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CHOLECYSTOKININ OCTAPEPTID (CCK-OP) ENHANCES THE PROLIFERATIVE RESPONSE OF RAT SPLENIC LYMPHOCYTES TO CONCAVALIN A (CON-A). Yoram Elitsur, Boris Albini, Emanuel Lebenthal. Children's Hospital of Buffalo, International Institute and State University of New York, Depts. of Microbiology and Medicine, Buffalo.

Gut hormones were shown to affect the digestive process. However, little is known about their interaction with the immune system. We investigated the effect of CCK-OP on 3H-thymidine incorporation into rat splenic (SL) and peripheral blood lymphocytes (PBL) induced by Con-A. Various doses of CCK-OP (10^{-8}M to 10^{-14}M) were incubated with Con-A (33µg/ml) and lymphocytes in cell cultures ($2X10^5$ cells/well) for 72 hrs; 3H-thymidine (0.4 µci/well) was added and cultures harvested 18 hrs. later. Incorporation of thymidine was quantitated in a Beckmann Liquid Scintillation System (Irvine, Ca). The results (Table) suggest an enhancement of Con-A-induced lymphoproliferative response of spleen cells by up to 48%. In contrast, PBL did not show any effect of CCK-OP on Con-A induced cell proliferation. CCK-OP alone did not induce lymphoproliferation.

Table: Ratio of Stimulation Indices (Con-A+CCK-OP/Con-A)

Conc.CCK-OP:	10^{-8}M	10^{-10}M	10^{-11}M	10^{-12}M	10^{-14}M
SL	1.26±0.04	1.43±0.28	1.48±0.42	1.40±0.21	1.14±0.21
PBL	0.99±0.25	0.87±0.23	N.D.	0.91±0.20	1.03±0.32

These data suggest that physiological concentrations of CCK-OP enhance lymphocyte proliferation and thus may partake in the regulation of the immune response.

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EFFECT OF IVIG ON CIRCULATING IMMUNE COMPLEXES (CIC) IN PEDIATRIC AIDS/ARC. Maadhava Ellaurie; Theresa Calvelli; Arye Rubinstein. Albert Einstein College of Medicine, Department of Pediatrics, Bronx, New York.

CIC were studied by Raji cell assay in 23 patients with AIDS and ARC before and after IVIG. All patients had elevated CIC prior to initiative IVIG treatment. Patients were followed for an average of 20 months during which time they received bi-weekly infusions of Cutter IVIG. A significant reduction of CIC following IVIG was found in 13 patients, 2 of whom subsequently died during follow-up. In 10 other patients there was an increase of CIC after IVIG treatment. This group exhibited a mortality rate of 70%. There was a good correlation between elevated CIC and decreased antigenic and mitogenic responses, low T_4/T_8 ratio and evidence of viral infection with EBV, CMV and Herpes. Among those patients in whom CIC decreased following IVIG, 33% showed some restoration of mitogen induced proliferation. In contrast, the group that had increased CIC showed no such improvement. No correlation between the level of serum IgG and M and CIC-IgG and CIC-IgM was found and no uniform differences in CIC-IgG or CIC-IgM were observed after IVIG. The mean concentration of IgG and IgM in CIC was 54mg% and 32mg% respectively. After treatment with IVIG the molecular weight of the CIC had decreased from a pre IVIG level of 2 million.

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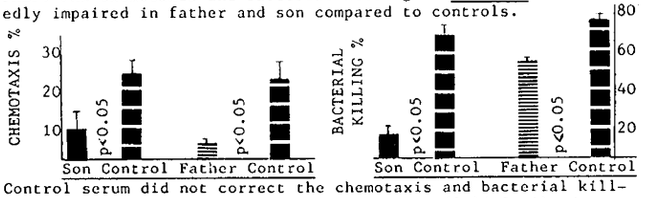
FAMILIAL IgG4 SUBCLASS DEFICIENCY WITH NL IgA ASSOCIATED RECURRENT INFECTIONS WITH DECREASED NEUTROPHIL CHEMOTAXIS AND BACTERIAL KILLING. Mark Ellis, K. Dumars, S. Gillman, C. VandeVen, D. Heiner, M. Cairo. (Spon. by I. Lott) Child. Hosp. of Orange County, Dept. of Med. U.C. Irvine Orange, CA

We evaluated a 14 month old white male, for recurrent bacterial otitis media and sinusitis. The patient's father had similar infections as a child. The mother was normal. Father and son had normal IgG, IgA, IgM, IgE, IgG1, IgG2 and IgG3. The child had undetectable IgG4 (normal 0.9-107 mg/dl) and the father had IgG4<5 mg/dl (5-362) as measured by radial immunodiffusion. The family was screened for defects in PMN function measuring chemotaxis activity in response to E.Coli-endotoxin, bacteria killing against S.aureus and superoxide production with Cyto B/FMLP. PMN chemotaxis and bacterial killing of S.aureus were markedly impaired in father and son compared to controls.



