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RECTAL TISSUE IMMUNOGLOBULIN PATTERNS IN CHILDREN WITH INFECTIOUS COLITIS. T.C. Halpin & M.E. Ament. Depts. of Pediatrics, CWRU, Cleveland, OH. & UCLA, L.A., Ca.

We have employed a single phase antibody radioimmunoassay to determine rectal tissue and secretion immunoglobulin (Ig) levels in children with documented infectious colitis. All children had positive stool culture and/or rectal swab for pathogen, abnormal proctosigmoidoscopy and pathologic diagnosis of acute colitis. Tissue was placed in rectal organ culture for 24 hours and explants were homogenized (H) and assayed for IgA, IgG and IgM. Secretions (S) were collected over center well of organ culture plate and assayed for Ig's. Results were expressed as µg Ig per mg tissue protein. Results were compared with a control population of children with Functional Abdominal Pain who had normal proctosigmoidoscopy and pathogen-negative stool cultures.

	N	Age(ave.)		IgG	IgM	IgA
Control	11	6.9 yr	H	1.42 ± .48	1.3 ± .47	2.6 ± .14
			S	5.08 ± 2.4	1.03± .20	4.23± 1.04
Infectious Colitis*	8	4.3 yr	H	9.92 ± 4.04	5.3 ± 3.66	10.15± 5.26
			S	20.76 ± 18.98	4.59± 3.07	25.64± 11.3

* Shigella sp. (5), E. coli (2) & Salmonella sp. (1).

There appears to be a marked symmetrical increase in rectal tissue Ig's whether comparing total Ig ($p<0.005$) or individual homogenates ($p<0.001$) or secretions ($p<0.01$). The presence of increased amounts of Iga, IgM & IgG in infectious colitis should influence further investigations into other inflammations of the colon (i.e., ulcerative colitis, cow's milk protein-induced colitis, etc.).

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RECTAL IMMUNOGLOBULIN PATTERNS IN HIRSCHSPRUNG's DISEASE (HD) AND OTHER NEONATAL INTESTINAL OBSTRUCTION SYNDROMES (NIOS). T.C. Halpin & R.J. Izant. Depts. of Ped & Surg, CWRU, Cleveland, OH. (Sponsored by R.E. Behrman.)

The etiology of HD remains unknown and can be confused with other NIOS. Rectal biopsy has traditionally established the diagnosis of HD. In the present investigation, rectal tissue was obtained with multipurpose suction tube at 1-2 cm above the mucocutaneous junction. Proctosigmoidoscopy was normal in all cases. In addition, rectal tissue was placed into organ culture for 24 hr. A single phase antibody radioimmunoassay was used to determine IgG, IgM & IgA levels in homogenized cultured rectal tissue (H) and its secretions (S). Results are expressed as µg of immunoglobulin/mg tissue protein. HD & NIOS have been compared to an older control group of children with Functional Abdominal Pain.

	N	Age(ave.)		IgG	IgM	IgA
Controls	11	6.9 yr.	H	1.42± .48	1.3 ± .47	2.6 ± .14
			S	5.08± 2.4	1.03 ± .20	4.23 ± 1.04
HD	6	2.3 d	H	5.73± 1.9	.82 ± .55	1.19 ± .48
			S	12.45± 3.98	.40 ± .28	2.98 ± 1.33
NIOS*	5	3.4 d	H	1.92± .71	.73 ± .43	1.11 ± .20
			S	4.66± 1.44	.66 ± .36	2.08 ± .48

*Meconium Plug (3), Left Colon Syndrome (1), Motility Disorder(1)

For HD, an increase of IgG in both homogenate and secretions was noted compared to controls ($p<0.005$) and NIOS ($p<0.005$). Results suggest there is either increased tissue synthesis of IgG or transfer of serum maternal IgG. The explanation of the observed increased IgG in rectal tissue of children with HD is unknown

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X-LINKED AGAMMAGLOBULINEMIA AND INFLAMMATORY BOWEL DISEASE. T.C. Halpin, S.H. Polmar, R.J. Izant, R.U. Sorensen, E.R. Reece & E.M. Smithwick. Depts. of Pediatrics and Surgery, CWRU, Cleveland OH. and Sloan Kettering Cancer Center, New York, N.Y.

A 15-year-old boy with x-linked agammaglobulinemia (X-LA) was evaluated for growth retardation, malabsorption and progressive inflammatory bowel disease. He had absent IgG, IgM and IgA by immunoelectrophoresis. Surface immunoglobulins (Ig) were absent by polyvalent and IgM antisera. EAC-rosette forming lymphocytes were decreased (6% vs. 14% control). E-rosette forming lymphocytes were near normal (47% vs. 61% control). PHA, ConA and PWM-stimulation were normal. Response to skin tests with Mumps and SKSD were normal; monilial antigen were nonreactive. Small bowel series revealed fixed, nodular ileum with widely separated loops. Rectal biopsy showed "nests" of lymphocytes. There were no plasma cells or detectable Ig's by immunofluorescent staining. Fecal fat excretion (coef.=82), infused IgG half-life (4 days) and Schilling Test (0% excretion) were abnormal. Laparotomy was performed and revealed inflammatory, polypoid-like ileal mucosa. There was massive infiltration of the mucosa and submucosa with mature lymphocytes. Polypoid-like lesions represented masses of lymphocytes. There were no detectable tissue Ig's as determined by single phase antibody radioimmunoassay. E-rosettes forming intestinal lymphocytes were 51%; EAC-rosetting lymphocytes were 5%. No virus was isolated from surgical tissue. This case appears to represent a new, unclassified inflammatory bowel disease of unknown etiology in a child with X-LA.

