●895 ASSOCIATION OF KAWASAKI SYNDROME WITH ATOPIC DERMATITIS. Catherine L. Brosius, Jane W. Newburger, Jane C. Burns, Patricia Hojonowski-Diaz, Donald Y.M. Leung. Sponsored by Raif Geha, Harvard Medical School, The Children's Hospital, Departments of Cardiology and Medicine, Boston, MA 02115.

Atopic dermatitis (AD) is associated with immunoregulatory abnormalities similar to those observed in acute Kawasaki Syndrome (KS) as well as with increased susceptibility to viral infections. We investigated whether the prevalence of AD is increased among children who acquire KS. In a case-control telephone survey, 83 KS patients and 83 controls with innocent heart murmur were matched for age and time period between clinic visit and interview. The nurse-interviewer was blinded to the study's hypothesis. Cases and controls were similar in socioeconomic status, racial distribution, and number of winters in New England. Atopic dermatitis was defined as a chronic eczematoid dermatitis (separate from rash of KS) associated with personal or family history of inhalant allergy. Nine (11%) KS patients but only 1 (1%) control patient had AD; the odds of AD was 9 times greater in KS than in control patients ($p < .01$, Mantel-Haenszel). Serum IgE levels were significantly higher ($p < .02$, Mann-Whitney) in 44 unselected KS patients (median 22, range <4 to 900 IU/ml) studied 6 to 12 months after onset than in 27 controls (median <4, range <4 to 164 IU/ml). We conclude that children who acquire KS are significantly more likely than matched controls to have atopic dermatitis.

●896 REVERSE TRANSCRIPTASE ACTIVITY (RTA) IN PERIPHERAL BLOOD MONONUCLEAR CELL (PBMC) CULTURES (CXS) FROM PATIENTS (PTS) WITH KAWASAKI DISEASE (KD). Jane C. Burns, Raif S. Geha, Jane W. Newburger, Fred S. Rosen, Mary M. Walsh, Amy L. Reinhart, Alice S. Huang, Donald Y.M. Leung. Harvard Medical School, Children's Hospital, Dept. of Medicine, Boston, MA 02115.

Polymerase activity has been reported in PBMC cxs of pts with KD (Nature 323:814, 1986). To further define its template specificity and persistence after clinical recovery and gammaglobulin (GG) therapy, we studied 89 cxs from 33 KD pts and 33 controls. Of the KD pts 23/33 (70%) showed RTA above the mean + 2 SD for control pts (5.6 picomoles dTMP incorporated). The mean peak RTA for KD PBMC cx supernatants was 8.2 pmoles ± 1.8 vs. 3.7 pmoles ± 0.5 for the controls ($p < .05$). GG therapy did not prevent expression of RTA which was detected 3-8 weeks post-treatment in cxs from 4/6 KD pts. RTA was detected in PBMC from pts as late as 9 years following acute KD. Co-cultivation of PBMC or cell-free supernatants from PBMC cxs with the HUT-78 cell line yielded RTA > control mean +2 SD (3.1 pmoles) in 14/18 (78%) attempts. Analysis of template/primer specificity showed no increase in dTMP incorporation when assay conditions were altered to favor DNA pol I or terminal transferase. Duplicate assays to compare polyrA:oligodT vs. polyrC:oligodG showed comparable activity for the KD cxs. We conclude that the polymerase activity associated with the particulate fraction of supernatants from KD PBMC is consistent with RTA and can be detected late after recovery which suggests a retrovirus as the causative agent of KD.

●897 PROTECTION AFFORDED BY WC3 ROTAVIRUS VACCINE AGAINST NATURAL (PREDOMINANTLY SEROTYPE 1) ROTAVIRUS INFECTION IN A PLACEBO-BLINDED EFFICACY TRIAL. H Fred Clark, Frances Borian, Louis M. Bell, Karen Modesto, Andrew Fieo, Stanley A. Plotkin, Division of Infectious Diseases, The Children's Hospital of Philadelphia and The Wistar Institute, Philadelphia, PA 19104

WC3 rotavirus vaccine or a placebo preparation was administered to 104 infants aged 3-12 months from Sept. 1985 to Jan. 1986 in a suburban Phila. middle class population. Infants were given a single dose of WC3 vaccine ($10^{7.3}$ pfu) or placebo and were monitored daily for one week for vaccine sequelae. Active surveillance for rotavirus diarrhea was performed until June, 1986. Sequelae observed during 7 days post vaccination for vaccine (V) and placebo (P) groups were: loose stools: 12%V, 9%P; vomiting: 10%V, 6%P; fever (≥100.6°): 16%V, 11%P; irritability: 41%V, 44%P; the observed differences were not statistically significant. Rotavirus-associated diarrhea occurred between Jan. and May, 1986 in 14/55 placebo recipients and 3/49 vaccinees (protection rate = 76%, $p = <0.02$). Clinical scoring of gastroenteritis (GE) revealed an incidence of moderate to severe rotavirus disease of 11/55 in (P) recipients and 0/49 in (V) (protection = 100%, $p = <0.001$). A rotavirus strain that was isolated and identified as serotype 1 was associated with 14/17 cases of rotavirus diarrhea. Preliminary serological data suggests that serotype 1 or serotype 3 rotavirus infection also occurred in approximately 20% of infants in whom rotavirus GE was not detected, in both vaccine and placebo groups. The results suggest that WC3 vaccine protects against severe rotavirus disease and may also protect against rotavirus infection.

