ABNORMAL LUNG FUNCTION 8 YEARS AFTER VENTILATION FOR HYALINE MEMBRANE DISEASE (HMD) WITHOUT BRONCHOPULMONARY DYSPLASIA (BPD). Ernst Schoenmann, Beverly Meyer, Richard Kraemer, Emilio Bossi, Department of Pediatrics, University of Berne, Switzerland.

Ventilation for HMD can lead to BPD resulting in long lasting functional lung impairment. Whether this also occurs when BPD does not develop, is not known. Therefore, we tested lung function in 18 children (median age 7 7/12 years) who had been ventilated for HMD without BPD, in 20 non-ventilated siblings (9 10/12) and in 20 controls matched for gestational age, birth weight, and sex (6 2/12). **Method:** Lung function was measured by whole-body plethysmography. Airway reactivity was assessed by carbachol provocation. Abnormal lung function was defined when hyperinflation ($TGV > 130\%$ predicted) and/or small airway obstruction (maximal expiratory flow at 50 % VC $< 80\%$) was present. **Results:** 10/18 ventilated children, 1/20 siblings ($p < 0.001$) and 3/20 matched controls (p vs. ventilated < 0.01) have abnormal lung function. The occurrence of airway hyperreactivity was not different between the groups (5/18, 4/20 and 6/20). **Conclusion:** Ventilation for HMD not resulting in BPD is associated with hyperinflation and/or collapse of the small bronchioli at a median age of 7 7/12 years. The abnormal lung function is probably not due to genetic predisposition (sig. less abnormal lung function in siblings, no difference between siblings and controls, no difference in airway hyperreactivity) nor to prematurity itself (sig. less abnormalities in gestational age- and birth weight-matched controls).

ALPHA-1-PROTEINASE INHIBITOR (Pi) PHENOTYPES IN CHILDREN WITH URINARY TRACT MALFORMATIONS Vytautas Basys, Petras Kaltenis, Donatas Stakisaitis, Pediatric Department of Vilnius University, 232600 MTP, Vilnius, Lithuania

Pi phenotypes associated with decreased serum level of this protein are known to be more prevalent in subjects with a variety of diseases. The aim of this study was to reveal a possible linkage between deficient Pi phenotypes and urinary tract malformations. 374 pediatric patients were examined, including 72 with duplicated system, 97 with hydronephrosis, 23 with renal hypoplasia or aplasia, 179 with vesicoureteral reflux (VUR) and 3 with polycystic kidney disease. Pi typing was done by isoelectric focusing. Control group consisted of 1442 subjects examined in a population study. An increased frequency of heterozygous phenotypes PiMZ, PiMS and PiMF was found in children with VUR grade 1 and 2 (PiMZ in 8.8%, PiMS in 5.2%, PiMF in 2.6% of patients comparing with 3.5%, 3.3% and 0.4%, respectively, in the controls). These findings might suggest a certain role of moderately decreased antiproteinase activity in the appearance of low grade VUR (possibly via chronic inflammatory process).

MATERNAL METOCLOPRAMIDE TREATMENT AND PROLACTIN CONCENTRATION IN HUMAN MILK.

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The breast milk prolactin (PRL) has been claimed to play a role in the control of electrolyte composition of the milk. Since metoclopramide has been shown to increase milk production in humans, we have made an attempt to investigate the production, the PRL and sodium concentrations in milk with and without maternal metoclopramide treatment (5 days, 30 mg/day). Both groups consisted of 11 mothers and their full-term newborn infants. The daily milk production was significantly higher in the treated group (276.4 ± 36.6 vs 150.9 ± 25.3 ml/day, $p < 0.01$). The PRL measured by RIA was similar in the milk samples of the metoclopramide treated and control groups (80.5 ± 17.7 vs 90.7 ± 27.3 ng/ml). The sodium concentration in the milk of mothers taking metoclopramide was 22.1 ± 1.6 mmol/l and 24.3 ± 3.2 mmol/l in the control group ($p = 0.59$). On the 5th postnatal day the plasma PRL of the newborns of mothers treated with metoclopramide does not differ from the value of the control babies (29.8 ± 2.6 vs 30.7 ± 2.4 ng/ml) indicating that the amount of metoclopramide transferred into the milk has no apparent influence on the hypothalamo-hypophyseal axis of the neonate. In conclusion: the maternal metoclopramide treatment augments the milk production without having any effect on the PRL and sodium concentration of human "mature" milk.

