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THE METABOLISM OF LEAD-203 (^{203}Pb) IN THYROPARATHYROIDECTOMIZED (TPTX) RATS AFTER INFUSIONS WITH CALCIUM (Ca) AND ZINC (Zn). John F. Rosen, Albert Einstein Coll. Med., Montefiore Hosp. & Med. Ctr., Dept. Ped., New York.

Previous data in children and animals have shown that dietary deficiencies of Ca and Zn increase the intestinal absorption of Pb thereby increasing Pb toxicity. To define further the interactions of Pb, Ca and Zn, TPTX rats were given IV 7 mg/kg of Pb + 25 μCi ^{203}Pb and placed in metabolism cages. 4 days later Ca (.232mM) or Zn (.015mM) were infused via a catheterized tail vein for 6H. Measurements were made of stable Pb and ^{203}Pb from aliquots of total organs, urine and blood, once rats were sacrificed from 6-72H after infusion. The results (* = p<.01) were expressed as cpm ratios of Treated (T)/saline-infused Controls (C). The T/C ratios were:

	Hrs.	URINE	KIDNEY	LIVER	BONE	BRAIN
Ca-Treated	6	27.7±2.1 *	.65±.04*	.43±.05*	.54±.09*	.57±.10*
	72	1.50±.4	1.47±.09*	1.12±.14	1.04±.09	.85±.10
Zn-Treated	6	.38±.08*	.39±.10*	.42±.08*	.59±.10*	.54±.04*
	72	1.17±.06	1.17±.06	1.49±.11*	1.19±.06	.92±.10

Though soft tissue displacement of ^{203}Pb was produced by Ca and Zn, the majority of ^{203}Pb released came from bone with subsequent repletion of soft and hard tissue at 72H.

These data indicate that interactions between Pb, Ca and Zn occur at several tissue sites besides the intestine. These complex interactions likely involve metal ion radii, and the activation of Ca- and Zn- sensitive ATPases and alkaline phosphatases, respectively.

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FAILURE OF EARLY ADMINISTRATION OF 1.25 DIHYDROXYCHOLECALCIFEROL(1.25OHD3) TO PREVENT EARLY NEONATAL HYPOCALCEMIA IN PREMATURE INFANTS(PI). Bernard L. Salle, Louis David, Jacques Senterre, Hubert Renaud, Roland Revelin. Neonatal Dept. Hop. Edouard Herriot, Lyon, France. (Intr. by P.R.Swyer).

From 3 to 120h of age, 3 matched groups of 6 PI (gestational age 32-36w) received a daily oral dosage of respectively 30 mcg of vitD3 (D3G), 10mcg of 25OHD3(20OHD3G) and 0.5mcg of 1.25OHD3 (1.25OHD3G). In all PI blood samples were drawn at 1-2,24,48,120 and 168; determinations of calcium(Ca), phosphorus(P), and immunoreactive parathormone(iPTH) and calcitonin(iCT) were performed on a total volume of 400μl of serum using sensitive micromethods. All PI were infused with 10% glucose from 3h and fed with human milk from 12h of age. There was no difference in serum Ca,P,iPTH and iCT mean±SD levels between the 3 groups at any period of time. Serum Ca(mg/100ml) decreased to nadir values at 48h(D3G:5.7±1.2; 25OHD3G:6.8±0.9; 1.25OHD3G:6.7±1.1). A progressive increase in serum Ca towards normal values was seen at 120 and 168h in the 3 groups. Serum iPTH(μlEq/ml) followed an opposite pattern with peak values at 48h(D3G:231±137; 25OHD3G:281±138; 1.25OHD3G:211± 149-N in children:63±18). Serum iCT(pg/ml) reached peak values at 24h(D3G:457±186; 25OHD3G:415±121; 1.25OHD3G:443±183-N in children:non detectable:<150pg/ml). These data indicate that early administration of oral 25OHD3(10mcg/day) or 1.25 OHD3(0.5 mcg/day):1/does not prevent the early neonatal hypocalcemia of PI-2/ does not shorten the time of normalisation of the calcemia -3/ does not modify the pattern of serum iPTH and iCT changes that we previously described in PI during the neonatal period.

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ETIOLOGY OF FIRST DAY HYPOCALCEMIA (FDH): ROLE OF PTH AND CALCITONIN? Heinrich Schedewie, D.A. Fisher, W.D. Odell, L.J. Deftos, M.J. Elders, T.L. Cantor, M. Dodge. UAMS/NCTR, Little Rock; Harbor General Hospital-UCLA, Torrance; VA Hospital-UCSD, LaJolla; Departments of Pediatrics and Medicine.

To determine the potential role of endocrine factors in the pathogenesis of FDH, we have measured plasma PTH, calcitonin (CT) and calcium (Ca) concentrations (conc.) in 38 infants with documented hypocalcemia (Ca ≤7.5 mg/dl) and in known high risk groups of FDH including premature infants (n=51), asphyxiated newborns (25), and infants of diabetic mothers (IDM, n=22). Sequential blood specimens were drawn during the first week of life. PTH and CT were determined by a sensitive double-antibody RIA. Ca was measured by atomic absorption. Mean CT conc. in premature and asphyxiated infants during the first 2 days of life were increased nearly 10-fold above the mean of schoolchildren or adult controls and they were significantly ($p < .01$) higher than CT conc. observed in healthy term infants. The highest CT conc. were determined in the group of hypocalcemic newborns (mean Ca ± SE:6.78 ± 0.1 mg/dl). PTH conc. were depressed and showed a delayed surge in the group of IDM and premature infants. However, in asphyxiated newborns hypocalcemia developed in spite of elevated PTH levels. Conclusion: Hypersecretion of CT may be an important cause of hypocalcemia during early postnatal life. Concomitant hypoparathyroidism may further augment the effect of hypercalcitoninemia.

