439 EFFECT OF PHOTOTHERAPY FOR NEONATAL JAUNDICE ON RIBOFLAVIN DEPENDENT ENZYMES, GLUCOSE-6-PHOSPHATE DEHYDROGENASE ACTIVITY (G6PD) AND REDUCED GLUTATHIONE (GSH) CONTENT OF BLOOD.

Lois H. Gagnon, Thomas R. Boggs, Mirtes V. Beirao, Univ. of Pa. Sch. of Med., Pa. Hosp, Dept. Pediat. Phila. For the past 5 years we have been doing serial determinations of the activity of the flavin adenine dinucleotide (FAD) dependent enzyme, glutathione reductase (GR) in RBC's of babies undergoing phototherapy. Activity, following in vitro exposure to FAD, is also measured (method of Glatzle). More than 20% increase is evidence of riboflavin deficiency. A summary of data collected in our nurseries from 1971-73 is given below. Similar data have been collected since. No evidence of phototherapy related deficiency has yet been found in these 5 years. Similarly there has been no evidence of treatment related decrease in G6PD activity or GSH blood content. However low GSH levels are not infrequently found in small sick babies regardless of Rx. Beginning studies of a second FAD dependent enzyme, methemoglobin reductase (MR) are showing similar results, except that MR activity in contrast to that of GR is lower in newborn RBC than in adult. Occasional instances of slightly lower activity following onset of Rx have all reversed themselves before cessation of Rx. These data differ from those of Gromisch et al. perhaps because of differences in maternal nutrition.

Activation	Before	Length of time after Photo Rx	10-56 hrs
Coef- GR	Photo Rx	1 day	2 days
Mean	1.02	1.03	1.00
	(N=51)	(N=23)	(N=21)

440 ANEMIA IN FAMILIES OF IRON DEFICIENT INNER-CITY SCHOOL CHILDREN: A FOLLOW-UP STUDY, 1972-1976.

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A hematologic survey conducted in 1972 identified 68 of 1210 children 5 to 8 years of age as iron deficient. The 68 families were investigated; in 1975, 38 of the families were reinvestigated. In 1975, 9 infant children had a mean hemoglobin concentration (HGB) of 10.4 gm/dl (SD=0.97) as compared to a mean HGB of 11.5 (SD=1.38) for 13 similarly-aged children in these same 38 families in 1972 ($p < 0.005$). For 39 children 3 to 10 years of age at both times of testing, mean HGB was 10.8 in 1975 as compared to 12.0 in 1972 ($p < 0.001$). For the 1210 children originally surveyed in 1972, mean HGB was 12.25 (SD=1.03). In 1976 a survey of 221 similarly-aged children in a single school revealed a mean HGB of 11.8 (SD=1.04) ($p < 0.001$).

During the period between investigations, there were dramatic changes in food costs, and nationally, in patterns of food consumption. A survey of local supermarket sales showed significant reductions in purchases of meat products in the impoverished area of Philadelphia where these investigations took place; no such change was demonstrated in a prosperous area of the city.

A rise in the prevalence of specific nutrient deficiency is known to occur in underdeveloped countries in association with rises in the cost of food staples. The data presented herein suggests that a similar phenomenon has occurred here.

441 INTESTINAL ORGAN CULTURE: THE DEFINITIVE DIAGNOSIS OF GLUTEN SENSITIVE ENTEROPATHY (GSE). Aubrey J. Katz, Z. Myron Falchuk (Spon. by R. Grand). Harvard Medical School, Children's Hospital Medical Center, Peter Bent Brigham Hospital, Department of Pediatrics and Medicine, Boston.

The "flat" jejunal lesion is not pathognomonic of GSE and is observed in various diseases. This study evaluates the "organ culture model of GSE" (JCI 53:587, 1974) as a tool for the specific diagnosis of GSE. We prospectively biopsied 70 patients (pts) with diarrhea and malabsorption 35 of whom had a "flat" jejunal lesion of varying degree. 22/35 were subsequently shown to have GSE by conventional criteria. Alkaline phosphatase activity (APA) was assessed before and after 24 hour culture in the presence and absence of gluten. All 35 pts had initial low values of APA which increased when cultured in the absence of gluten. ($P < 0.001$). APA rise was inhibited in 18 of 22 pts with GSE in the presence of gluten (gluten vs gluten free $P < 0.005$) but in none of 13/13 pts with non GSE jejunal flattening.

	BASELINE*	GLUTEN	GLUTEN FREE
GSE (n=22)	59±9.8 (mean ±SE)	100±16	190±16
Non GSE (n=13)	69±21	219±100	178±35

*APA=μm p-nitrophenol phosphate/gm protein/min +P<0.005.