INTERFERON PRODUCTION IN NEWBORNS: THE EFFECT OF DIPYRIDAMOLE. Aldona Rainytė-Audinienė, Vytautas Basys, Liudmila Prijmiagi, Irina Kremerman, Pranas Simulius, Department of Pediatrics, Vilnius University, Vilnius, Lithuanian Republic.

The ability of dipyridamole to stimulate interferon production in adults is known since 1978. The aim of our investigation was to reveal the existence of such ability in newborns. 43 newborns with both uncomplicated and complicated respiratory infections were examined. They received dipyridamole (Curantyl[®]) orally in a single dose equal to 2-3 mg/kg body weight. Control group consisted of 50 newborns with identical disease not treated with dipyridamole. Serum level of interferon was determined before treatment and at 24, 48, 72 and 96 hours after the treatment. Interferon titers before treatment in both groups were below 1:8. In newborns treated with dipyridamole interferon titers at 24 and 48 hours were 1:16 and 1:45, respectively, and decreased to the basic level after 72-96 hours. Interferon titers in the control group did not change significantly. Our results suggest that newborns react to interferonogen dipyridamole in similar way as do adults and elder children.

EFFECT OF CYCLOSPORIN A (CSA) AND VERAPAMIL ON MEMBRANE FLUIDITY OF CULTURED HUMAN SKIN FIBROBLASTS (HSF) Vytautas Batchiulis*, Hermann Toplak, Ulrich Wiesmann, Division of Metabolic Diseases, University Children's Hospital Bern, Switzerland and Pediatric Department(*) of Vilnius University, Lithuania

Calcium entry blockade is nephroprotective in the case of CSA toxicity. Therefore we studied the effect of CSA and verapamil on membrane fluidity of cultured human skin fibroblasts. Fluorescence anisotropy (rG), inversely related to membrane fluidity, was measured in Hank's solution in the presence of CSA (250, 750, 1500, 10000 ng/ml) and verapamil (10^{-6} , 10^{-5} , 10^{-4} M) at 37°C using the fluorescent marker 1, 6-diphenyl-1,3,5-hexatriene. CSA evoked a dose dependent decrease of rG from 0.103 ± 0.006 to 0.090 ± 0.008 , when compared to control (0.132 ± 0.011). At the concentration of 10^{-6} M verapamil increased rG from 0.173 ± 0.011 to 0.198 ± 0.012 . High concentration of verapamil (10^{-4} M) decreased rG from 0.173 ± 0.011 to 0.138 ± 0.005 . The therapeutic concentration of verapamil has stabilizing effect on cell membrane. It might be assumed that verapamil reduces CSA toxicity by modifying cell membrane fluidity.

LIVER CELL HETEROGENEITY IN BILIARY ATRESIA CIRRHOSIS

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To evaluate whether hepatocytes functional heterogeneity persists in biliary cirrhosis, we measured the zonal distribution of Glucose 6 phosphatase (GP) and NADPH dehydrogenase (ND) activities in ten normal human livers and in livers from 10 patients with extra-hepatic biliary atresia (EHBA), using cytochemistry /microspectrophotometry. In normal human liver, GP & ND respective mean activities were 37.3 ± 5.9 & 32.8 ± 5.5 density units (d.u) in the periportal zone and 24.1 ± 6.3 & 47.9 ± 8.6 d.u. in the perivenular zone. In EHBA patients, GP and ND respective mean activities were 51.3 ± 7.1 & 37.1 ± 7.2 at the nodule periphery and 39.2 ± 9.2 & 44.0 ± 6.5 at the nodule centre. We conclude that liver cell heterogeneity persists in EHBA cirrhosis, with GP more active at the nodule periphery than at the nodule centre but ND more active at the nodule centre, on the contrary to what has been observed in animal CCl₄ toxic cirrhosis. This zonation suggests that in EHBA, afferent vessels are located at the nodule periphery.