1008 THYMULIN (FTS) AND THYMOSIN α_1 - DIAGNOSTIC CRITERIA FOR AIDS IN CHILDREN. Arye Rubinstein, Larry Bernstein, Brian Novick, Genevieve Incefy,

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Thymosin α_1 levels are markedly elevated in homosexual patients with AIDS or AIDS-related complex (prodrome) at a time when their T-cell function and OKT4/T8 ratios are depressed. On the other hand Thymosin α_1 and FTS are significantly reduced in patients with primary immunodeficiency diseases affecting cell mediated immunity. We have studied 14 infants with AIDS or AIDS prodrome with reversed OKT4/T8 ratios. FTS levels were diminished in all patients. Thymosin α_1 levels were elevated in 12 infants and normal or low in 2 (both treated with steroids, an agent known to depress thymosin α_1 levels). It appears that the FTS/thymosin α_1 ratio may serve as a diagnostic parameter to discriminate between congenital primary immunodeficiency and AIDS in infants.

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1009 THE SPECTRUM OF AIDS IN INFANTS. LACK OF EVIDENCE FOR INTRAFAMILIAL SPREAD. Ayre Rubinstein, Marc Sicklick, Larry Bernstein, Marguerite Mayers,

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36 infants with AIDS or AIDS related complexes (without opportunistic infection) have been studied over a period of 4 years. In the last 17 months 12 patients developed opportunistic infections. The clinical and immunological spectrum of AIDS in infants is similar to that reported in adults. Some of the unique features in the pediatric age group are: recurrent parotitis, interstitial pneumonia without pneumocystic carinii and a higher incidence of bacterial infections preceding the development of opportunistic infections. Most infants (except for 3) were born to i-v drug using mothers who had (except for 3) an immunodeficiency suggestive of AIDS or a clinical disease meeting the CDC criteria for AIDS. Pediatric and adult household contacts of infants with AIDS were studied clinically and immunologically. In no instance was an older child found in the family or foster care home who had developed AIDS. In 2 families 4 children were found who had elevated serum IgG levels, 2 of whom had also reversed OKT4/T8 ratios. None of these children had any other immunological abnormality or clinical symptoms. This data argues against a transmission of AIDS by a casual or intimate-non-sexual contact.

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1010 TREATMENT OF AIDS WITH INTRAVENOUS GAMMAGLOBULIN. Arye Rubinstein, Marc Sicklick, Larry Bernstein, Bernard Silverman, Brian Novick, Morris Charytan

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B cell abnormalities have been reported in adults with AIDS. These abnormalities include poor antibody responses to neo-antigens, and poor in vitro lymphocyte mitogenic responses to Pokeweed Mitogen. Similar abnormalities have been reported by us in infants with AIDS and AIDS related complex. These infants have had multiple bacterial infections including sepsis. 16 patients, 11 infants and 5 adults have been treated with i-v gammaglobulin (Cutter Laboratories) 200mg/kg biweekly for 3 months to 3 years. Clinically, a significant reduction in bacterial infections was noted in all patients. 2 infants developed new opportunistic infections while on treatment. In most patients an increase in the OKT4/T8 ratio and in lymphocyte blast transformation to phytohemagglutinin and pokeweed mitogen was noted. In 2 out of 4 studied patients suppressor T cell functions were restored. During the first infusions of gammaglobulin transient elevations of serum immune complexes and a drop in total hemolytic complement were sometimes noted. However, with time serum immune complexes decreased to normal levels. None of these changes have been observed in 19 untreated infants with AIDS. I-V gammaglobulin in conjunction with other immunotherapeutic modalities may be of benefit for AIDS patients.

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1011 ABNORMAL MEMBRANE DEPOLARIZATION IN THE PATHOGENESIS OF THE CHEMOTACTIC DEFECT IN NEONATAL PMNS. Fulvio Sacchi, Mary M. Coello, Mark M. Boucek and Harry R. Hill, Univ. of Utah, Salt Lake City, Utah.

