1093 INCIDENCE OF INFECTION IN RELATION TO DURATION OF RUPTURE OF MEMBRANES (ROM) IN PREMATURE INFANTS. J.J. Yoon, A.E. Esquea, J.A., Rao and M.N. Wijesinghe, Albert Einstein Coll, of Med., Bronx-Lebanon Hosp. Ctr., Dept. of Ped. and Obst., Bronx, NY (Sponsored by M. Cohen). From July 1976 to Dec. 1979, 300 premature infants, weighing between 1000 and 2300 gm were studied for the incidence of infection in relation to duration of ROM. They were divided into 6 groups of infants with 0-15/60, 16/60-23, 24-71 and greater than 72 hours of ROM. There were 82, 127, 51 and 40 infants in each group respectively. The incidence of proven in-fection was 8% (24) out of which 5% (15) had positive blood culture, 3% (4) had meningitis and 3.3% (10) had urinary tract infection. The incidence of clinical sepsis was 33.3% (100). The incidence of cultuce, 3% (4) had meningitis and 3.3% (10), The incidence of culture, 3% (4) had meningitis and 3.3% (10). The incidence of culture in-sepsis was not related to the duration of ROM. The recence of antibiotic therapy was significantly related to the duration of ROM particularly in in-fants more than 32 weeks and 1500 gm. When they were small and extreme-by immature, 2/3 of them were on antibiotic therapy and it was not related to the duration of ROM. As the duration of ROM increases, the incidence of amionitis and maternal antibiotic therapy increased only when the in-fants were greater than 32 weeks and 1500 gm. There were no correlations between the duration of ROM and the incidence of meconium stained am-nionitic fluid, fetal distress and severe perinatal asphysia. Infants 32 weeks we here duration of ROM and the incidence of meconium stained am-niotic fluid, fetal distress and severe perinatal asphysia. Infants 32 weeks notic fluid, fetal distress and severe perinatal asphysia. Infants 32 weeks or less with greater than 72 hours of ROM had the lowest incidence of severe perinatal asphysia. The mortality rate was not related to the dura-tion of ROM. However, when there was no associated proven infection, significantly higher mortality was noted in infants with greater than 24 hrs. of ROM which was often associated with severe perinatal asphyxia. This study revealed that the duration of ROM did not seem to effect the inci-dence of infection, fetal distress, perinatal asphyxia and mortality in premature infants.

CELLULAR IMMUNE RESPONSES IN FAMILIES WITH INFLUENZA R. John M. Zahradnik, Arthur L. Frank & Larry H. Taber, (Spon. by W.P. Glezen). Baylor College of Medicine, 1094 Departments of Microbiology & Pediatrics, Houston.

Cellular and humoral immunologic responses were measured in 39 families, 109 members (72 children) following the 1980 influenza B epidemic. Virologic and/or serologic evidence of recent infection was detected in 19 families (36 children and 17 adults) with information on infection in 1977 available for most. Ficollhypaque separated peripheral blood lymphocytes were obtained on all 109 family members 2-12 weeks post-infection and lymphocyte transformation (LT) studies performed with influenza B antigen. LT responses were seen in 28/36 children and 11/17 adults with recent infection; 9/21 children and 6/22 adults with evidence of remote infection. Thirty-six children (1-5 y.o) have been followed since birth; for 22, this marked their primary infection with influenza B. Significant LT response was seen in 16/22 (3) non-transformers also lacked an antibody response). In 3/16, LT responses were present 4-8 weeks post-infection in the absence of neutralizing antibody (NT). LT was not seen in 15/16 uninfected children. There was a tendency for a linear correlation be-tween LTresponse and post-influenza NT antibody titer in adults, but no such correlation in children to either pre- or post-infection NT antibody titer.

This study demonstrates that primary influenza B infection in young children elicits cellular immune responses equivalent to that of adults. LT responses is higher in recent (73%) as com-pared to remote (35%) infections and there was no correlation with the NT antibody titer achieved.

METABOLISM

VITAMIN D DEFICIENCY RICKETS WITH ABNORMAL LIVER 1095 FUNCTION TESTS AND MYOPATHY AS THE PRESENTING MANIFES-TATIONS OF CYSTIC FIBROSIS. <u>Edward H. Abraham</u>, Jeffrey S. Gerdes, Robert G. Castille, Harry S. Shwachman, <u>Robert H. Wilkinson</u>, <u>Constantine S. Anast</u>. Harvard Medical School, Children's Hospital Medical Center, Boston, Mass.

Abnormalities in mineral and bone metabolism have been reported in Cystic Fibrosis (CF), but overt rickets as a presentation of CF is extremely rare. The role of vitamin D in CF has recently received attention. The deficiencies of fat soluble vitamins associated with CF may be accounted for by impairment of both intestinal absorption and liver function. Rickets is herein reported in a poorly developed 9-month old girl less than the 3rd %ile for weight with a history of large frequent foul stools receiving 400 IU of vitamin D daily. She presented with hypocal-cemia (Ca4.7mg%), hypophosphatemia (PO₁ 2.8), elevated alkaline phosphatase (657 mU/ml), depressed serum 25(0H)vitamin D (6.6ng/ml) elevated serum PTH, and clinical and radiological evidence of rickets. Although there was no family history of CF, studies rickets. Although there was no family history of CF, studies revealed an elevated sweat chloride and pancreatic exocrine in-sufficiency consistent with CF. The patient also had abnormal liver function tests and a distal myopathy. Treatment was init-iated with 1,25(OH) vitamin D and came to include a standard vitamin D preparation and pancreatic enzymes. This led to full recovery of the rachitic process with normalization of circulating 25(OH) vitamin D (35.1 ng/ml) and PTH with concurrent resolution of the liver function abnormalities and the myopathy. The resolu-tion of the myopathy with vitamin D therapy strongly suggests that it was a reflection of the vitamin D deficiency state.

