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LACK OF STIMULATION OF SUCRASE AND ISOMALTASE WITH FRUCTOSE IN CHILDREN WITH SUCROSE-ISOMALTOSE MALABSORPTION (S-IM). P.A. Krasilnikoff & H. Skovbjerg. Dept. of Paediatrics, Gentofte Hospital, and Dept. of Biochem. C, The Panum Inst., University of Copenhagen, Denmark.

In normal adults fructose stimulates the activity of sucrase and maltase. In a single patient with S-IM Greene et al.(1) found an increase in the sucrase activity after fructose ingestion (~ 40-60% of the caloric intake (c.i.)) and suggested dietary fructose as a form of therapy to these patients. In order to elucidate this possibility we have by methods earlier described (2) investigated the activity and rocket immunoelectrophoretic pattern of the dissaccharidasis in small intestinal biopsies from 2 children with S-IM (age 13 and 16 yrs) before and 2 weeks after a daily intake of an otherwise isocaloric diet containing 300 g of fructose (~50% of c.i.). Before the diet both children showed a sucrase activity below detection level (1 U/g prot.) and a residual isomaltase activity of 7,9 and 2,9 U/g prot., respectively. After the diet there was still no sucrase activity and the changes in the isomaltase level were not significant. The immunoelectrophoretic studies were in complete concordance with these findings. In conclusion even a very high dietary supplement with fructose can not initiate an increase of the missing enzymes in S-IM and represents no therapeutic alternative. (1) Greene, H L et al. Biochem Med 1972; 6: 409.(2) Skovbjerg, H, Krasilnikoff, P A. Pediatr. Res 1981; 15: 214.

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ACCELERATED APPEARANCE OF SUCRASE & MALTASE ACTIVITIES IN SMALL INTESTINES OF INFANT MICE INFECTED WITH ROTA VIRUS. J Collins, W G Starkey, M P Osborne, J Stephen, D C A Candy. Departments of Microbiology, Physiology & Paediatrics & Child Health, University of Birmingham, UK.

The specific activities of small intestinal brush border enzymes (lactase (L), sucrase (S), maltase (M)) were measured in pooled crypt and villous enterocytes from upper small intestines of infant mice during the course of rotavirus infection. Activities were assayed in 3 pools of enterocytes, each from 3 mice, at 72 and 96 h post infection. Infant mice were infected by oral challenge on the 7th day of life.

h post-infection:	72	96
L^+ infected:	1.62(0.42)***	2.94(0.78)
L^+ controls:	5.22(0.42)	5.16(0.24)
S^+ infected:	0.028(0.008)	0.368(0.109)**
S^+ controls:	0.052(0.025)	0.019(0.005)
M^+ infected:	3.12(0.12)***	6.42(0.00)***
M^+ controls:	1.38(0.30)	1.38(0.18)

*mean (SEM) ** P<0.05 ***P0.01 + µmol/h/mg protein

S and M activities rose at 96h post-infection; this was 5 days earlier than the expected rise in these enzyme activities. Thus rotavirus infection induced precocious maturation of S and M. The concept that villi become populated with crypt-like enterocytes is therefore less tenable in the infant mouse, since S and M are marker enzymes of mature enterocytes.

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OLIGOPEPTIDE UPTAKE INTO BRUSH BORDER VESICLES OF NEWBORN AND ADULT RAT INTESTINE. ITS DEPENDENCE ON HYDROLYSIS.

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Oligopeptide absorption in intestine is distinct of free amino acid transport and very effectively in developing animals. In the evaluation of absorptive function after birth we studied accumulation of dipeptide into brush border membranes which were incubated at various time with labelled gly-phe, gly-pro, and gly-sar. The hydrolysis rates were determined by the analysis of free amino acids with column chromatography after incubation.

Results: Time dependent uptake is increasing up to 1 min and does not exhibit high peak values after a few seconds. Uptake is slightly higher in newborns and closely related to rates of hydrolysis. Results indicate slow permeation of intact dipeptide through the brush border membrane of newborn and adult intestine and the importance of hydrolysis in the process of oligopeptide absorption.