Conclusion: 80% of pts with GSE exhibited in vitro gluten sensitivity while gluten had no effect on non-GSE flat jejunal mucosa. In vitro gluten sensitivity appears to be specific for the diagnosis of GSE, thus perhaps obviating repeat biopsies and challenge to confirm the diagnosis in these pts.

442 RELATION OF SPECIFIC DYNAMIC ACTION (SDA) TO WEIGHT GAIN IN NORMAL AND MALNOURISHED RATS. Ingeborg Krieger

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In two independent studies of infants recovering from malnutrition Krieger et al. (J. Ped. 1969) showed a positive linear correlation between SDA and weight gain and lack of a correlation with nitrogen (N) intake, while Ashworth (Nature 1969), noting also a correlation with weight gain, called for a "new concept of SDA." It is not known whether during growth normal individuals show the same relationships. In this study malnourished rats had metabolic rate measurements 4 and 17 hours after food removal. The difference was 3.9% during growth arrest at 5 weeks, but 20.0 and 28.7% during growth recovery at 7 and 14 weeks, suggesting that the mechanism is the same as in human infants with malnutrition. Measurements were therefore conducted in 39 normal 4 to 36 week rats. The metabolic rate, 4 hours after food removal, rose from 15.6 to 45.1 kcal/day at 10-13 weeks, and then declined to 38.5 kcal/day at 32-36 weeks. The difference between the 4 and 17 hour values, which can be attributed to SDA, was determined in 21 rats. It was 28.8% in 5-15 week old rats who gained 5.2 g/day and 1.1% at 24-36 weeks when weight gain had ceased.

CONCLUSION: SDA reflects energy requiring anabolic processes in normal and malnourished rats. Similar observations in malnourished human infants thus do not represent an abnormal phenomenon of catch-up growth. These findings are not incompatible with the correlation between SDA and N-intake known to exist in adults who are in N-balance. A new concept of SDA is indeed necessary.

443 ENTEROPATHOGENIC E. COLI IS A CAUSE OF RECURRENT DIARRHEA IN OLDER CHILDREN. William M. Liebman, (Spon. by Dr. Carolyn Piel) University of California, San Francisco, Department of Pediatrics, San Francisco. 94143

Various strains of E. Coli are known to cause acute gastroenteritis in newborns and infants. In older infants and children, infection with enteropathogenic E. Coli may cause chronic diarrhea. Pathogenic E. Coli were cultured from the stools of 31 children ranging in age from 4 months to 13 years. Of these, 4 were less than 2 years old, 11 were 2 to 5 years old, and 16 were over 5 years old. Isolated strains of E. Coli included 0127 in 11, 0111 in 8, 0126 in 5, 0128 in 3, 055 in 2, 026 in 1, and 0119 in 1. Recurrent episodes of diarrhea consisting of at least 4 loose or watery stools per day, were observed in all patients. Episodes recurred every 4 to 7 days in 9 patients, every 1 to 4 weeks in 14, and every 1 to 4 months in 8. Fever was present in 1 patient, weight loss in 2, and anorexia in 1. Physical examination and laboratory studies were unremarkable in all patients. Proctoscopy and rectal biopsy performed in 9 patients yielded normal results. Treatment with neomycin in 26 and colymycin in 5 eliminated the diarrhea and eradicated the E. Coli from stool cultures. These results indicate that enteropathogenic E. Coli may be a frequent cause of recurrent diarrhea. This type of infection appears to respond readily to treatment with antibiotics.

444 CIMETIDINE CAN CONTROL PEPTIC ULCER DISEASE IN CHILDREN. John R. Lilly, David C. Hitch (Spon. by Frederick C. Battaglia). University of Colorado School of Medicine, Department of Surgery, Denver.

Cimetidine is a newly developed, potent H₂ antagonist of gastric acid secretion currently under restricted investigation in the United States for treatment of peptic ulcer in adults. Efficacy in children was evaluated in a prospective study in 3 patients 14, 2 and 14 years old with life-threatening ulcer disease. In the adolescent, giant duodenal ulcers consequent to loss of all large and 90% of the small bowel healed rapidly despite marked persistent hypergastrinemia (5 x's normal). Ulceration recurred within 10 days after drug discontinuation necessitating its resumption and ongoing use. Major hemorrhage from stress ulcers (steroid-induced, biliary atresia/post-Kasai operation) in the other two children ceased within 24 hours after institution of cimetidine. Serial gastric acid studies in the smaller children showed inconstant control of gastric secretion probably due to lack of experience with dosage. Nevertheless, there was prompt radiographic healing of duodenal ulceration in both patients. Drug toxicity was monitored by sequential hematologic, renal, hepatic and neurologic studies. Although most likely not drug-related, a transient decrease in bile output in one patient and lethargy in another represented the only possible toxic side effects observed thus far.