907 MUMPS VIRUS INDUCED DIABETES MELLITUS IN A RODENT MODEL. Terry Yamauchi, Mark A. Sperling, Heinrich Schedewie, John Elliot. Univ. Ark Med. Sci., Nat. Center Tox. Research, UCLA-Harbor Gen. Hosp., Dept. Ped., Little Rock and Jefferson, AR. and Torrance, CA.

Mumps virus have been postulated in the etiology of diabetes mellitus. The following experiments were to delineate the relationship between mumps virus infection and diabetes in a rodent model.

Adult Wistar rats were inoculated intraperitoneally with a laboratory adapted strain of mumps virus. Control animals were injected with a similar volume of spent tissue culture media. Standard glucose tolerance tests were performed on all animals at the onset of experiments and regular intervals thereafter. The rodents were maintained in seperate facilities and tested periodically for hyperglycemia and glucosuria. In addition, on a weekly basis infected and control animals were sacrificed and pancreatic tissues examined.

Two weeks after inoculation with mumps virus 17 of 20 rats demonstrated abnormal glucose tolerance curves which persisted 4-6 weeks. Insulin secretion, temperature elevation, appetite and activity did not differ in the two groups. Histological specimens revealed ablation of beta cell components in the pancreas of mumps virus inoculated rodents. Mumps virus was not recovered from any of the animals.

Mumps virus may be a diabetogenic agent in the rodent model. The mechanism of this action is postulated to be selective destruction of pancreatic beta cells. However, the production of ineffective insulin cannot yet be ruled out.

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Abstract withdrawn

MORPHOGENESIS

908 FETAL LUNG DEVELOPMENT AFTER AMNIOCENTESIS. Will R. Blackburn, Phylis A. Logsdon, and Jan Delli-Bovi. University of South Alabama College of Medicine, Pediatric Pathology, Mobile, Alabama.

The effects of amniocentesis (Ax) on the growth and development of fetal rat lung were studied in littermate fetuses subjected to minimum (Ax-Min; 0.1 ml) and maximum volume (Ax-Max; 0.5 ml+) amniocentesis at 17 days gestation. Non-operated littermates served as controls. Lungs of 12-14 fetuses/group were analyzed at term (22 days) for weight, composition (DNA, glycogen, lipid, phospholipid, phosphatidyl-choline) and histology (light and EM). Only Ax-Max reduced the weight of the lung ($P>.1$). DNA content was not influenced by Ax. Both Ax-Max ($P>.02$) and Ax-Min ($P>.1$) reduced lung glycogen. Ax did not alter the quantity of lung lipid at birth. The phospholipid fraction/lung was reduced ($P>.05$) after Ax-Max but not Ax-Min. The lungs of Ax-Max fetuses showed reduced alveolar space size and type II pneumocytes with few lamellar bodies and little glycogen. Histologic changes were not observed after Ax-Min. These studies indicated that large volume Ax reduces lung size by obliterating the fetal lung space rather than inhibiting lung cell proliferation. Ax-Max also reduces the phospholipid and surfactant pool size. Ax-min results in few detectable changes in lung growth or development. These studies explain certain aspects of the pulmonary dysfunction of infants with prolonged oligohydramnios.

911 GROWTH PROCESSES DURING EARLY MAMMALIAN DEVELOPMENT: FLUCTUATING POLYAMINE CONCENTRATIONS IN THE PREIMPLANTATION MOUSE EMBRYO. Donna L. Daentl and Laurence J. Marton. (Spon. by Carolyn F. Piel). Univ. of California San Francisco, Depts. of Growth and Development, Pediatrics, Neurosurgery, Laboratory Medicine and the Ctr. for Craniofacial Anomalies.

Studies of the interrelationships between biochemical changes related to growth and mouse embryo development may suggest factors which could be crucial for normal human development.

To determine the possible importance of polyamines for earliest mammalian development, microdeterminations were made of putrescine, spermidine and spermine during the first four days of mouse development. The fertilized egg has high levels of both putrescine and spermidine: putrescine (.127 ± .004 pmoles/egg) is 6 times and spermidine (.171 ± .006 pmoles/egg) is 2 times the concentration in the mature oocyte. During subsequent development putrescine declines 6-fold to a minimum at the 2-cell stage, but then dramatically increases to a maximal value at the morula stage. By the time of blastocyst differentiation, putrescine concentration is considerably lower, but spermidine is at a maximum. On day 4, just prior to implantation, spermine concentration is at a peak, while levels of putrescine and spermidine are low.

These data, together with the finding that incubation of 2-cell embryos in an inhibitor of spermidine synthesis results in cessation of development by the morula stage, suggest that polyamine synthesis has an important role during earliest mammalian development.

909 ZINC LEVELS IN ONE CASE OF FETAL ALCOHOL SYNDROME. M. Castro-Magana, P. J. Collipp, S. Y. Chen, S. Amin, and V. T. Maddaiah. Nassau County Medical Center, SUNY, Stony Brook Health Sciences Center, Dept. of Pediatrics, East Meadow, New York 11554.

Concentration of zinc (Zn) in hair, urine and serum were measured by atomic absorption spectrophotometry in one 14-month old girl with typical features of Fetal Alcohol Syndrome (FAS) (peculiar facies, congenital malformations, pre and post natal growth deficiency). She was born to an alcoholic woman who continued drinking heavily throughout her pregnancy. Zinc level was low in hair (66 μg/g) and urine (266.8 μg/g of creatinine) but normal in serum(95 μg%), reflecting probable chronic depletion of Zn. (Normal values are: hair 193 ± 18 μg/g, serum 75-160 μg%, urine 400-600 μg/g.) It has been shown that the offspring of Zn-deficient rats have marked growth retardation and high incidence of congenital malformation, some of which are similar to those seen in FAS. Alcoholic patients have been found to lose increased amounts of zinc in urine. Therefore, it is tempting to speculate that congenital Zn deficiency plays an important role in the pathogenesis of this syndrome.

912 CILIARY MORPHOLOGY AND CARDIAC MALROTATION. Park S. Gerald, Samuel R. Schuster, Roger N. Ruckman, Eveline E. Schneeberger. Harvard Medical School and Children's Hospital Medical Center. Departments of Pediatrics, Surgery, Cardiology, and Pathology. Boston, Massachusetts.

Kartagener's syndrome [situs inversus totalis (SIT), bronchiectasis and sinusitis] has been shown by others to have morphologically abnormal sperm tails and cilia, evidenced by absence of dynein arms. We used brush biopsies of the nasopharynx under local anesthesia for the study of ciliary morphology by EM in 9 patients with dextrocardia. Three of these have SIT and recurrent respiratory disease. One SIT lacks dynein arms and is a typical Kartagener's syndrome. A second SIT has abnormal (thickened and knobby) outer dynein arms in all cilia. This may be a new type of Kartagener's syndrome. The third SIT has normal dynein arms and some features of cystic fibrosis (CF), with intermittently elevated sweat sodium. The cultured fibroblasts of this patient do not show a CF-like response to ouabain, suggesting that this may not be CF. Of the 6 dextrocardiacs without SIT, all have normal dynein arms except one. In this patient (with L-TGA and without respiratory disease), the outer dynein arms are abnormally positioned in all cilia. We propose that dynein arm abnormalities may be an etiologic factor in a significant fraction of cardiac malrotations.