† 830 EFFECT OF RESPIRATORY VIRAL INFECTION ON THE UPTAKE AND IMMUNE RESPONE TO DIETARY ANTIGENS: POSSIBLE ROLE IN POTENTIATION OF ALLERGY. J. Freihorst, P.A. Piedra, P.L. Ogra, SUNY at Buffalo, Children's Hosp., Dept. Ped., Div. Infect. Dis., Buffalo, NY.

Groups of Balb/c-mice were sham infected or infected intranasally (I/N) with respiratory syncytial virus (RSV). The animals were repeatedly exposed I/N to ovalbumin (OVA), with or without adjuvant (Alum). At different intervals, levels of anti-OVA antibody and OVA concentrations were determined in serum and pulmonary lavage fluid (PLF) employing the techniques of Elisa (for IgG, IgA, IgM) and passive cutaneous anaphylaxis (for IgE). In all mice, anti-OVA IgG in the serum reached peak titers after 24 days, with statistically significant higher titers in the infected mice compared to uninfected controls. OVA-specific IgA and IgM could be detected in low concentrations in both groups. Antigen-specific IgA in PLF was detectable from 17 to 31 days after exposure, with higher titers for the RSV-infected animals. OVA-specific IgE response in serum and PLF was demonstrable in 20% of the infected mice and in none of the controls. OVA concentration in serum peaked at 2 hours after administration and appeared to be higher in RSV infected animals as well as non infected animals treated with adjuvant, when compared to uninfected animals without adjuvant treatment. These observations suggest that acute bronchopulmonary infection with RSV is associated with significant increase in the development of IgG and IgE immune response to other concurrently inhaled antigens. These findings may explain the observed nonspecific increase in allergic reactions to dietary antigens during viral infection.

† 831 IMMUNOGENICITY OF PURIFIED VARICELLA ZOSTER VIRUS GLYCOPROTEINS. Roger H. Giller, Stanley Winistorfer, Charles Grose. University of Iowa College of Medicine, Department of Pediatrics, Iowa City, IA.

Development of an effective, noninfectious subunit vaccine to the varicella zoster virus (VZV) will require identification of viral gene products critical for generating protective host responses. To better define the antigens which provoke immunity to VZV, viral glycoproteins (gps) were purified and tested for their ability to stimulate human lymphocytes. VZV specified gps were radiolabelled with [^{14}C]-glucosamine then extracted from sonicates of infected cell monolayers using nonionic detergents. Mixed VZV gps were isolated by passing infected cell extracts over lentil lectin conjugated Sepharose and competitively eluting bound gps with α-methylmannoside. Gp I (98/62), gp II (140/66) and gp III (118), the predominant gps expressed in VZV-infected cells, were purified with murine monoclonal antibodies employing a novel avidin-biotin immunoaffinity method. Purity of the isolated VZV gps was confirmed by fluorography of SDS-PAGE gels. Both mixed viral gps (Stimulation Indices = 6-15) and individually purified gps (I, II, III) (SI = 3-8) induced proliferation by MNC from VZV-immune subjects. Responses observed were VZV-specific since proliferation failed to occur if MNC were obtained from VZV-susceptible individuals or if antigens were prepared from uninfected monolayers. Coculturing studies of separated MNC subpopulations indicated that responses to the gps represented proliferation by T4+ lymphocytes and required antigen presentation by adherent MNC. We conclude that virally encoded gps contain epitopes which stimulate VZV-specific proliferation by human T cells.

832 INCIDENCE OF MAJOR INFECTIONS IN CHILDREN WITH DISEASE CAUSED BY THE HUMAN IMMUNODEFICIENCY VIRUS (HIV). Asha Gupta, Aditya Kaul, Joel Fiedler, Sharon Nachman and Donald Gromisch. (Sponsored by Lawrence Shapiro). New York Medical College, Department of Pediatrics, Valhalla, New York.

42 children with HIV disease documented by positive serology have been followed at our institution over the last three years. 27 children presented with and continue to have clinical picture of AIDS-related complex. 15 developed major infections. These were Pneumocystis carinii (8), disseminated M. avium (3), disseminated CMV (1), pneumococcal meningitis (2) and pneumococcal endocarditis(1). Intravenous gammaglobulin is not routinely used in children with HIV disease at our institution. It will be informative to know if our incidence of severe bacterial infections is different from institutions where this treatment is routine. Prospective randomized trial to determine the efficacy of this treatment is in order.

● 833 DYSLIPOPROTEINEMIA IN PEDIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS. Norman T. Ilowite, Paul Samuel, Marc S. Jacobson, Sp. by Phillip Lanzkowsky, SUNY at Stony Brook, Schneider Children's Hospital of Long Island Jewish Medical Center, Dept. of Peds, New Hyde Pk, N.Y.

Patients with SLE are at increased risk for premature atherosclerosis. Dyslipoproteinemia, an etiologic factor, has been demonstrated in adult SLE patients, although the relationships of disease activity, diet and steroid therapy are obscure. To differentiate the roles of steroid therapy and active disease in the dyslipoproteinemia of pediatric SLE, we measured fasting lipid profiles in 7 newly diagnosed, untreated patients and repeated these studies in 5 after high dose steroid therapy, coincident with clinical improvement. Values for total cholesterol (TC), triglycerides (TG), very low, low and high density lipoprotein cholesterol (VLDL-C, LDL-C, HDL-C), apoproteins A-I (apoA-I) and B (apoB) are shown in mg/dl for the patients and adolescent controls (*$p < .05$ vs. controls) (#$p < .05$ pre vs. post treatment).

