THE RELATION BETWEEN NUTRITIONAL STATUS, DIAGNOSIS AND COMPLICATIONS AT A WARD FOR GASTROINTESTINAL DISEASES AND INTERNAL MEDICINE DURING HOSPITAL STAY.
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Data concerning the correlation between malnutrition and complications during hospital stay are limited and mainly confined to surgical wards. The aim of the present study was to investigate the incidence of malnutrition and to correlate nutritional status with complications during hospital stay at a ward for Gastrointestinal Diseases and Internal Medicine.
Nutritional status was assessed by using the Subjective Global Assessment (SGA; a clinical score), the Nutritional Risk Index (NRI; 1.489*alb(g/L)+41.7*(actual/usual weight), NEJM 325:525) and the Nutritional Index (NI; 20.7-0,24*alb-19.68*pre-alb-1.86*total lymphocytes-0.04*%ideal weight, Clin Nutr 4:61) (values adapted to our hospital). A list of complications was composed and complications divided in severe and mild. The incidence of malnutrition (SGA, NRI, NI), complications, confounding variables and diagnoses were determined in 155 patients at a ward for Gastrointestinal Diseases and Internal Medicine during hospitalisation.
The prevalence of malnutrition in patients was 45% (SGA), 57% (NRI) and 60% (NI). The mean number of complications was higher in malnourished patients Odds Ratio 2.71 (SGA), 2.83 (NRI), 3.1 (NI) respectively. After correction for functional status, hospital stay, medicine use, recent surgery and diagnosis (using a logistic regression model) a correlation between nutritional status and complications was still seen, and was significant using the NI (2.4, CL 1.07-5.4).
Conclusion: in about 50 % of the patients at a non-surgical ward malnutrition was present. A correlation was seen between malnutrition and complications, even after correction for other factors like disease category. This study suggests an important role for nutritional intervention to reduce the incidence of complications during hospital stay.

● TAURODEOXYCHOLATE SHOWS DEVELOPMENT-RELATED DIFFERENCES IN ALTERING CHLORIDE PERMEABILITIES AND INTRACELLULAR CALCIUM IN RABBIT COLONOCYTES IN CULTURE. N. Neelakantam, J. Sahi, M. Reddy, G.N. Desai, D. Vidyasagar, and M.C. Rao. Dept. of Physiology & Biophysics and Dept. of Pediatrics, Univ. of Illinois, Chicago, IL.

Bile acids such as taurodeoxycholate (TDC) are potent stimulators of Cl- transport in the adult mammalian colon presumably by increasing intracellular $Ca^{2+}$($[Ca^{2+}]_i$). In contrast, TDC has no effect in whole tissue preparations (muscle + mucosa) of the neonatal colon. To determine the cellular basis of these differences, the effects of TDC on Cl- transport and on $[Ca^{2+}]_i$ in epithelial cells isolated from the distal colon of newborn (7-9 days), weanling (25-28 days) and adult (6 months) rabbits and maintained in primary culture for 24 hrs were examined. Cl- transport was assessed in colonocytes in suspension and in those attached to a Collagen IV matrix. Cl- transport was measured using the fluorescent probe 6-methoxyquinyl acetoethyl ester (MQAE), and calculated as influx in mM/sec/$10^5$ cells. In both attached cells and cells in suspension, TDC caused a dose-dependent increase in Cl- permeability (0.48±0.1 to 1.96± 0.13) which was partially inhibited by the Cl- transport inhibitors, diphenylamine 2-carboxylate (50$\mu$M) + furosemide (10$\mu$M) (1.1 ±0.3). However, TDC (25$\mu$M - 1mM), did not alter Cl- permeability in either weanling or neonatal colonocytes. The lack of responsiveness in the weanling and neonate is not due to the absence of secretagogue-stimulated Cl- transport since the cAMP-stimulator forskolin (1$\mu$M) caused a 2-3 fold increase in Cl- permeability in all three groups. To determine if the effects of TDC could be related to its actions on $[Ca^{2+}]_i$, the effects of TDC and the $Ca^{2+}$ ionophore, ionomycin (1$\mu$M), on $[Ca^{2+}]_i$ were measured in cells in suspension using fura-2 (Fluorescence units: 340/380 nm ratio). TDC significantly increased $[Ca^{2+}]_i$ in adult (1.45±0.2 to 1.9±0.2, n=4) but not in neonatal or weanling colonocytes. In contrast, ionomycin increased $[Ca^{2+}]_i$ in adult (1.5±0.2 to 2.1±0.3) and weanling (1.6±0.8 to 2.6±0.3), but not in newborn colonocytes. Ionomycin also increased Cl- permeabilities in adult and weanling colonocytes(≈2-fold) but had equivocal effects on the neonatal colonocytes, stimulating in some and having no effect in others. In summary, these results demonstrate that rabbit colonocytes transport Cl- in response to TDC only in the adult. This may in part be due to the inability of TDC to alter $[Ca^{2+}]_i$ in the neonate and weanling. The $[Ca^{2+}]_i$ signalling mechanism may be immature in the neonatal cells, since they failed to respond consistently to ionomycin. However, factors other than $[Ca^{2+}]_i$ must be playing a role in the TDC response mechanism since the weanling was non-responsive to TDC but was presponsive to ionomycin. Thus, the regulation of colonic ion transport shows ontogenic changes in the $Ca^{2+}$-signalling cascade.