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HUMAN PLASMA POLYAMINE-CONJUGATED PEPTIDE, PUTRESCININ Thomas W. Seale, Wai-Yee Chan, Jayesh B. Shukla, Owen M. Rennert. University of Oklahoma, Health Sciences Center, Department of Pediatrics and Department of Biochemistry and Molecular Biology, Oklahoma City, Oklahoma.

To define a cystic fibrosis factor and its potential relationship to polyamine metabolism an investigation of the low molecular weight peptide profile of human blood was undertaken. Previous investigations indicated polyamines were detectable in serum and plasma following extensive alkali or acid hydrolysis. We report the existance of a polyamine peptide conjugate in human plasma. This peptide was isolated by fractionation of citrated whole human plasma following chromatography on Biogel P10. The elution profile, monitored at 220nm, resulted in the identification of 6 peaks. These polypeptides had a molecular weight range of 2 to 10,000 daltons as evidence by retention on Diaflomembranes and gel chromatography. The major peak (putrescinin) was purified by Biogel P6 chromatography, paper chromatography and DEAE celulose chromatography. This plasma peak was present in CF patients and controls, and disappeared from blood following coagulation. The molecular weight of putrescinin was 4600 daltons. Amino acid analysis revealed 43 amino acid residues with absence of methionine, cysteine and abundant quantitites of glycine, glutamate asparate, serine and alanine. One mole of ornithine was detected per mole of putrescinin. Putrescine and spermidine was covalently bound to putrescinin; 3-5 moles of putrescine and 0.3-0.5 moles of spermidine/mole peptide. The conjugated polyamine content was 10-30 times that of free plasma polyamine. The significance of this peptide in coagulation and to CF will be discussed.

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URINARY METHYLCITRATE AS DISTINGUISHING FEATURE IN NEONATAL PROPIONYL-CoA CARBOXYLASE (PCC) DEFICIENCY. Stephen J. Sepe, Catherine E. Costello, John T.Herrin Harvey L. Levy and Barry Wolf. Harvard Med.Sch., Mass. Gen.Hosp. Neuro. and Child Services, Mass. Inst. of Tech., Boston, and Yale Univ. Sch. Med., New Haven.

PCC deficiency has usually been distinguished by markedly increased concentrations of propionic acid (PA) in blood. These increases have been particularly striking in neonates who are ill as a result of PCC deficiency. With one exception such neonates in our experience have had elevations of serum PA ranging from 150-400 uM, levels that are 100 fold greater than normal (2.32±0.32) for this age. The exception was a female infant who died at 4 weeks of age after a course characterized by lethargy, weight loss, metabolic acidosis, hyperammonemia and hyperglycinemia. Several gas chromatographic (GC) analyses of serum both before and after hyperalimentation revealed only relatively mild increases of PA. Urinary methylcitrate (MC), however, was found to be markedly increased by GC-mass spectrometry. Intravenous biotin was ineffective in reducing the accumulations of either MC or PA. Enzyme studies of fibroblasts grown from skin obtained postmortem revealed a specific deficiency of PCC (11 picomoles/min/mg protein; normal 863±102) and normal β-methylcrotonyl-CoA carboxylase activity (346; normal 283±35).

In PCC deficiency, MC, a condensation product of propionyl-CoA and oxalacetate, may accumulate at the expense of propionyl-CoA. Such individuals would have relatively small increases in PA and the PCC deficiency might be unrecognized unless analysis for urinary MC is performed.

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DIFFERING FOOD COMPOSITION FOR NOCTURNAL INTRAGASTRIC THERAPY IN TYPES I AND III GLYCOGEN STORAGE DISEASE (GSD). Alfred E. Slonim, Annie B. Terry, Roberto Moran, Paul Benke, Harry L. Greene, Ian M. Burr, Vanderbilt Univ. School of Medicine, Vanderbilt Hospital, Dept. of Pediatrics, Nashville, TN and Univ. of Miami, Dept. of Pediatrics, Miami, FL.

GSD-I and GSD-III, although clinically similar, are metabolically quite different and so may demand different nutritional therapeutic approaches. To test this, some of the metabolic responses of patients with GSD-I and GSD-III were measured in response to meals of glucose, beef and glucose plus beef. Glucose ingestion in GSD-I led to a fall in plasma glucagon, lactate, alanine and valine, whereas in GSD-III, although glucagon fell, a discordant rise in lactate, alanine and valine took place. Beef ingestion in GSD-I led to a rapid fall in glucose and insulin and a sharp rise in glucagon, alanine and valine, while in GSD-III, there was a considerable rise in glucose, insulin and glucagon and only a modest and transient rise in alanine and valine. These results suggest that enhanced gluconeogenesis is detrimental in GSD-I, whereas in GSD-III, it is beneficial and essential. This motivated us to treat GSD-I with nocturnal intragastric therapy (NIG) of high carbohydrate content and GSD-III with NIG therapy of high protein content. Both types of patient showed marked clinical and metabolic response to their respective therapies.