Although a number of investigators have reported that neonatal PMNs have a profound defect in chemotaxis, almost nothing is known about the pathogenesis of this disorder. In the present study we have examined membrane depolarization and calcium requirements for chemotaxis in neonatal and adult PMNs. Suspension of adult PMNs in calcium deprived media resulted in a decreased chemotactic response to FMLP (19% inhibition; $p < 0.05$). Incubation of adult PMNs with the calcium channel blocking agent verapamil also significantly impaired chemotaxis (32% inhibition $p < 0.001$). In marked contrast, the neonatal PMN demonstrated an enhanced chemotactic response when suspended in calcium deprived media (26% increase; $p < 0.05$) and after incubation with verapamil (57% increase; $p < 0.001$), suggesting that inhibitory levels of intracellular calcium might already exist. Adult and cord blood PMN membrane potential changes in response to FMLP were also studied using the membrane potential-sensitive dye, 3-3' dipentylloxycarbocyanine and a spectrofluorescence assay. Adult PMNs showed a marked increase in fluorescence emission in response to FMLP (32% increase; $p < 0.001$) indicating significant depolarization of the cell membrane. In contrast, FMLP stimulation of neonatal PMNs failed to evoke any significant change in fluorescence and, thus, membrane potential changes. These data suggest that altered intracellular calcium metabolism may exist in the neonatal PMN, preventing normal membrane depolarization from occurring.

1012 CHARACTERIZATION OF THE MOLECULAR BASIS FOR PMN DYSFUNCTION & RECURRENT INFECTION IN THE GP-138 DEFICIENCY SYNDROME. F. Schmalstieg, D. Anderson, W. Shearer, M. Tosi, B. Rudloff, W. Dickey, & T. Springer. U. of Texas Galveston, Baylor, Houston, & Harvard, Boston.

A deficiency of a high MW glycoprotein present on the surface of normal PMNs, was found in unrelated patients (3 F, 1 M) with leukocytosis, periodontitis, & recurrent infection. Multiple adhesion-dependent functions including random and directed migration of affected PMNs were profoundly diminished. SDS-PAGE of NP-40 lysates of these PMNs and controls indicated that the deficient protein had a MW of 138,000 daltons. Further characterization showed the protein had a large carbohydrate content and was a major surface glycoprotein with a pI of 5.2-5.4. Galactose oxidase - NaB 3H_4 surface labeling demonstrated the three F were totally deficient in surface GP-138, but the M patient had 5-10% of normal on the PMN surface. Immunoprecipitation experiments with polyclonal anti-GP-138 on normal PMNs suggested that the functional protein consisted of two or more polypeptide chains. To further test this hypothesis, monoclonal antibodies against known leukocyte cell surface proteins were tested using fluorescent activated cell sorting (FACS). LFA-1, p150.95, and OKM1 as well as the beta subunit these polypeptides share, were absent in the two F tested. The M patient was identical except he had ~30% of normal OKM1 by FACS. This data suggests GP-138 and OKM1 may be similar molecular species. Thus, PMN dysfunction in the GP-138 deficiency syndrome is related to impaired expression of at least three related high MW glycoproteins (OKM1, LFA-1, & p150.95) necessary for adhesion dependent cell function.

1013 REVERSAL OF SIGNS AND SYMPTOMS OF ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) WITH PREDNISONE. William Andiman, John Dwyer, Karen Lomax, Yale Univ Sch Med, New Haven, CT.

We describe in detail the case of a male infant of a prostitute and IV drug abuser. The child presented at 9 mo of age with severely delayed growth (6.6 kg) and development, recurrent bacterial infections, lymphadenopathy, hepatosplenomegaly (8 cm), persistent oral candidiasis, and chronic pulmonary infiltrates. Immunologic studies showed polyclonal hypergammaglobulinemia and inverted T-cell T4/T8 ratios. Skin tests to Candida and phytohemagglutinin were positive. Lung biopsy showed peribronchiolar lymphocytic infiltrates. Axillary lymph nodes contained obliterated margins with increased plasmacytes centrally. Liver biopsy demonstrated mild ballooning of hepatocytes. Bone marrow biopsy revealed hypercellularity with increased plasmacytes. EB virus was repeatedly isolated from lymph node, bone marrow, and blood lymphocytes, although antibodies to EBV did not appear for another 4 mo. Sera from both mother and child did contain antibody to human T-cell leukemia virus. Multiple sera were negative for antibody to cytomegalovirus and herpes virus. These viruses failed repeatedly to grow from tissue specimens. Initially, the child's course was observed off all treatment. After 8 wks of slow deterioration, he became lethargic, tachypneic, and febrile without apparent source of infection. At that point, he was begun on oral prednisone (PDR), 2 mg/kg/day. After 1 wk, he became afebrile with normal respirations. His adenopathy dissolved along with resolution of interstitial infiltrates. After 4 wks of PDR therapy, weight was 8.7 kg, serum immunoglobulins were normal, and spleen tip was just palpable. After tapering of PDR, all indicators of disease reverted toward pre-PDR values. Reinstitution of PDR again changed all parameters in a beneficial manner. We conclude that PDR may be effective in treatment of the AIDS prodrome.