898 CHARACTERIZATION OF SPECIFIC ANTIBODY TO CHLAMYDIA TRACHOMATIS IN HUMAN BREAST MILK (HBM), SERUM (S) AND CERVICAL SECRETIONS (CS) AND ITS ROLE IN DISEASE OF MOTHERS AND INFANTS. Z-D. Cui, L.J. La Scolea, Jr, J.C. Cumella, and P.L. Ogra. SUNY at Buffalo, Children's Hospital, Depts. Pediatr. and Microbiol., Buffalo.

There is limited information on the protective role of antibody to C. trachomatis infection in human breast milk and other body fluids and secretions. A total of 46 patients was analyzed for the presence and characteristics of C. trachomatis antibody in HBM, S and CS. Antibody to C. trachomatis was detected by a direct ELISA method using partially purified elementary bodies of strain LGV-2 as antigen. Antibody specificity was confirmed using the appropriate absorption and blocking controls. Evidence of infection was indicated by a positive culture or presence of specific antigen. In an unprecedented finding, chlamydial antibody, primarily secretory IgA (SIgA), was detected in HBM in 7 of 33 normal women. SIgA was detected with a geometric mean titer (GMT) of 28.8. The other antibody isotypes were the following: IgG 4.79, IgM 2.4, and IgA 9.7. Serum IgG GMT in patients with recent infection were 128.8±1.8 in comparison to those with no recent infection at 6.3±8.9. Serum IgM was present, GMT 6.8±3.7, in patients with infection in contrast to those with no infection. In CS of patients with recent infection, IgG (7.9±5.4) and SIgA (15.9± 1.8) were present, and surprisingly in patients having no evidence of recent infection there was a persistence of SIgA at a titer of 8.6±3.7, suggesting repeated past exposure to C. trachomatis or the presence of antigen below the threshold of assay sensitivity.

†899 CHARACTERIZATION OF ANTIBODIES TO VARICELLA-ZOSTER VIRUS BY WESTERN BLOT. Lon Dubey, Anne Gershon, Sharon Steinberg, Philip LaRussa, Philip Oh. Dept. Pediatrics, Columbia University College of Physicians & Surgeons, NY, NY.

The immune response to varicella-zoster virus (VZV) includes antibody (Ab) to virally-specified proteins (P) and glycoproteins (GP). We analyzed Ab after chickenpox (CP), zoster (Z), and live attenuated varicella vaccine (LAVV) by western blot (WB). Responses to 2 of the 3 major GPs were identified by comparing bands on standard WBs using infected VZV cell lysate as antigen with a reference WB in which Z serum was reacted with purified GP I and GP II. A mouse monoclonal Ab to GP I confirmed its location, and all 3 GPs were visualized on silver stain of SDS-polyacrylamide gel. Preimmune sera and an uninfected cell lysate were controls. All convalescent sera from patients with CP(N=5) had antibody to GP I (bands at 99 and 92 kilodaltons [kd]), GP II (125 kd), and GP III (113 kd). In addition 5/5 had a strong band at 147 kd and 4/5 had a band at 39 kd, near the molec. wt. of VZV deoxypyrimidine kinase. Bands were well-preserved to GP I in 5 remote CP sera, but faint at GP II and III and at 147 kd. In 7/8 pts. post Z, bands at all 3 GPs and at 39 kd were darker than after recent CP despite equivalent fluorescent antibody to membrane antigen (FAMA) titers. Z sera also had bands at 51, 48, 46, and 43 which were absent or faint post CP. After 2 doses of LAVV, 10/11 vaccinees had bands at GP I. Fewer (6/11) had a band at GP II and only 3/11 had a band at GP III. However,the WBs of 4 vaccinees after breakthrough CP were similar in intensity and band location to WBs post Z. Ten leukemic children who lost FAMA Ab after LAVV and later reseroconverted without clinical illness also had WBs resembling the anamnestic profile seen post Z. Even when FAMA negative, these silent FAMA reconverters were protected from serious CP. WB is useful in distinguishing secondary from primary immune response to VZV.

†900 GENETIC COMPARISON OF ANIMAL AND HUMAN GROUP B ROTAVIRUSES USING CLONED cDNA PROBES AND TERMINAL FINGERPRINT ANALYSIS. Joseph J. Eiden, Malcolm McCrae, Ken Theil, Shigehiro Sato, Robert H. Yolken. (Spon. by Robert H. Yolken) The Johns Hopkins University School of Medicine, Department of Pediatrics, Baltimore, MD.

We have recently discovered that an antigenically distinct rotavirus (group B rotavirus) is associated with gastroenteritis (GE) in rats and humans from Baltimore, MD. We now report the comparison of the genomes of the Baltimore strain of GBR (GBR-B) with the GBR strain associated with epidemic GE in China (GBR-C). GBR cDNA clones were prepared from fecal isolates of these viruses. Dot hybridizations with these cDNA clones demonstrated nucleic acid sequence homology among GBR-B, GBR-C and bovine and porcine isolates of GBR. Northern blot techniques defined specific genome segments responsible for hybridization among the GBR. We also examined the relatedness of GBR strains by means of one-dimensional (1-D) fingerprints with T1 RNase digests of genomic RNA. Conservation of terminal sequences has previously been observed between genomic segments of different strains of standard rotaviruses (group A rotavirus), and the analysis of these terminal sequences has been employed in the grouping of rotavirus strains. 1-D fingerprints of GBR strains indicated variability rather than conservation in the sequences at the ends of gene segments obtained from individual GBR. These studies document the relatedness of GBR strains from different animal species and geographic locations. We also find that GBR are not just antigenically and genetically distinct from group A rotaviruses: GBR display structural differences in their terminal genomic sequences not noted with GAR.