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FAT DIGESTION IN THE STOMACH OF PREMATURE INFANTS: ORIGIN OF THE LIPOLYTIC ACTIVITY. Margit Hamosh, Carol Salzman-Mann, Kolinjavadi N. Sivasubramanian, Gordon B. Avery, Teresa Plucinski, John B. Watkins and Paul Hamosh. Georgetown University School of Medicine and Children's Hospital, Washington, DC and Children's Hospital, Boston, MA.

Digestion of dietary fat in the adult is intitiated in the stomach by a lipase similar to that present in lingual serous glands (Hamosh et al. J. Clin. Invest. 55:908, 1975; Lab. Invest. 37, 1977). Recently, we have reported similar activity in gastric aspirates of premature infants (Hamosh et al. Physiologist 20:40, 1977). In order to determine the origin of the lipase, we have tested esophageal and gastric aspirates obtained from four infants with congenital esophageal atresia. Lipolytic activity (tested with doubly labeled ^{3}H-glyceryl-^{14}C-tripalmitin) was present in both esophageal (14.24 ± 10.6 n mol/ml/hr) and gastric (6.97 ± 2.31 n mol/ml/hr) aspirates; the reaction products were partial glycerides, glycerol and free fatty acids; pH optimum was 5.4. The data support previous observations that lipolytic activity in the stomach is due to enzymes secreted from the oro-pharynx (tongue). However, lipolytic activity in the stomach of these children strongly suggests the presence of a gastric lipase. (Supported by Grant NIH HD10823).

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GOAT'S MILK ACIDOSIS. Harold L. Harrison and Michael A. Linshaw (Spon. by C.T. Cho), Univ. Ks. Med. Ctr., Dept. of Pediatrics, Kansas City, Kansas.

Faced with a generation of parents reared in an era of food fads and special diets, physicians must remain alert for strange diets and their consequences on children of various ages. We recently evaluated a 3 week old infant with hyperchloremic metabolic acidosis and failure to thrive which was directly attributable to undiluted goat's milk feedings. This term infant presented with severe tachypnea. Serum electrolytes in mEq/l were Na 131 K 6, Cl 116, CO_2 4. Serum pH was 7.14, Hb 16.8 gm%. Serum values in mg/dl were BUN 30, Cr 0.4, Ca 10.6, PO_4 8.1. Urine pH was 5.0. Upon withdrawal of the goat's milk and after 12 hours of intravenous fluid therapy, the infant's clinical condition improved. By 48 hours, the serum electrolytes were normal. After 10 days of sustained weight gain on commercial formula, goat's milk was reintroduced. Within 18 hours, the baby lost weight and developed tachypnea and laboratory evidence of a compensated metabolic acidosis. Goat's milk was discontinued and his symptoms quickly resolved. Goat's milk is high in protein, potassium, chloride, phosphorus and calcium. On this milk, the infant's net acid excretion increased and was largely attributable to an increase in ammonium excretion. This response is similar to that seen in patients loaded with hydrochloric acid. The admission blood values reflect the composition of goat's milk. We suggest that undiluted goat's milk is an inappropriate food source for infants during the first month of life.

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THE EFFECT OF ACQUIRED POSTNATAL MALNUTRITION ON PANCREATIC ENZYMES IN THE RAT. T. Hatch, D. Branski, J. Krasner, E. Lebenthal. Divis'on of Gastroenterology, The Buffalo Children's Hosptial, SUNY at Buffalo, New York.

In order to characterize the response of the pancreas to malnutrition during the critical neonatal growth phase, acquired postnatal malnutrtion was induced in the rat by using the expanded litter. An experimental nursing litter of 16 rats and control litters of 7-8 rats were formed. At 19 days of age, pups were sacrificed, the pancreas resected, weighed and prepared. Mean pancreatic weight was decreased in malnourished rats to a greater extent (49% vs 60%) than the decrease in total body weight. Decreased organ weight was due mostly to a decrease in DNA content and in cell number, with a small but significant decrease in cell size.

	A	L	T	CH	CP-A	CP-B
Normal	*17±14	29±13	12±4	1±0.5	0.1±0.04	1±0.3
Mal	12± 7	15± 6	9±3	1±0.4	0.1±0.04	1±0.6

Enzyme activities expressed per total organ were all diminished; trypsin (T), carboxypeptidase A (CPA) and B (CPB), and amylase (A) to an intermediate extent; and chymotrypsin(CH), the least. Specific activities of the enzymes and total organ activities were decreased in a non-parallel fashion. Specific activities of lipase (L) and trypsin (T) were decreased ($p < .05$) lipase the most severely; the remaining enzymes were resistent to change. This can be explained by either a selective effect of malnutrition on a critical development period for lipase and trypsin, or that lipase, in general, is more vulnerable to insult.

(*Units ± S.D. = µmoles/mg protein/min.)