691 IMMUNOCOMPETENCE IN OBESITY. R.K. Chandra, G. Woodford, B. Au and K.M. Kutty. Memorial Univ. of Newfoundland, Janeway Child Health Centre, Dept. of Pediatrics, St. John's, Newfoundland.

Obese individuals are susceptible to respiratory and cutaneous infections and post-operative wound sepsis. In a group of 21 children and adolescents diagnosed to be obese on the basis of weight-for-height and skin-fold thickness, a comprehensive assessment of immunity function showed serum immunoglobulins within the normal range, slight elevation of serum IgE and adequate antibody response. Subpopulations of circulating lymphocytes were comparable in proportion and number in the obese and the non-obese. Cutaneous delayed hypersensitivity and mitogen-induced lymphocyte DNA synthesis were reduced, the latter particularly when autologous plasma was used in cell cultures. Serum levels of complement components, opsonization and phagocytosis were normal, but intracellular bacterial killing was impaired. In the obese group, the mean serum lipid concentration was higher, and transferrin saturation and zinc level lower than in the control group. It is suggested that alterations in the immunocompetence of obese individuals may in part be the result of changed nutritional milieu and that such changes may contribute to increased susceptibility to infection.

692 VENOM IMMUNOTHERAPY FOR INSECT HYPERSENSITIVITY. Bradley E. Chipps, Martin D. Valentine, Anne K. Sobotka, and Lawrence M. Lichtenstein. The Johns Hopkins University, Departments of Pediatrics and Medicine, Baltimore, Maryland. (Spon. by Richard C. Talamo)

Twenty-five children with histories of hypotension, respiratory distress or a marked generalized cutaneous response following a sting by insects of the Hymenoptera order were selected after fully informed (familial) consent, for a trial of immunotherapy with insect venom. As indicated by venom skin tests, IgE antibody measurements and leukocyte histamine release, five were sensitive to only honeybee venom, thirteen to vespids (yellow jacket, white-faced hornet, yellow hornet) and seven to both species; each child was treated over 4-5 months by injections of each appropriate venom, reaching a maintenance dose, previously established in adults, of 100 µg. Routine toxicity studies were carried out, without significant abnormalities being noted. Radioimmunoassays for antibodies to honeybee venom phospholipase A and yellow jacket venom indicated that all children had an increase in IgG (5-25fold) which had usually peaked at the time of challenge. Three children were stung in the field by the appropriate insect without sequelae. The clinical protection of the remaining twenty children was assessed by deliberate sting by the appropriate insect(s) in an emergency room setting. Without therapy 12 reactions were expected; none were observed. This is the first study which shows that venom immunotherapy is well tolerated and clinically protects insect sensitive children.

693 RESPONSE TO ADENOSINE DEAMINASE (ADA) REPLACEMENT THERAPY IN A CHILD WITH SEVERE COMBINED IMMUNODEFICIENCY DISEASE (SCID) AND ADA DEFICIENCY. Aida Daoud, Beatrice C. Lampkin, John W. Dyminski, Mary S. Coleman, James Donofrio and John J. Hutton, Children's Hospital Medical Center, Cincinnati, O., and University of Kentucky and VA Hospital, Lexington, Kentucky.

A boy was diagnosed to have SCID at age 4½ mos. No thymic shadow, adenoidal tissue or skeletal anomalies were found on X-ray. IgG and IgM were markedly decreased. Lymphocyte response to phytohemagglutinin (PHA) revealed a stimulation index (SI) of 0.96 and candida, SK-SD and PPD skin tests were negative. Enzymatic assays for ADA showed only trace amounts in red cells, lymphocytes and granulocytes. Patient was treated with transfusions of irradiated frozen red cells and irradiated frozen plasma according to the method of Polmar, et al. Gradual clinical improvement occurred and he is now 18 mos old and clinically well. After starting therapy his immunoglobulins increased to above normal levels. However, in contrast to the patient reported by Polmar, et al, our patient still has no thymus by chest X-ray and his absolute numbers of lymphocytes have varied markedly, as have his % of T and B cells. Similarly, SI's in response to PHA, ConA, pokeweed and LPS have varied between 0-40% of normal. These variations have occurred in spite of a normal level of ADA in his red cells. No inverse correlation between the nucleotides and ADP levels was noted. These results indicate that replacement therapy has been beneficial clinically. However, because of the fluctuation in the immune responses, the role of replacement therapy with ADA is unclear and further studies are indicated.