Control serum did not correct the chemotaxis and bacterial killing defects. Both O_2^- was normal. Father is HLA (2,)(15,14) and son is (2,31)(15,7). High resolution chromosomal banding is pending. We conclude that familial IgG4 subclass deficiency with NL IgA can be present in association with defects in PMN chemotaxis and bacterial killing.

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RESPONSE OF HIV INFECTED CHILDREN TO PNEUMOCOCCAL VACCINE. Senih M. Fikrig, Rachel Menez-Bautista, Howard Menikoff, Gerald Schiffman. Depts. of Pediatrics and Microbiology and Immunology, SUNY-Health Science Center at Brooklyn, New York.

17 children with AIDS, ARC and other manifestations of HIV infection as well as asymptomatic ones were vaccinated with pneumococcal polysaccaride vaccine. Antibody to 12/23 types represented in the vaccine was tested a month later. Only 2/17 subjects responded to types 6A, 9N and 14 - poor antigens in normal subjects - whereas 6 to 13/17 subjects showed good response (>40% of baseline value or over 200 nanograms antibody N/ml) to types 1, 3, 7F, 8, 12F, 18, 19F, 23F and 4. The antibody production was not related to the classification of HIV infection status. Patients with AIDS, ARC, as well as asymptomatic ones showed similar degree of responses. Two patients that showed no response to 12 pneumococcal types were classified as ARC. Results indicate that HIV infected children can respond to B cell antigens even though protection provided by the pneumococcal vaccine may be limited.

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TWO COLOR FLOW CYTOMETRIC ANALYSIS OF CORD BLOOD LYMPHOCYTES. Thomas A. Fleisher, Claire Hagengruber, Gerald E. Marti and R. M. Blaese. NIH, Bethesda, MD

We have studied the surface phenotype of cord blood lymphocytes obtained from ficoll hypaque gradients using 17 pairs of monoclonal antibodies (Mab). For this evaluation, lymphocyte gates were set based on cell volume and side scatter such that there was less than 2% monocytes. T cell characterization revealed that the CD8+ (T8,Leu2) cells were significantly decreased compared to adult normals (13.6%vs26.5%) while CD4 (T4,Leu3) cells were normal (47%vs48%). This decrease in CD8 cells was reflected by a decrease in the % of T cells as measured by CD3 (T3) and CD5 (T1)(61%vs76%) and an increase in the mean T4/T8 ratio (4.3vs2.0). There was no evidence for an increase in CD4/CD8 double staining cells (<1%) or T cell activation (HLA-DR & IL-2R < 1% on CD3+ cells). Examination of B cells revealed that the % and absolute number was markedly increased when compared to adult normals (12.8% & 594/mm^3 vs6.5% & 114/mm^3) based on CD19 (B4) and CD20 (B1). Of significance was the observation that more than 1/2 of the cord blood B cells stained with the T cell marker CD5 (T1) while in adult normals this is seen on less than 20% of the B cells. This CD5+ B cell has surface Ig as well as CD19 and CD 20. Studies of NK cells demonstrated presence od CD16 (Leu11)(7%) but virtual absence of HNK-1 (Leu7). In addition, this paired Mab analysis failed to identify almost 20% of the lymphocytes. Thus, cord blood lymphocytes have phenotypic differences in T cells, B cells and NK cells when compared to adult normals as well as having a population of lymphocytes that fail to stain using a large panel of Mab directed to lymphocyte surface antigens.

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IMMUNE HEMOLYTIC ANEMIA AFTER BONE MARROW TRANSPLANTATION FOR SEVERE COMBINED IMMUNODEFICIENCY. Diane Foster, Robert J. Mamlok, Fatih Ozkaragoz, Christopher Leveque, Armond S. Goldman, and Randall M. Goldblum. University of Texas Medical Branch, Departments of Peditrics, Human Biological Chemistry and Genetics and Pathology, Galveston, TX.

Bone marrow transplantation (BMT) has occasionally been complicated by the development of hemolytic anemia from specific alloantibodies to either ABO or Rh antigens. We report a case of post-BMT hemolytic anemia in severe combined immunodeficiency (SCID) due to IgG antibodies that agglutinate all red blood cells (RBC) in a broad panel of allotypes.

A 15 month old female with recurrent respiratory distress and oral candidiasis had panhypogammaglobulinemia, decreased T-lymphocytes ($312/mm^3$) and mitogen responsiveness. A diagnosis of SCID prompted transplantation of unfractionated bone marrow from her HLA identical, MLC non-reactive mother without prior ablative therapy or post-transplant graft-vs-host disease (GvHD) prophylaxis. Bone marrow engraftment proceeded rapidly without evidence of GvHD. Eight weeks after BMT her hemoglobin acutely fell to 3.4g/dl with 42% reticulocytes. Direct and indirect antiglobulin tests were positive. Although the donor (A-) and recipient (A+) differed at two Rh loci, the antibody specificity was not restricted to the Rh allotypes.

This appears to be the first report of post-BMT immune hemolytic anemia in SCID due to an antibody of broad rather than allospecificity. Ongoing studies may provide a better understanding of the mechanisms of autoantibody production in immune hemolytic anemia.