

† 990 ASYMPTOMATIC HYPOGAMMAGLOBULINEMIA IN A MOTHER PRESENTING AS NEONATAL MENINGITIS IN TWO SIBLINGS. Gregory S. Milleville, John H. Kersey and Rolf R. Engel, Hennepin County Medical Center and University of Minnesota, Dept. of Pediatrics, Minneapolis.

The unusual occurrence of group B streptococcal (GBS) disease in siblings lead to immune studies of a boy who mimicked his older sister in developing septicemia and meningitis from GBS by the 4th day of life. His serum IgG level was only 40 mg%. His IgM was 36 and IgA was < 7. Maternal levels were all low: IgG 40, IgM 36, IgA 14 and IgE <10 mg%.

Despite 14 days of penicillin and intravenous immunoglobulins, the boy's GBS (type 1c) meningitis recurred within 3 days with fever and a rise in the CSF leukocyte count from 20 to 2,025. He recovered completely during 2 more weeks of antibiotic Tx and at 7 months his Ig levels are normal. His sister had neonatal septicemia, meningitis, septic arthritis, and pneumonia which responded to 32 days of Tx. At 3 years, she is healthy with normal Ig levels: IgG 761, IgM 113, and IgA 62 mg%.

The mother is healthy and has not had excess infections. GBS (type 1c) was also recovered from her vagina. She was unresponsive to rubella immunization. She has low levels of IgA in saliva: 2 mg%, and breast milk: 3 mg%. Her panhypogammaglobulinemia is associated with normal numbers of B cells that have IgA, IgG, and IgM surface markers. Her lymphocytes had a normal *in vitro* response to phytohemagglutinin, Concanavalin A, and pokeweed mitogen.

This unique family emphasizes the importance of passive immunity in protecting newborns against GBS and it suggests that serious bacterial infections in neonates should prompt inquiry into the mother's immune competence.

991 SERUM AND MILK ANTIBODY LEVELS TO GIARDIA LAMBLIA Paolo Miotti, Robert Gilman, Larry Pickering, Guillermo Ruez Palacios, Thomas Cleary, Robert Yolken, Johns Hopkins Medical School, Baltimore, University of Texas Medical School, Houston, Institute of Nutrition, Mexico City.

There is little data relating to the prevalence and role of systemic and local antibody to Giardia lamblia (GL) antigens in mothers and children living in different environments. We obtained human milk and serum specimens from mothers and children living in Dacca, Bangladesh, Baltimore (MD) Houston (TX) and Mexico City. We compared the prevalence and levels of milk IgA and serum IgG antibodies to GL in the different populations by means of an ELISA assay utilizing purified antigens from cultured GL organisms. We found that 101/104 (97%) mothers living in Bangladesh, 22/26 (84%) mothers living in Maryland 9/17 (52%) mothers living in Texas and 23/37 (67%) mothers living in Mexico had detectable levels of milk IgA antibody to GL. The levels (geometric mean +/- SD) of antibody in the milk samples from mothers in Bangladesh (11.25 +/- .16 µg/ml) and Mexico (17.69 +/- 1.22 µg/ml) were significantly greater than the levels detected in the women living in Maryland (1.78 +/- 1.11 µg/ml) or Texas (1.70 +/- .097 µg/ml). In terms of total milk IgA, the IgA directed at GL in Bangladesh (1.54%) and Mexico (3.20%) were significantly greater than the corresponding percentages in Maryland (0.59%) and Texas (0.70%). Analysis of serial specimens obtained from lactating mothers over a period of 2 years revealed that most of the mothers had a constant level of antibody, although occasional increases in milk and serum antibody levels were noted. These studies indicate that serum and milk antibodies to GL are widely prevalent throughout the world but that the levels of milk antibody are significantly higher in areas of high exposure to GL.

992 NATURAL KILLER (NK) LYMPHOCYTE ACTIVITY ENHANCED BY INTERFERON. Ayman El Mohandes and D. Spencer Brudno (sponsored by Glen Rosenquist) George Washington University School of Medicine, Department of Child Health and Development, Division of Neonatology, Washington, D.C.

NK activity was studied in cord blood lymphocytes using K562 target cells. The mononuclear layer was depleted using an adherence technique. High effector to target ratios (50:1 and 100:1) were used in an 18 hour Chromium(51) release assay. K562 lysis was then measured after cord blood lymphocytes were incubated with interferon (1000 U/ML and 2000 U/ML).

EFFECTOR:TARGET		50:1		100:1	
		Adult(N=7)	Infant(N=10)	Adult(N=7)	Infant(N=10)
BASELINE		13.4 ± 2.3**	3.1 ± 0.7	21.9 ± 2.5**	7.0 ± 2.6
INTERFERON (1000 U/ML)		19.8 ± 3.3*	7.8 ± 2.3	26.3 ± 1.9**	9.0 ± 3.1
INTERFERON (2000 U/ML)		21.8 ± 2.3*	9.7 ± 7.1	31.4 ± 2.3**	8.4 ± 2.2

Means ± S.E.M. * p<.005, ** p<.001 Adults vs. Infants

NK activity was found to be statistically lower than adult means with both E:T ratios at baseline activity and after interferon enhancement. However, at the 100:1 ratio cord blood lymphocytes enhanced with 2000 U/ML of interferon showed a decline in the enhancement curve which could represent the effect of higher concentrations of interferon on suppressor lymphocyte subpopulations in the neonate.

