685 MEASLES REVACCINATION; THE PERSISTENCE AND DEGREE OF ANTIBODY RESPONSE ANALYZED BY TYPE OF IMMUNE RESPONSE. Jaime Deseda-Tous, James D. Cherry, Mary J. Spencer, Robert C. Welliver, Kenneth M. Boyer, James P. Dudley, John M. Zahradnik and Peter J. Krause, Dept. Ped., U.C.L.A., Los Angeles, CA.

During a measles immunization campaign blood samples were collected from 203 children before vaccination. Follow up specimens were collected from 125 children 3 wks. later and from 88 children 10 mos. later. Of the 125 children with follow up study, 88 had been previously vaccinated, 10 had a history of measles and 27 denied vaccination or illness. Measles HAI antibody geometric mean titers (GMTs) were 13, (day 0), 38 (3 wks.), and 23 (10 mos.). Twenty-six of the 125 had HAI titers of ~5 prior to vaccination and showed a ≥4-fold antibody titer rise. IgM measles antibody was detected in 12 of these children after protein-A and 2-ME treatment with a GMT at 3 weeks and 10 months of 61 and 40 respectively. There was no history of measles exposure or vaccination in this group. In the remaining 14 children with initial titers < 5 no IgM antibody response was detected, and despite a 4-fold increase in HAI titer at 3 wks. (GMT 28), the 10 month titer had dropped considerably (GMT 9). Ten of these 14 children had a history of previous measles vaccination. The remaining 99 children had detectable initial titers between 5 and 320. After grouping these according to their pre-booster titers, no significant change in GMTs was observed when day 0, 3 wk. and 10 mo. results were compared. Our study suggests that HAI antibody titers in children previously immunologically stimulated against measles are not altered significantly by revaccination, even when the pre-booster titers are low or not detectable.

686 MONOCYTES FROM PATIENTS WITH GENETIC DEFICIENCY OF THE THIRD COMPONENT OF COMPLEMENT (C3) PRODUCE C3 IN VITRO. L. Peter Einstein, Prudence J. Hansen, Chester A. Alper, John S. Davis, Fred S. Rosen, Mark Ballow, and Harvey R. Colten. Harvard, U. Va., and U. Conn. Medical Schools and Children's Hospital Medical Center, Boston, MA. 02115.

A method has been described for maintenance of human monocytes in culture for up to six months. The cells synthesize and secrete the second (C2) and third (C3) components of complement and lysozyme, phagocytose latex beads, rosette with IgG or C3 coated erythrocytes, and kill L. monocytogenes.

Monocytes from two unrelated C3 deficient patients, one of whom has been described (J. Clin. Invest. 56, 703, 1975), were examined in culture. Serum from each of the patients contained less than 1% of the normal C3 concentration (not due to hypercatabolism) but monocytes from each produced C3 at approximately 25% of the normal rate when studied after two weeks in vitro. The C3 produced in vitro by monocytes from one of the patients was shown to have the molecular weight of normal serum C3 and to dissociate appropriately under reducing conditions. Monocytes from C3 deficient patients could not be distinguished from normals on the basis of morphology, rosetting with C3 coated erythrocytes, or rates of C2 and total protein synthesis. The ability of monocytes from C3 deficient patients to produce C3 in vitro is in contrast to studies of monocytes from C2 deficient humans and macrophages from C4 deficient guinea pigs in which specific biosynthetic defects for C2 and C4, respectively, persisted in vitro.

687 POTASSIUM REVERSIBLE INHIBITION OF CHEMOTAXIS BY LEVORPHANOL, Senih M. Fikrig, Phillip Chan, and Kamala Suntharalingham, Department of Pediatrics and Biochemistry, S.U.N.Y., Downstate Medical Center, Brooklyn, New York

The chemotactic responsiveness of neutrophils (PMN) is dependent to Na^+, K^+, ATPase pump and is impaired by inhibitors of the pump such as Ouabain. Levorphanol, a morphine analogue, known to effect PMN membrane functions and cause outflaw of K^+, in $1x10^{-3}$ M concentrations has also been found to inhibit chemotaxis induced by Zymosan and E. coli filtrates. Inhibition was partially reversed by addition of $1x10^{-2}$ M KCl. The action of levorphanol might be on the Na^+, K^+ activated ATPase, since it was found to inhibit the same on erythrocyte membrane fragments.

Material Added to PMN (M Concentration)	Chemotaxis Inhibition Zymosan	E. Coli Filtrate
Levorphanol ($1x10^{-3}$)	83-97%	84-96%
KCl	0	0
Levorphanol ($1x10^{-3}$) + KCl ($1x10^{-3}$)	40%	34%

688 MONOCYTE CHEMOTAXIS IN HEALTH AND DISEASE. Thomas J. Fischer, Robert B. Klein, Thomas C. Borut, Sherrie E. Gard, Gary S. Rachelefsky, and E. Richard Stiehm. UCLA School of Medicine, Department of Pediatrics, Los Angeles.

