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As changes of erythrocyte glutathione metabolism is a sensitive index of the in vivo oxidative processes, reduced glutathione (GSH), oxidized glutathione (GSSG), stability of GSH and oxidation state of haemoglobin have been measured. Data of 11 children with acute celiac disease/ACS/ 11 children on gluten free diet/GFCD/ were compared to those of 11 children with nutritive iron deficiency/ID/ and to 11 healthy children as controls. Erythrocyte GSH content of ACD group elevated significantly, the GSSG level decreased compared to normal controls. GSSG and GSSG/GSH ratio also differed from those of ID group, and a marked difference has been measured in glutathione stability, too. In ACD group methaemoglobin and haemoglobin were measured. Anomalies of erythrocyte metabolism normalised during the diet period. The decreased GSSG/GSH value of ACD group refers to decreased activity of selenoenzyme glutathione peroxidase, a significant selenium deficiency $228 \pm 158 \text{ ng/gHb}$ has been proved to normal controls $465 \pm 136 \text{ ng/gHb}$. Our results suggest that during gluten challenge ACD patients suffer from chronic oxidant stress which partially can be explained by the weakness of scavenger system and by selenium deficiency.

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AREA UNDER pH 4 : ADVANTAGES OF A NEW PARAMETER IN THE INTERPRETATION OF ESOPHAGEAL pH MONITORING DATA.
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24 Hour esophageal pH monitoring has become the preferred test to quantify acid gastro-esophageal reflux. Agreement has been achieved that refluxed acid gastric content into the esophagus constitutes a major cause of reflux esophagitis. We therefore calculated the "area under pH 4" (A) in 560 consecutive pH monitor in infants 1-12 months old, and related this parameter to the % time with $\text{pH} < 4$ (reflux index, RI): RI < 10%: A 185 ± 295 (mean $\pm$ 1 SD); RI 10-19%: A 1046 ± 1206 ; RI 20-29%: A 1967 ± 2038 ; RI 30-39%: A 3307 ± 2955 ; RI > 40%: A 7977 ± 7227 . The "A" is related to the RI whereas according to the RI the "A" increases significantly ($p < 0.001$); the high SD, however, obtained in each group illustrates the great variability in "A" (or acidity of the reflux episodes) in all groups. Esophagoscopy (and biopsy) was performed in 112 infants (20%). Specificity in the prediction of esophagitis was higher for the "A" (88%) compared to the RI (50%). Sensitivity of both parameters was comparable (96% versus 93%). Our results strongly suggest that the acidity of the reflux episodes ("A") is a determining factor in the prediction of esophagitis. These data need to be confirmed by more studies (on adult patients) before general application of this parameter can be advised.

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PULSE GROWTH DURING CATCH UP IN COELIAC DISEASE
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Celiac children exhibit a considerable increase in growth velocity during the remission phase.

Objective of the research is to analyse the dynamics of catch up growth on gluten-free diet.

Methods. Height and weight of 98 Celiac children, diagnosed according to the 1970 ESPGAN protocol, have been obtained at monthly intervals on gluten free diet by quality controlled procedures. Growth velocities have been computed between monthly measures.

Results. When individual's growth velocities are computed at 3-6 months distance between measurements, most cases show a single peak of height and weight velocities. When velocities are computed at monthly intervals each case exhibits at least 2 peaks in the first year of gluten free diet. "Mean" velocity curves have been obtained by measuring 8 parameters from 98 individual curves. For height as well as for weight the pattern shows a first peak after 63 days of diet and a second peak after 200-210 days of diet.

Conclusions. Catch up growth in treated celiac children is not a continuous and linear process, but it occurs by a pattern of Pulse Growth, with an alternation of peaks and nadirs. Pulse growth is not specific of celiac disease, but probably common to several chronic conditions in childhood. The results obtained in celiac cases give a first concrete evidence of pulse growth in weight as well as in height in children.

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MOLECULAR STUDY OF ADULT HYPOLACTASIA IN RABBIT AND HUMAN SMALL INTESTINE.

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In mammals, including most human populations, intestinal lactase activity is very high in the suckling and declines to low levels after weaning. There are two lactase phenotypes among human adults, one with persistent high lactase activity and one with hypolactasia(1). In the present study lactase mRNA levels have been measured by cDNA probes on northern blot of poly(A)+RNA normalized by B-actin in the small intestine of rabbit, at different ages, and on slot blots of total RNA and poly(A)+ RNA of human adults with persistent high lactase activity and with hypolactasia. An ECO RI-Hind III fragment covering more than the 5'-half of the rabbit lactase cDNA and the full human lactase cDNA were used as probes (2). mRNA levels have been compared with those of lactase activity (3). In the rabbit mRNA appears by the end of the gestational period and a level, comparable to that found in the suckling animal, is still present in the adult life. The lactase activity appears at the same time of the mRNA, but it declines to very low levels postweaning. Similarly, in man, at RNA level, no clear differences were found between adults with hypolactasia and adults with persistent high lactase activity.