Uptake (u) and hydrolysis (h) of brush border membranes

		u^+	h^+		u^+	h^+
gly-phe	*newborn*	*1.33++*	*4.0++*	*adult*	*0.95*	*7.75*
gly-pro		*1.05++*	*2.25++*		*0.45*	*5.4*
gly-sar		*0.15*	*0.1*		*0.25*	*0.55*

+(nM/mg prot/min), ++(significantly different to adults p ≤0.05)

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ORAL REHYDRATION SOLUTIONS - EXPERIMENTAL STUDIES OF NET WATER AND ELECTROLYTE ABSORPTION Cristobal FL, Sandhu BK, Burston D and Brueton MJ Dept Child Health Charing Cross & Westminster Med Sch London

Net water absorption from 5 oral rehydration solutions (ORS) of varying composition has been studied using an in vivo steady state perfusion technique in rat jejunum Tritiated water and phenol red were used as markers, each group involved 5 to 7 animals. The reference ORS used (A) was the World Health Organisation formula containing in mmol/l Na 90 and glucose 110. B and C were commercially available ORS containing in mmol/l B - Na 50, glucose 91 and sucrose 94 and C - Na 35 and glucose 200. D and E were similar to A with added aminoacids. D containing glycine and E glycine and glycyglycine.

The optimal net water absorption ±1SD was 7.83 ±12.2µl/min/g using A. This was significantly (P<0.001) greater than B 20.6±4.91; C 39.44±9.64; D 48.43±6.06 and E 49.07±9.95µl/min/g. The absorption using D or E was greater than using C (P<0.001) or B (P<0.001). Associated studies of electrolyte absorption and changes in unidirectional fluxes will also be presented. The influences of osmolality, pH and organic solutes will be discussed, with the implications for the management of diarrhoea.

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SUPPLEMENTATION OF EFA RICH POWDER WITH ARTIFICIAL BILE IN POSTOPERATIVE CBA: EFFECTS ON BLOOD EFA AND PGs. T.Shimizu, S.Oguchi, S.Ohyama, M.Sato, Y.Yamashiro, K.Yabuta. Dept. of Pediatrics, Juntendo University School of Medicine, Tokyo, Japan.

We reported that essential fatty acid(EFA) deficiency occurred in postoperative congenital biliary atresia(CBA) children could be corrected by supplementation of EFA rich powder through enterostomy in the patients whose total bile acid levels in the excreted bile were higher than critical miceller level. This study investigated the effects of EFA rich powder supplementation with artificial bile through enterostomy on blood EFA and PGs levels in 5 patients(7mos-18mos) with poor bile excretion less than critical miceller level. Linoleic acid(LA) and PGE_1 levels increased significantly ($p<0.05$) with daily infusion of the powder (38g LA with no AA/100g powder) for 1 month, dissolved in excreted own bile mixed with artificial bile made of 2mM/l taurocholate solution. However, arachidonic acid(AA) deficiency with derangement of PGE_2 and $PGF_{2\alpha}$ metabolism did not improved. These findings suggest that EFA rich powder supplementation with artificial bile is an effective treatment for LA deficiency in postoperative CBA children with poor bile excretion, but it is required to continue the regimen more than 1 month for correction of AA deficiency and derangement of PGE_2 and $PGF_{2\alpha}$ metabolism. And addition of AA to EFA rich powder should be considered in the further study.

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DEFICIENCY OF ESSENTIAL FATTY ACIDS IN PLASMA AND RED BLOOD CELL PHOSPHOLIPIDS AND VITAMIN E STATUS IN CHILDREN WITH LIVER DISEASE. P.Cheeseman, Y.Poovorawan, C.Stewart & A.P.Mowat Dept.Child Health King's College Hospital London, *Efamol Ltd,Guildford

To assess the incidence and severity of essential fatty acid (EFA) deficiency in chronic liver disease in children and its relationship to vitamin E status we have measured, by GLC, fatty acid(FA) composition of phospholipid in plasma and RBC , and plasma vitamin E (VE) by HPLC in 10 normal children and 57 with liver disease (22 biliary atresia (BA) 13 jaundiced, 11 biliary hypoplasia (BH) 9 jaundiced ,10 chronic active hepatitis (CAH), 14 miscellaneous liver diseases (MISC)). In CAH and in MISC both plasma VE and FA values in RBC were normal. In both BA and BH EFA were deficient especially in RBC with significant mean % decreases in (w-6) linoleic (BA,BH 14,21) , arachidonic (30,41), (w-3) linolenic (72,61), and its metabolites (53,44). There was a significant increase in saturated FA (25,29) and particularly palmitoleic acid (431,859). FA 20:3w9 , associated with severe deficiency was not detected . Similar but less marked changes occurred in plasma. In 27 VE deficient children the mean % total w-6 and w-3 FA of RBC phospholipid were significantly reduced (26, 50) when compared to 40 children with normal VE. Correction of VE deficiency by IM injections in 2 patients returned EFA to normal levels. We conclude that both VE and EFA deficiency are common in cholestatic disorders and that VE is important in maintaining the EFA content in phospholipid membranes as found in the RBC.