925 RESPONSE TO POLIO VACCINATION IN CHILDREN INFECTED WITH HUMAN IMMUNODEFICIENCY VIRUS.

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Vaccination strategies in children infected with human immunodeficiency virus (HIV) are controversial; live virus vaccines are generally contraindicated in immunodeficient children. 23 HIV-infected children received at least 2 doses (median = 3) of oral polio vaccine (OPV) before HIV infection was suspected, 3 others received inactivated polio vaccine (IPV). The median interval from OPV immunization to serum sampling was 2.8 years (range 1 mo to 15 yrs), the median interval from IPV immunization to sampling was 1 mo (range 1 to 4 mo). Congenital HIV infection was present in 23 and postnatally acquired infection was present in 3. Antibody to poliovirus 2 was determined by microneutralization. Protective levels of antibody (≥ 1:4) were found in 21/23 (91%) of persons given OPV (GMT = 22.9), and in 3/3 given IPV (GMT = 25.4). The stage of clinical illness correlated with the antibody titer; patients with AIDS had a GMT = 5.7, patients with ARC had a GMT = 26.9, and HIV infected apparently well children had a GMT = 84.4. Protective levels of antibody occur in the majority of HIV infected children immunized against poliovirus type 2. The potential for rare adverse events and intrafamilial spread suggests that IPV is the preferred vaccine for HIV infected children. Although no adverse events, attributable to polio vaccination, were documented in these children, further study of the effects of vaccination on the manifestations of HIV infection is needed.

▲926 HETEROGENEITY OF NEUTROPHIL (PMN) FUNCTION AND MONOCLONAL ANTIBODY (MAb) BINDING IN NEONATES AND ADULTS

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Studies have shown that PMN from adults and neonates exhibit heterogeneity of function and 31D8 MAb binding. The heterogenity of 31D8 binding correlates with PMN motile heterogeneity. MO1 MAb binds to PMN glycoproteins which allow the PMN to adhere and move normally. We further studied PMN heterogeneity in 10 neonates and 10 adults by placing PMN in the upper compartment of chemotactic chambers and allowing them to transverse 10 μm polycarbonate filters in response to C5 frag. PMN were analyzed for viability, band count and MAb binding with 31D8 and MO1. The % of non-motile (NM) neonatal and adult PMN recovered from the upper compartment after 90 min. were 7 ± 1% and 4 ± 1%, respectively ($P<0.01$). The % highly motile (HM) neonatal and adult PMN from the lower compartment were 5 ± 2% and 11 ± 2%, respectively ($P<0.01$). The viability and % bands in NM PMN were not significantly different from control PMN. The NM PMN had significantly less 31D8 and MO1 MAb binding than the HM PMN subpopulations. Neonatal NM and HM PMN had significantly less 31D8 and MO1 MAb binding than adult NM and HM PMN. These data suggest that variable 31D8 and MO1 antigen expression may contribute to differences in motile subpopulations of PMN in neonates and adults and are at least in part responsible for differences between neonatal and adult PMN motility. Decreased PMN chemotaxis in neonates may also result from a larger subpopulation of NM PMN and a smaller subpopulation of HM PMN.

927 LONGITUDINAL EVALUATION OF A HUMAN IMMUNODEFICIENCY VIRUS (HIV) POSITIVE, ANTIBODY (AB) NEGATIVE INFANT WITH AIDS.

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HIV infection in infants is usually documented by the presence of serum Ab to the virus. Individuals who are persistently viremic but without detectable serum Ab have been reported. A 4 month old infant of a parenteral drug abuser presented with failure to thrive, generalized lymphadenopathy and hepatosplenomegaly; at 6 mos of age Pneumocystis carinii pneumonitis developed. Initial evaluation (age 4 mos) revealed polyclonal hypergammaglobulinemia, $T4/T8 = 1.5$ and absolute lymphocyte count = 6460/mm^3. Results of longitudinal serological evaluations are summarized in the table.

	HIV Ab		Virus detection	
Age (mos)	ELISA	Western Blot	HIV culture	Ag ELISA
4	-	-	N.D. (not done)	N.D.
6	-	+/- (p24 only)	N.D.	+++
11	+	+ (p24 only)	+	+++
13	+	+ (p24 only)	N.D.	+++

This prolonged high grade antigenemia in the presence of low levels of specific Ab is analogous to the phenomenon of pseudotolerance observed in congenital rubella and cytomegalovirus infections and suggests that this infant was infected in utero. In infants at risk for HIV infection Ab assays alone may prove to be inadequate for diagnosis. Viral isolation and/or HIV Ag detection techniques are important tools in such situations.

●928 ANTIBODY MEDIATED ENHANCEMENT (AME) OF RESPIRATORY SYNCYTIAL VIRUS (RSV) INFECTION IN A MACROPHAGE-LIKE CELL LINE (P388D1).