Conclusion: In adult mammals and in adult human hypolactasia, the control of lactase gene expression is very likely to be at a post-transcriptional level.

1) Auricchio S. et al. Lancet 2, 324-326, 1963; 2) Mantei N. et al. The EMBO J. 7, 2705-2713, 1988; 3) Asp NG. et al. Anal. Biochem. 47, 527-538, 1972.

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ESOPHAGEAL MOTILITY(EM) IN CHILDREN WITH HIRSCHSPRUNG'S DISEASE(HD). A. Staiano, S. Cucchiara, E. Corazzari, MR. Andreotti, A. Della Rocca, Dept. Pediatrics, Univ. of Naples, Gastroenterologia I, Univ. La Sapienza of Rome, Italy.

It has been reported that patients with severe idiopathic constipation may be affected by diffuse abnormalities of the myenteric plexus (Gastroenterology 1985;88:26-34). HD may affect any part of the colon but little attention has been given to a possible dysfunction of gastrointestinal tract other than the colon. The aim of our study was to evaluate EM in unoperated children with HD, without fecal impaction for continuous treatment with daily enemas. Esophageal manometry was performed in 12 children (mean age: 2.4 years) with HD, diagnosed by barium enema, rectal manometry and deep rectal biopsy. Manometric tracings, blindly read, were compared with those of 10 age-matched children with emesis without proven gastrointestinal motility disorders. Lower Esophageal Sphincter (LES) pressure, LES relaxation with swallows and duration of esophageal waves did not differ between HD and control children. Amplitude of esophageal waves (mm Hg) was significantly higher in HD children than in controls (73.5 ± 15.9 , mean $\pm$ SD; 50.7 ± 20.7 , respectively, $p < 0.05$). Simultaneous and double peaked esophageal waves were significantly more frequent in HD children than in controls (56.9 ± 19.8 ; 7.8 ± 5.9 ; $p < 0.001$). This study shows that EM abnormalities are a frequent occurrence in HD children and suggests that in these patients motor dysfunction is not limited to the colon as it may affect other gastrointestinal tracts.

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CORRELATION OF SERUM HYALURONIC ACID (HA) AND LAMININ (LAM) WITH STANDARD LIVER HISTOLOGY AND HISTOMORPHOMETRY-PROVEN LIVER FIBROSIS IN CHILDREN AFFECTED BY CHRONIC HEPATITIS B VIRUS (HBV) INFECTION
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Non invasive assessment of the progression of liver disease in chronic HBV infection is crucial in childhood, where long follow-ups are expected. Value of several markers of liver fibrosis, cirrhosis and portal hypertension in children with chronic HBV infection however is at present either questioned (i.e. N-terminal propeptide of type III procollagen) or poorly known (i.e. HA, LAM). We therefore studied by RIA method the serum levels of HA(1) and Laminin Pepsin Resistant Fragment P1 (LAM P1) (2) in relation to: A) standard histologic grading of liver disease severity (Student t), B) degree of portal tract fibrosis (um) assessed by computerized histomorphometry using a Zeiss Kontron Videoplan analysis system (Pearson correl.) in 53 children with chronic B hepatitis (age 7.7 ± 3.9 y; range 2-13 y), 13 HBV carriers and 18 age and sex matched healthy controls.

Results:

A.	n	HA ($\mu\text{g/L}$)	$\pm$ SD	n	LAM P1 (U/ml)	$\pm$ SD	Signif. vs. CAH+CIR
Controls	18	22.3 ± 7.6		15	1.39 ± 0.16		HA LAM P1
Carriers	13	18.8 ± 8.8		17	1.56 ± 0.36		$p < .01$ $p < .025$
CPH	21	24.4 ± 21.5		13	1.57 ± 0.20		$p < .001$ NS
CAH	20	66.2 ± 153.0		20	1.55 ± 0.24		$p < .02$ NS
CAH+CIR	12	110.8 ± 124.2		4	1.65 ± 0.36		-

(CPH=Chronic Persistent Hepatitis; CAH= C Active H; CIR=Cirrhosis)

B. Portal fibrosis vs HA $r = .63$, $p < .001$; vs LAM P1: $r = .49$, $p < .002$

Conclusions: HA serum levels may prove a useful quantitative marker for evaluating histologic liver CIR and fibrosis in children with chronic HBV infection. Serum levels of laminin, a constituent of basement membranes in the perisinusoidal space, probably reflect several degrees of portal hypertension, rather than liver CIR itself. Correlations between LAM P1 serum levels and hepatic sinusoids capillarization are presently under study in our patients.

1) Hepatology, 1985, 5: 638-42. 2) J Clin Chem Clin Biochem, 1985, 23: 572-3