● **THE MAJOR CAUSE OF WEIGHT LOSS IN PATIENTS WITH HIV INFECTION IS INADEQUATE CALORIC INTAKE.** E.A. Neal, J. Koch, C. Kriegsman, M. Scott, J.P. Cello, and D.C. Rockey. Division of Gastroenterology, San Francisco General Hospital, Department of Medicine, University of California, San Francisco.

Weight loss and malnutrition are nearly universal among AIDS patients. The pathogenesis is thought to be multifactorial and can include decreased caloric intake, malabsorption, and/or metabolic dysregulation. Identifying the factor(s) leading to weight loss can guide the practitioner to rational intervention. Our aim was to determine the incidence and investigate the etiology of weight loss in a group of ambulatory HIV/AIDS patients. **Methods:** We retrospectively reviewed 100 consecutive HIV positive patients referred (self or primary provider) to our outpatient nutrition clinic. Nutritional assessment included weight history, a review of symptoms, dietary analysis of 3-day diet records, anthropometrics (including midarm muscle circumference (MAMC) and triceps skin fold (TSF)), and serum albumin. Caloric requirements were estimated using the Harris Benedict equation with standard adjustments. **Results:** Seventy-three patients (73%) with greater than 5% unintentional weight loss were identified. All reported results are for those patients in this group. The mean CD4 count was 111 ± 130.8 (SD) per $mm^3$ and the mean number of prior opportunistic infections was 1.4 (range 0-4). Weight at presentation of the cohort was 89.5 ± 8.5% of UBW. Weight loss occurred over 10 ± 5.5 months (SD). The mean serum albumin was 3.5 g/dL and MAMC/TSF were less than the 20th percentile, respectively. Fifty-nine percent (40/73) of patients consumed <90% of their estimated caloric needs. Forty-two percent of patients with weight loss submitted stool samples to assess malabsorption after consuming a high fat diet (>100 grams fat /24 hours). Nineteen of 31 patients (61%) had qualitative or quantitative fat malabsorption. Overall, 53 of 73 (73%) patients presenting with >5% unintentional weight loss had evidence of inadequate caloric intake, malabsorption, or both. **Conclusion:** In this series of HIV positive patients, weight loss was common. In the majority of those with weight loss, basic nutrient requirements were not met, due to either inadequate intake, malabsorption, or both. In a minority of patients with weight loss, caloric intake was adequate and fat malabsorption was not demonstrable. **Implication:** The etiology of weight loss in the majority of HIV/AIDS patients is due to undernutrition. The relatively well preserved serum albumin levels and low adipose stores seen in this group are consistent with simple starvation rather than cachexia (or metabolic dysregulation). In those without documented malabsorption and adequate caloric intake, the etiology of weight loss may have been metabolic dysregulation, undetected fat malabsorption, or both. Once weight loss is attributed to inadequate caloric intake, strategies to correct simple starvation should include a combination of nutritional counseling, possible appetite stimulants, and/or enteral nutritional support. When caloric intake is adequate and malabsorption is excluded, therapies such as anabolic steroids and cytokine modulators may be considered.

● **Cloning of a human intestinal folate carrier cDNA, functional expression of its cRNA in *Xenopus* oocytes and determination of the distribution of homologous RNA at the tissue and cellular levels.** Toai T. Nguyen, David L. Dyer, Daniel D. Dunning, Stanley A. Rubin, and Hamid M. Said. V.A Medical Center, Long Beach, CA 90822; and California College of Medicine, University of California, Irvine, CA 92717.

The mechanism of intestinal folate transport has been the subject of intensive investigation. However, very little is known about the molecular identity of the transport system(s) involved, especially in humans. In this study, we screened a human small intestinal cDNA library (Clontech) using as probe a mouse small intestinal folate carrier cDNA clone isolated in our laboratory (Said *et al.*, BBA, in press), and identified a partial clone. Using a modified Marathon™ cDNA amplification protocol (Clontech), we isolated a full-length clone, *hIFC1*. This cDNA consists of 2,643 base pairs with an open reading frame coding for a putative polypeptide of 591 amino acids with a predicted $M_r$ = 64,826, pI = 9.4, 13 transmembrane domains, three protein kinase-C phosphorylation sites, and one N-glycosylation site. The polypeptide is predicted to carry a net positive charge of 14.1 at physiological pH, which may be important for its interaction with the negatively charged substrate. This *hIFC1* clone shares 74 % and 66 % homologies at the DNA and amino acid sequence levels, respectively, with the mouse small intestinal folate carrier cDNA probe. The functional identity of the *hIFC1* clone was established by expression in *Xenopus* oocytes. A 8-fold increase in folate uptake was observed in oocytes micro-injected with 50 ng *hIFC1* cRNA compared to water-injected controls. The distribution of poly(A)$^+$ RNA species complementary to *hIFC1* in different human tissues was examined by Northern blot analysis. We observed a predominant band at 3.3 kb (in decreasing intensities) in placenta > small intestine, colon, thymus, prostrate, testes > ovaries, spleen, peripheral blood leukocytes > heart, brain, liver, skeletal muscle, pancreas > kidney, lung. We also observed two minor bands at 4.5 and 5.9 kb, which could be *hIFC1* precursor mRNA or other isoform(s) mRNA, in all samples, except with further reduced intensities in spleen, liver, pancreas, kidney, and lung, suggesting a possible tissue specific differential expression of those isoforms. *In situ* hybridization using anti-sense and sense *hIFC1* probes on sections of human jejunum showed specific preferential *hIFC1* expression in the epithelial cells in comparison to surrounding connective tissue, strongly associating *hIFC1* with intestinal epithelial function.