	TC	LDL-C	HDL-C	VLDL-C	TG	apoA-I	apoB
SLE pre	137±23	79±19	24± 6*	33±12*	171±62*	58±12*	67±13
SLE post	213±21*#	113±23#	50± 9#	48± 9*#	176±61*	100±22*#	84±19
Nl cont	160±27	93±25	60±21	8± 7	65±40	141±22	84±21

At diagnosis, patients exhibited low plasma HDL-C and apoA-I with elevated plasma VLDL-C and TG. This pattern can be explained by decreased lipoprotein lipase activity. After steroid treatment, LDL-C, HDL-C, VLDL-C and apoA-I increase. Thus, dyslipoproteinemia in pediatric SLE can be attributed to active disease, independent of corticosteroid therapy. The relative roles of the dyslipoproteinemias of active disease and steroid therapy in the premature atherosclerosis of SLE remains to be determined.

● 834 IgG2 SUBCLASS DEFICIENCY PRESENTING AS H. INFLUENZAE B (HIB) DISEASE AFTER IMMUNIZATION WITH HIB VACCINE. Richard A. Insel, Bruce Gellin, Claire Broome, David Smith. Univ. Rochester School Med and Dent, Dept Peds; Center for Disease Control; Praxis Biologics, Rochester NY, Atlanta GA.

Twenty-six healthy children previously immunized (imm) with the Hib capsular polysaccharide (Hib CP) vaccine at age 19 to 38 mos (GM=28 mos; 1 of 26 <24 mos) developed Hib systemic disease at age 22 to 45 mos (GM=32 mos), which was 2 to 44 wks (GM=12.6 wks) after imm. Disease consisted of 16, 6, and 4 cases of meningitis, epiglottitis, and other infections, respectively. Antibody (Ab) to the Hib CP in serial convalescent serum samples collected 24 days-8 mos after infection failed to increase to 1μg/ml in 17 of the 26 and had a GMT of 0.56μg/ml, which contrasted with Ab of an age-matched control group of Hib infected, nonimm children (GMT=2.7μg/ml, $p<.05$). IgG2 subclass deficiency was detected in 9 of the 26 and was associated with low or borderline-low levels of IgG and IgA in 5 and 6 of the 9 respectively. Postinfection Ab to the Hib CP was <1μg/ml in 7 of the 9 with IgG2 deficiency (GMT=0.29μg/ml), but all had normal Ab titers to tetanus and diphtheria toxoids. At present, reimm with the Hib CP and imm with pneumococcal CPs induced poor Ab responses in 1 of 1 and 3 of 3 with IgG2 deficiency, respectively. These findings suggest that a considerable proportion of cases of this new entity - Hib disease after Hib CP imm - is associated with IgG2 subclass deficiency and poor Ab responses to Hib infection.

▲ 835 SUBCELLULAR LOCATION OF MAC-1 (CR-3) IN HUMAN NEUTROPHILS: EFFECTS OF CHEMOTACTIC FACTORS AND PMA. Douglas H. Jones, Donald C. Anderson, Bean L. Burr, Helen E. Rudloff, David K. Dennison, C. Wayne Smith, Frank C. Schmalsteig. Departments of Pediatrics, Baylor College of Medicine, Houston, Texas, and the University of Texas Medical Branch, Galveston, Texas.

Induction of surface Mac-1 by inflammatory stimuli is fundamental to a broad spectrum of granulocyte adherence reactions. To study the mechanisms regulating expression and intracellular translocation of Mac-1, neutrophils were disrupted by nitrogen cavitation and subcellular organelles were isolated on Percoll gradients. Mac-1 was visualized by a sensitive Western Blot Assay made quantitative by digital imaging. Unstimulated neutrophils contained 29%, 27% and 26% of total (5.60 $\mu g/10^7$ cells) Mac-1 in beta (1.10 g/ml density granules) pre-gamma (1.07 g/ml density granules) and gamma (plasma membrane) fractions, respectively. fMLP elicited a ≥50% translocation of Mac-1 in the pre-gamma to the gamma fraction but no translocation of Mac-1 from the beta fraction and a ≤5% release of vitamin B_{12} transport protein. In contrast, PMA (1μg/ml) stimulation resulted in a ≥50% decrease in both the beta and pre-gamma Mac-1 pools concomitant with an increase in the gamma fraction and, a ≥50% release of vitamin B_{12} transport protein. Under both conditions, a 4-7 fold increase of surface Mac-1 was detectable by flow cytofluorography. Among both control and stimulated neutrophil suspensions, only 5% and 20% of Mac-1 partitioned with Triton-X 114 in beta and gamma fractions, respectively. These findings indicate that Mac-1 is contained in multiple distinct intracellular pools, and that it may exist both as an integral membrane protein and in other forms. Its "up regulation" by chemotactic factors is associated with a recruitment of a pre-gamma granular pool but does not require the participation of the beta fraction associated Mac-1 pool.