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RSV bronchiolitis occurs in infants <6mos of age in the presence of maternally acquired antibody (Ab). A killed-RSV vaccine also produced serum Ab responses but recipients developed severe disease with subsequent natural RSV infection. To determine if anti-RSV Ab might contribute to the disease's pathogenesis, P388D1 cells were infected in vitro with RSV at a MOI of 0.025 in the presence of serial 4-fold dilutions of monoclonal (mc) Ab to the fusion (F), glycoprotein (G), or nucleocapsid (NC) proteins of RSV. Cells were washed after 2hr of viral adsorption and every 24h for 48h. At 48h cells were stained with a fluorescent Ab to RSV and the percent of RSV-positive cells was determined using a FACS-analyzer with Consort 30 software. Supernatants were tested for infectious virus production by plaque assay on HEp-2 monolayers. Data were analyzed by comparing Ab-treated cells with cells infected with no anti-RSV Ab present. AME was demonstrated at dilutions of 1:800 to 1:25,600 for anti-F and anti-G Ab treated cells by FACS analysis. (Anti-F:8.3-9.8% (+) vs 3.1% (+) in RSV-only, $p < 0.01$; anti-G:26.0-40.6% (+) vs 11.8% (+) in RSV-only, $p < 0.01$.) Maximal infectious virus production at 48h was increased by ≥1 log. (Anti-F at 1:6400=2.4×10^4 PFU/ml vs 1.9×10^3 for RSV only; anti-G at 1:400=2.2×10^3 PFU/ml vs 2.1×10^2 for RSV only) Cells treated with anti-NC Ab or an irrelevant mc Ab at the same dilutions showed no significant AME. This model suggests a possible role for AME in the pathogenesis of in vivo disease.

929 KINETICS OF HAEMOPHILUS INFLUENZAE TYPE B ANTIGENURIA FOLLOWING IMMUNIZATION AND NATURAL INFECTION.

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The kinetics of Haemophilus influenzae type b (Hib) antigenuria was determined in children receiving Hib vaccine and other subjects hospitalized for natural infection with Hib who presented with meningitis, epiglottitis, septic arthritis and pneumonia. Urine specimens were collected before immunization and on days (d) 1, 3, 7 and 10 post-vaccination, or prior to antibiotic therapy and at regular intervals during hospitalization. Quantitation of the PRP antigen was achieved by the use of an indirect ELISA method. All vaccine recipients were negative for PRP prior to immunization. The peak concentration of PRP excretion occured on the first day after immunization at 87% and declined to 73% by d 3 and 13% by d 10. Children excreted the highest concentration of antigen on d 1 and 3 after immunization (129 and 131 ng/ml) which declined to 0.92 ng/ml on d 7. Children with natural infection had a peak excretion on the first 3 days of hospitalization at 88% which declined to 60% by d 7 and 33% by d 9. Children with natural infection excreted higher concentrations of PRP antigen, first 3 days mean 286 ng/ml, than vaccine recipients, and excretion declined to 102 ng/ml by d 7 and 15 ng/ml by d 9. The magnitude of PRP excretion was a direct function of the type of Hib infection. Furthermore, immunization with Hib vaccine can be associated with antigenuria, and a positive diagnostic antigen test may not indicate infection in the recently vaccinated child.

†930 RESPONSE TO VARICELLA ZOSTER VIRUS (VZV) GLYCOPROTEINS (GPs) IN CHILDREN WITH LEUKEMIA (ALL).

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Three major VZV GPs [1, 2, 3] have been identified. We used a Dot-ELISA to determine the geometric mean titer [GMT] of antibody to these GPs in children with ALL who had past natural varicella (NV) or who received the live attenuated varicella vaccine(LAVV). In vaccinees with household exposure(HHE) to VZV (n=24), pre-HHE (n=19,$\bar{x}$=13 months post-LAVV) GMTs to all GPs were low in sera from those who subsequently developed lesions (n = 7)[GMTs = 33, 38, 18] and in those who did not (n = 12)[GMTs = 74, 12, 6]. Post-HHE GMTs were higher in those who developed lesions (n = 7)[GMTs = 1413, 1050, 780] compared to those who did not (n = 17)[GMTs = 127,71,47]. Vaccinees who developed zoster(Z) (n = 5,$\bar{x}$ = 15 months post-LAVV) had lower pre-Z GMTs [127, 21, 8] than vaccinees who did not (n=30,$\bar{x}$=12 months post-LAVV)[GMTs =197, 62, 40]. Post-Z GMTs showed boosts for all GPs [GMTs=1940, 1689, 404]. Unvaccinated children with ALL & NV who developed Z (n = 12,$\bar{x}$ = 20 months post-NV) had the following GMTs [269, 51, 10] compared to those who did not (n = 30,$\bar{x}$ = 42 months after NV)[GMTs = 182, 112, 20]. Post-Z GMTs showed boosts for all GPs (n=6) [GMTs = 640, 640, 570]. In summary, GMTs to GP-1 > 2 > 3. In vaccinees, at the time of HHE, the height of the antibody titer to the GPs was not helpful in predicting protection from development of breakthrough lesions, but a boost was most evident in those who did develop lesions, probably due to greater viral replication in vesicles. Vaccinees who developed Z had lower GMTs to all GPs, but especially to GP-3, compared to those who did not. Unvaccinated children with ALL & NV who developed Z had lower GMTs to both GP-2 & 3 compared to those who did not.