or etiology. The normals' mean excretion was 1.43 mg/gm dry wgt. (.35-3.4). Those with green stools had a mean of 8.16 mg/gm (1.63-29.05), and those with yellow or brown stools had a mean of 3.72 mg/gm (.6-10.48). Bile acids in green stools were significantly elevated above normals (p .01) and brown stools (p .025). Thirteen of 15 responded to Pepto Bismol or cholestyramine and more than doubled fecal excretion, but 0 of 8 with brown stools responded. Treatment required an average of 4 wks. (3 days-3 mos.). B₁₂ absorption was abnormal in 2 of 3 with green stools and 0 of 2 with brown stools. This study suggests that abnormal bile acid metabolism is present in chronic, watery, green diarrhea, and bile acid binding agents (cholestyramine and Pepto Bismol) are indicated for treatment. We speculate that ileal dysfunction plays a pathogenetic role in this condition.

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SERIAL MEASUREMENTS OF SERUM CARNITINE IN REYE'S SYNDROME (RS). Lester L. Lansky, Leland Hong, George Hug,

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Serum carnitine was determined in 3 children with stage III RS and 1 child with stage II RS during the acute phase of their illness. Normal levels (.037 \pm .014 μ mol/ml) occurred on admission followed by a 1.5-2.5 fold elevation (.072-.086 μ mol/ml) 8-24 hours later. Serum carnitine in 2 children with stage III RS peaked just prior to or shortly after exchange transfusion. One child with stage II RS exhibited normal carnitine values in the first 4 days, as did one child with stage III RS. Forty children from 2-15 years with no liver or muscle disease served as controls. A 6-year-old girl with acute viral hepatitis (non A, non B) demonstrated normal-subnormal levels (.042-.004 μ mol/ml) during the acute phase of her illness (>2 SD below the mean). Elevation of lactic acid (normal 20 mg/dl), blood ammonia (up to 100 μ g/dl) AST, ALT (up to 35 u/ml), CPK (50 lu/l) and stage of coma (Huttenlocher staging) did not correlate with serum carnitine values. All RS patients received similar intensive medical support and ICP monitors. The RS children survived and serum carnitine levels returned to normal on recovery. Elucidation of the contribution of carnitine to the pathogenesis of generalized mitochondrial dysfunction in RS remains to be determined. Further studies are in progress to define the role of carnitine aside from its previously described function for transport of acyl long chain fatty acids across outer and inner mitochondrial membranes.