● 993 BONE MARROW (BM) LYMPHOID PROGENITOR CELLS IN SEVERE COMBINED IMMUNODEFICIENCY (SCID). Steven Neudorf, John Kersey, Alexandra Filipovich, University of Minnesota, Minneapolis, Minnesota.

SCID is a heterogeneous disorder often associated with lymphopenia; hypothesized to be due to defective lymphoid differentiation or proliferation. We studied this hypothesis by analyzing BM mononuclear cells from 6 pt with SCID (as well as 5 age matched controls) for the presence of lymphoid progenitor cells. Such cells are E rosette⁻, surface Ig⁻ and include cells that express terminal transferase (TdT), the common ALL antigen (CALLA) or p24. BM cells were studied using immunofluorescent microscopy. Over 1000 cells per slide were examined and counted. Two patterns were seen. Group 1 (n=2) with no detectable lymphoid progenitor cells were more lymphopenic (p=.03) than pt in group 2 (n=4). Both groups were more lymphopenic than age matched controls (p=.04). All pt had SCID based on absent response to mitogens, recurrent infections and failure to thrive. 5/6 pt had hypogammaglobulinemia. Group 2 contained 1 pt with adenosine deaminase deficiency on transfusion therapy and 1 pt with the spurious lymphocyte syndrome. These results suggest that some pts with SCID have reduced numbers of BM lymphoid precursors and the absence of such cells may be related to the severity of lymphopenia seen.

	MARROW			BLOOD ₃
	%Tdt	% p24	%CALLA	ALC/mm ³
Gr 1	0 + 0	0.1 ± 0.1	0 + 0	556 ± 4.2
Gr 2	2.0 ± 1.8	16.7 ± 1.3	4.5 ± 2.2	2328 ± 1282
Norm.	5.5 ± 1.8	23.0 ± 3.2	14.5 ± 9.9	6343 ± 3264

994 NEUROLOGICAL COMPLICATIONS IN INFANTS WITH AIDS. Brian Novick, Monica Ulmann, Alfred Spiro, Anita Belman and Arye Rubinstein, Albert Einstein College of Medicine, Department of Pediatrics, Bronx, New York.

The clinical symptomatology of children with AIDS is similar to that observed in adults including fever, weight loss, diffuse lymphadenopathy and opportunistic infections. In adults, unusual neurological complications including progressive encephalopathy were recently reported. Since 1979, we have noted neurological abnormalities in 6 children with AIDS. 5 patients have not attained appropriate milestones, with severe global delay in areas of gross motor control, fine motor control, social functioning and language. Physical examination revealed spastic diplegia, hyperreflexia and positive Babinski in 4 with diminished muscular tone in 5 infants. 1 had seizures. Cerebrospinal fluid was acellular with normal protein and glucose. Skull X-rays have been negative. CT scans, with and without contrast in 4 children, displayed progressive cerebral atrophy. 1 showed intracerebral calcifications. EEG's showed diffuse slow waves with subsequent studies demonstrating further slowing of electrical activity. 4 of the 6 patients have continued neurologic deterioration with further loss of developmental milestones.

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995 ANTIBODIES TO HUMAN T-CELL LEUKEMIA VIRUS (HTLV) IN CHILDREN WITH ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS). James M. Oleske, Donald Francis, Cirilo Cabradillo, Rajendra Singh, Mary M. Zabala, Vantambadi Kalyanaraman, Thomas N. Denny, Houman Andieh, Antonio de la Cruz, Jane Getchell, Prakash Kaur, Anthony Minnefor (Spon. by Franklin C. Behrle). University of Medicine and Dentistry of New Jersey. Department of Pediatrics. Newark, New Jersey and Center for Disease Control, Atlanta, Georgia.

Six children previously documented with AIDS and seven age matched controls from a similar socio-economic environment were studied for the presence of antibodies (Ab) to HTLV. Additionally, 5 adult AIDS cases and 13 similarly matched adult controls were also examined for HTLV Ab. All determinations were done blindly by two assay systems: indirect membrane immunofluorescence (IF) using HTLV infected HUT-102 cells and Ab to structural proteins of HTLV by a radioimmune precipitation assay (RIP). All 6 pediatric AIDS cases were positive for IF-Ab while negative for RIP-Ab. In contrast, 6 of 7 pediatric controls were negative for IF-Ab and all 7 negative for RIP-Ab. The IF-Ab positive control child was only weakly positive. All pediatric AIDS cases had non-specific Ab directed against infected HUT-102 cells which required absorption. None of the pediatric controls had this non-specific Ab. All 5 adult AIDS patients were positive for IF-Ab while 2 were also positive for RIP-Ab. Two of 13 adult controls were weakly positive for IF-Ab and all negative for RIP-Ab. This data supports the concept that an HTLV-like virus is important in the etiology of AIDS.