

It has been found that human milk contains several growth modulators. Epidermal growth factor (EGF) is a small polypeptide growth promoting factor for various cells. It is reported that human(h) EGF exists in human milk and it is considered to be one of the most important growth modulators in human milk. We measured hEGF levels of human milk, cow's milk and several formulas by newly developed sensitive RIA method (offered by Prof. N. Yanai, Shizuoka College of Pharmacy, Shizuoka, Japan). Human and cow's colostrums, transient milks and mature milks were obtained from healthy subjects, and they were centrifuged at 100,000 G and supernatant fluids were provided for assay. Results (1) hEGF in human colostrum, transient milk and mature milk were 196.7 ng/ml, 71.7, 39.5, respectively. (2) hEGF levels of cow's and formula milks were lower than minimal detectable dose of our method except one formula for premature baby containing about 1/250 as much as that of mature milk. Conclusions It seemed that hEGF in human milk directly acts on gastrointestinal mucosa and promotes its growth and maturation. The finding that hEGF level is the highest in colostrum suggests that hEGF is needed physiologically especially at early neonatal period.

Human milk contains glycoproteins, glycolipids and oligo-polysaccharides. The purpose of this study was to investigate the presence of some acid glycohydrolases which are involved in the degradation of such molecules. Alpha-fucosidase, alpha-mannosidase, beta-galactosidase, beta-glucuronidase and beta-N-acetylglucosaminidase were studied in colostrum and serum from 25 mothers using methylumbelliphenyl-glucoside derivatives as substrates. Results: a) human colostrum contained all the five glycohydrolases tested; b) after centrifugation each enzyme was present in both the supernatant and the cellular sediment; c) for each subject the enzymatic activities were higher in the supernatant of colostrum than in serum. Conclusions: 1) the presence of alpha-fucosidase, alpha-mannosidase, beta-galactosidase and beta-N-acetylglucosaminidase in human colostrum, which we have demonstrated, has not yet, to our knowledge, been described; 2) a process of passive diffusion of such enzymes from serum to the mammary gland seems to be unlikely; 3) acid hydrolases could be involved in the metabolism of complex carbohydrates molecules contained in human milk.

We have shown that 16, 16 dimethyl PGE₂ augments mucosal hyperplasia following massive small bowel resection in the rat. Likewise restricting dietary linoleic acid inhibits adaptation. PG may therefore be an important mediator of adaptation. We studied the effect of aspirin (ASA) inhibition of mucosal PG synthesis on mucosal adaptation following 70% proximal jejunoileal resection in 160g male Sprague-Dawley rats. Sixteen of 27 resected and 8 of 16 sham operated rats were given ASA 20mg/kg subcutaneously every 8 hours for 14 days following surgery; the remainder were given vehicle. Animals were fed a purified diet containing 5% linoleic acid ad lib. After 14 days, ex vivo mucosal PGE₂, PGF_{2α}, and thromboxane B₂ synthesis, and mucosal weight, protein, DNA, and maltase levels were determined. PG synthesis rates are shown below Mean ± SEM (ng/g tissue/min.).

	PGE ₂	PGF _{2α}	THROMBOXANE B ₂
Resected Aspirin.	1333 ± 80	635 ± 57	64 ± 4.9
Resected Control	281 ± 37	206 ± 62	27 ± 5.2

Despite marked inhibition of mucosal PG synthesis (p < .01), animals receiving ASA had comparable mucosal weight, protein, and maltase levels, when compared to controls. Endogenous PG may not be important in mucosal adaptation.

Abstract.

In human premature infants, a stimulated acid secretory response to a mixed meal had been demonstrated recently. Pepsinogen secretion in response to a meal in newborns particularly the prematures has not been investigated. To determine whether formula feeding could stimulate pepsinogen output, we evaluated the changes in pepsinogen levels in the stomach content of premature infants at mean gestational age of 34.5 weeks, at various time post prandially. Gastric aspirates collected from premature infants in a study carried out for evaluation of the rate of gastric emptying up to 100 minutes after a bolus formula feeding were assayed for pepsinogen. The formulas used were: a special premature formula (Similac PM 60;40) and another formula (Modified Similac PM 60;40). The pepsinogen activity in those premature is by 1/10 to 1/20 of the amount of pepsinogen recorded in older children. The basal pepsinogen in gastric aspirates was 0.1±0.05 (X±SD) U/Kg. Pepsinogen activity as measured in 13 premature infants increased 20 minutes after feeding with both formulas and remained higher than basal activity through the entire study period. Peak values were recorded after 60 minutes. These results demonstrate that in human premature infants a mixed meal led to an increase in pepsinogen activity in the stomach content, suggesting a response to a meal stimulus by the chief cells in the stomach of premature infants.

Superoxidase dismutase (SOD) is the main scavenger of superoxidase radicals in the mammalian body. The liver contains high levels of two SOD enzymes; Cu-Zn (SOD1) localized in cytosol and Mn SOD (SOD2) localized in mitochondria. The aim of the present study was to investigate the levels of SOD1 and SOD2 during development using enzymatic and immunological methodologies. Monospecific antibodies were prepared in rabbit against purified rat SOD1 and SOD2 polypeptides. Homogenates of fresh rat livers obtained from newborn, young, and adult rats were assessed for levels of SOD1 and SOD2 activity. It was found that the levels of SOD1 activity increased greater than ten-fold after birth, reaching adult levels after weaning. In contrast, the SOD2 level increased only four-fold after birth and reached essentially adult levels at weaning. SOD1 and SOD2 in newborn and young rat livers were not different in their molecular weight or charge from adult liver. Immunoblot quantitation indicated that changes in activity during ontogeny levels reflected the number of SOD1 and SOD2 molecules. These results show that SOD1 and SOD2 are differentially and independently regulated during ontogeny. It may be suggested that cytosolic and mitochondrial SOD may play different roles in superoxide scavenging during liver development.

The posttranslational processing of SI was investigated in organ culture of human intestinal biopsies labelled with ³⁵S-methionine and immunoprecipitated with monoclonal antibodies. SI is synthesized as a high mannose precursor molecule of Mr 210,000 which is slowly converted in the Golgi to the complex glycosylated form of Mr 245,000. The half-time for the conversion of the Mr 210,000 to the Mr 245,000 is about 2 h. Owing to the absence of pancreatic secretions in the culture medium, only pro-SI and neither of the subunits S or I can be identified by SDS-PAGE. Trypsin, when added to homogenates from biosynthetically labelled biopsies, cleaved pro-SI to its subunits. Elastase and chymotrypsin were not effective. Based on this finding the transit time of pro-SI from the Golgi to the brush border membrane was estimated. In the presence of trypsin, the appearance of S and I and the decrease in the labelling intensity of pro-SI was observed after 2 h of chase. This is indicative for a membrane insertion of pro-SI and its exposure to trypsin in the culture medium. The half-time for the conversion of pro-SI by the aid of trypsin to S and I was between 30 min and 1 h which corresponds to the transit post Golgi-time.