1096 TRANSPORT OF LIPOPROTEIN CHOLESTEROL BY THE OVARY IN VIVO. John M. Andersen. (Spon. by Charles R.Rosenfeld) University of Texas Health Science Center, Department of Pediatrics, Dallas.

High density lipoproteins (HDL) regulate cholesterol synthesis in the ovary of the rat. This study was done to characterize the cholesterol transport process responsible for the regulatory events. Rats were treated with 4-aminopyrazolopyrimidine to de-crease the plasma cholesterol level from 56 to 4 mg/dl and de-crease the ovarian cholesterol content from 7.70 to 4.20 mg/g. One h prior to the transport studies the rats were given aminoglutethimide. Specific lipoproteins were infused to maintain a steady-state plasma cholesterol level. The uptake of both HDL-cholesterol (HDL-C) and low density lipoprotein-cholesterol(LDL-C) was linear with respect to time for 4 h. The uptake of HDL-C and LDL-C was concentration dependent and demonstrated high affinity, LDL-C was concentration dependent and demonstrated nigh attinity, saturable processes with Km values of 72 and 68 mg/dl and Vmax values of 0.97 and 0.74 mg/g/h for HDL-C and LDL-C respectively. hCG administration decreased the Km for HDL-C to 22 mg/dl, but did not alter the HDL-C Vmax or any of the transport parameters for LDL-C. The maintenance of normal plasma LDL levels for 24 h prior to the transport study completely blocked LDL-C uptake, but did not diministration HDL C transport compandia pre-infusion studies did not diminish HDL-C transport. Comparable pre-infusion studies using HDL did not alter LDL-C or HDL-C uptake. These studies show that LDL-C and HDL-C are taken up by the ovary by separate pro-cesses with different characteristics. The results suggest that HDL-C very likely provides much of the cholesterol substrate for steroid hormone synthesis in the ovary.

1097 DILEMMAS OF NEWBORN SCREENING FOR HYPERLIPOPROTEINEMIA Billy F. Andrews, Philip Kalayil, and Edith Dunlap University of Louisville School of Medicine, University Hospital, Department of Pediatrics, Louisville, Kentucky

The incidence of congenital hyperlipidemia and early detection for institution of dietary and/or drug therapy is most desired by a society beset with major related health problems. Between January 30, 1975 to June 30, 1980, 12510 infants were studied with lipid screening (cholesterol, triglyceride, and lipoprotein electrophoresis). Repeated specimens and careful family histor-ies were taken on infants with abnormal values. 51 infants had hyperlipidemia (0.4 per cent). One infant had glycogen storage disease. 71 per cent of the families had a history of hypertension and/or heart disease and 43 per cent, diabetes. 84 per cent of the hyperlipoproteinemic children placed on dietary therapy responded with lower triglyceride and cholesterol values and improved lipoprotein electrophoretic patterns. Diagnosis of hyperlipoproteinemia in the newborn is expensive and requires repeated testing and family history for confirmation. Dietary therapy is effective in lowering lipid values in the majority of children. Immediate family members, e.g., parents, uncles, aunts, and grandparents have major medical problems. Strong family history of medically associated diseases should dictate future screening in infancy. Although blood lipids are effectively lowered by diet, large controlled studies are necessary to prove preventive aspects.

SYNERGISTIC EFFECT OF CALCIFEROL AND PARATHYROID HORMONE ON CALCIUM ABSORPTION IN NEWBORN RATS (NBR). W. H. Bergstrom and B. Goldstein, Dept. of Pediatrics, SUNY, Upstate Medical Center, Syracuse, NY. Serum calcium (CaS) in NBR was 9.0±.05 mg/dl at spontaneous or Caesarean delivery; maternal CaS was 8.7±.08. At 20 hr, CaS was 7.6±.13 in nursing NBR and 7.1±.07 in NBR gavaged with calciferol-free simulated rat milk(F). When F was supplemented with ergo-calciferol (D2, 41U/rat=8001U/Kg), CaS at 20 hr was 7.0±.05. When F contained 1-25(0H)2D1 (10 ng/rat=2ug/Kg), CaS at 20 hr was calciferol (D2, 41U/rat=8001U/Kg), CaS at 20 hr was /.01.05. When F contained 1-25(0H)2D3 (10 ng/rat=2µg/Kg), CaS at 20 hr was 8.1±.06. Ca-free F gave a 20 hr CaS of 6.9±.05; with 1-25(0H)2D3 CaS was 7.0±.08, indicating that enhancement of CaS by 1-25(0H)2D3 was due to Intestinal absorption rather than bone Ca mobilization. When F contained no D2, parathyroid extract (PTE, .05 ml
1M) at delivery had no effect on CaS at 20 hr (7.0±.08). On D2-supplemented F with PTE at delivery, 20-hr CaS was 7.4±.10. Absorption of Ca was increased by 1-25(0H)2D3 but not by D2.

CaS increased when both D2 and PTE were given, suggesting that hypoparathyroidism rather than insufficient renal hydroxylase or unresponsive mucosa caused the lack of response to D2 alone. The feto-maternal CaS gradient indicates a placental pump which

may account for neonatal hypoparathyroldism on the basis of intra-uterine suppression, as has been inferred for human subjects. Insofar as these results apply to human newborns, they suggest that the effectiveness of 1-25(0H)2D3 in the amelioration of

neonatal hypocalcemia derives from functional hypoparathyroidism rather than from renal or intestinal unresponsiveness.