694 DIFFERENCES IN TRYPSIN SUBSTRATES AND INHIBITORS ON PMN CHEMOTAXIS AND PHAGOCYTOSIS. A Todd Davis, Pamela G. Grady, C. L. DeFranco, and Emmanuel Shapira. Northwestern Univ. Med. Sch., Children's Memorial Hospital, Department of Pediatrics, Chicago.

There are many apparent similarities between PMN chemotaxis and phagocytosis. Previous investigators have suggested that serine esterases are involved in the chemotactic response in so far as a variety of inhibitors and substrate analogues inhibit chemotaxis. We studied the effect of various trypsin inhibitors and a substrate on human PMN chemotaxis and phagocytosis. Reversible inhibition of chemotaxis was demonstrated with the low molecular weight trypsin substrate N-benzoyl-L-arginine ethyl ester (BAEE). The low molecular weight active site titrant, nitrophenyl-P-guanidino benzoate (NPGB) revealed irreversible inhibition of chemotaxis, whereas the low molecular weight inhibitor, benzamidine (BENZ) and the high molecular weight inhibitor, soybean trypsin inhibitor (STI), caused reversible inhibition. The various inhibitors and the substrate gave varying inhibition profiles. The following concentrations gave complete inhibition of chemotaxis: BAEE, 100mM; NPGB, 0.5mM; BENZ, 10mM; STI, 5mM. Neither the active site titrant, NPGB, nor the low molecular weight inhibitor, BENZ, affected phagocytosis as determined by the Maaloe technique at concentrations completely inhibiting chemotaxis. These results may suggest different underlying mechanisms initiating chemotaxis and phagocytosis.

695 B AND T CELL ASSAYS IN ACUTE LYMPHOBLASTIC LEUKAEMIA. Leverett L. de Veber and David A. Bell (Sponsored by James E. Boone). Department of Paediatrics, Mar Memorial Children's Hospital and Department of Medicine, University Hospital, London, Ontario, Canada.

Assays of B and T cells were carried out on the peripheral blood of 12 children with acute lymphoblastic leukaemia. Six cases with few blast cells in the peripheral blood showed a decrease in T cells and an increase in IgM-lambda B cells. There were no unusual clinical features in this group. Three cases showed no specific findings on B and T cell assays. Three cases with white counts over 100,000 showed only B cell markers on the blast cells. Two of these cases have had frequent relapses within months of diagnosis with one death. The third case is still in remission eight months from diagnosis. One case of acute myeloblastic leukaemia by morphology, special stains and E/M with a white cell count of 104,000 showed large numbers of B cells in the peripheral blood. Serum immune globulin determinations and serum electrophoresis patterns were essentially normal in all cases.

No cases of "T cell leukaemia" were noted. "B cell leukaemia" is a relatively uncommon variety of A.L.L. and cases reported in the literature have uniformly a poor prognosis with occasional exceptions.

Despite problems in the techniques, and in interpretation of the findings, T and B cell assays provide important information in the subclassification and prognosis of A.L.L. in children.

696 IDENTIFICATION AND CHARACTERIZATION OF HUMAN SUPPRESSOR LYMPHOCYTES. Stanley M. Fineman, Fuad B. Mudawwar, and Raif S. Geha, (Spons. by Fred S. Rosen), Harvard Medical School, Children's Hospital Medical Center, Department of Pediatrics, Boston.

The capacity of human peripheral blood lymphocytes (PBL) to suppress in vitro immune responses was investigated.

PBL were incubated with Concanavalin A (Con A) (50 ug/ml) for 48 hours, washed in Hank's Balanced Salt Solution containing α-methyl-D-mannoside (5 mg/ml), and subsequently added at a 1:1 ratio to cultures of normal untreated lymphocytes. Con A treated cells suppressed the proliferative response of untreated lymphocytes to mitogens (phytohemagglutinin, Con A), antigens (tetanus toxoid) and alloantigens (one-way mixed lymphocyte culture), as measured by ^{3}H-thymidine incorporation into DNA. Con A treated cells also suppressed pokeweed mitogen induced immunoglobulin production in vitro by normal lymphocytes, as measured by radioimmunoassay.

Con A treated cells lost their suppressor activity following treatment with mitomycin C (50 ug/ml) or X-irradiation (> 500 RADS). It was shown that the Con A activated suppressor cell was a T cell which was nylon nonadherent and which bore surface receptors for histamine. Treatment of PBL or of T cells with antisera to B cell alloantigens (Ia like antisera) either before or after activation with Con A did not interfere with the suppressor activity of these cells.

Assay of suppressor activity of Con A treated PBL could be helpful in the study of immunodeficiency diseases, graft-versus-host disease, autoimmune diseases and atopic states.