Monocyte chemotaxis was determined by an agarose technique in normal newborns, infants, older children, and adults and in selected patients. Monocytes were obtained from heparinized blood by Ficoll-Hypaque separation and exposed to zymosan treated serum as the chemotactic source. Newborn values (50 ± 33; ± 1 S.D.) were significantly different from adult values (216 ± 75, $p<.005$). An age related increase was noted (6 weeks to 2 yrs., 31 ± 25; 3 yrs. to 5 yrs., 71 ± 57; 6 yrs. to 10 yrs., 142 ± 64). Normal adult mean values were observed in the 11 to 16 year age group (214 ± 70).

Monocyte chemotactic defects have previously been described in several conditions. We have noted marked defects (>2 S.D.) in one patient with chronic mucocutaneous candidiasis and 4/29 patients with atopic dermatitis. In 8 patients on salicylate therapy, a mean decrease of monocyte chemotaxis of 25% was seen with salicylate levels >15 mgs% compared to a mean increase of 8% when neutrophil chemotaxis was concurrently measured. We also noted decreased monocyte chemotaxis in several seemingly normal subjects with absent delayed hypersensitivity skin tests to tetanus toxoid but normal in vitro lymphocyte transformation to tetanus.

These findings extend previous studies demonstrating maturational defects in human phagocytic cells, re-emphasize the possible role of defective monocyte chemotaxis in immunodeficient states, and suggest a possible mechanism for the anti-inflammatory effects of salicylates.

689 THE USE OF DIPHTHERIA AND TETANUS TOXOIDS TO ASSESS CELL-MEDIATED IMMUNITY (CMI). Stanley P. Galant, Natalie Flod, Irene Shimizu, Gale A. Granger. (Spon. by Thos. L. Nelson). Univ. of Calif., Irvine, Calif. College of Medicine, Univ. of Calif. (Irvine) Affiliated Hospitals, Dept. of Peds., Irvine, Calif.

In a previous study, we demonstrated that diphtheria-tetanus (DT) toxoids produce positive cutaneous delayed hypersensitivity (CDH) in the immunized child regardless of age. In this study, we compare the CDH response to DT and T toxoids with in vitro parameters of CMI: lymphocyte DNA synthesis and leukocyte inhibition factor (LIF) in five immunized adults. Cord blood lymphocytes were used as controls for each assay. A dose response with both toxoids compared the CDH reaction with each in vitro assay, establishing the maximum response and threshold dose which gave a positive response. All subjects had a positive CDH response to both antigens (≥5mm induration at 48 hours), positive DNA synthesis (stimulation index above 3) and LIF release (migration≤80%), while cord blood lymphocytes were usually negative to all in vitro assays. The subjects with the largest CDH reactions generally had the greatest lymphocyte DNA synthesis and lowest threshold doses. DNA synthesis was approximately ten times as sensitive an assay as CDH and 10^5 times as sensitive as the LIF technique. No difference in sensitivity was noted between DT and T toxoids. We conclude that the CDH response with either toxoid in the concentrations used is a good indicator of CMI in the immunized individual. These toxoids are particularly valuable for evaluating CMI in the young child.

690 LOCALIZATION OF C-REACTIVE PROTEIN IN SYNOVIUM OF PATIENTS WITH RHEUMATOID ARTHRITIS. Jonathan D. Gitlin, Joan I. Gitlin and David Gitlin. University of Pittsburgh School of Medicine, Children's Hospital of Pittsburgh, Department of Pediatrics, Pittsburgh, Pennsylvania.

In patients with rheumatoid arthritis (RA), persistent elevation of C-reactive protein (CRP) is related to the presence of disease activity. Since CRP can activate complement and activated complement may be involved in the pathogenesis of RA, possible synovial localization of CRP was examined. Sections of synovial tissues from 6 patients with RA, 2 patients with degenerative osteoarthritis and 4 patients with diseases unrelated to RA were studied using fluorescent antisera against CRP, IgG, IgA and fibrinogen. The results were quite clear: CRP localizes in individual nuclei scattered in the outer region of the synovial mesothelium of RA patients. CRP localization was quite unlike that of the other proteins studied and was not found in non-RA synovia. Nuclear bound CRP was not of local origin since tissue cultures of RA synovia did not produce CRP and did produce IgG and IgA. The observed synovial binding of CRP is consistent with the possibility that complement activation by CRP may be involved in the pathogenesis of RA. It is also clear, however, that specific nuclei in RA synovia contain an abnormal substance or agent which can bind CRP. The degree of nuclear binding was found to be independent of serum or joint fluid levels of CRP; thus the anomalous nuclear material is independent of CRP synthesis and could possibly represent the presence of a virus.