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CYTOMEGALOVIRUS (CMV) SEROLOGY IN SUDDEN INFANT DEATH SYNDROME (SIDS): LACK OF CORRELATION WITH CHLAMYDIA TRACHOMATIS SEROLOGY. Laura S. Hillman and Morey Gardner, Wash. Univ. Med. Sch., St. Louis Children's Hosp., Dept. Ped., and Jewish Hosp. of St. Louis, Dept. Med., St. Louis, MO.

We found *Chlamydia trachomatis* (C.t.) seropositivity to be significantly more frequent in SIDS than living controls. Because infection with CMV has been proposed to act synergistically with C.t. in producing infant respiratory infection and in utero viral infections have been postulated to play a role in SIDS, CMV complement fixing antibody titers were measured in serum from 29 SIDS, age 12.3±5.8wks, and 23 living hospitalized controls (C), age 13.5±6.7wks on which C.t. immunofluorescent antibody serologies had been performed. Cord blood (CB) from 19 black lower socioeconomic infants was similarly studied. Although 34% of SIDS were CMV seropositive, suggesting previous or ongoing maternal CMV infection, this was not different from controls. Only 4 of 11 SIDS with high C.t. titers (>1:1024) were CMV seropositive, all at low titers. Although CMV and C.t. titers were correlated ($r=.48$, $p<.05$) in cord blood, C.t. and CMV titers were not correlated in SIDS or controls. Thus, there is no evidence for a higher incidence of maternal experience with CMV in SIDS or for a post-natal association of CMV seropositivity with C.t. seropositivity.

Percent CMV Seropositive by Titer

Titer	Total		White		Black	
	C(23)	SIDS(29)	C(13)	SIDS(16)	C(10)	SIDS(13)
>1:4	26%	34%	15%	38%	40%	31%
>1:16	4%	14%	8%	19%	0%	8%
>1:32	0%	7%	0%	13%	0%	0%

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TEMPERATURE SENSITIVITY OF ECHOVIRUS TYPE 11 CAUSING CHRONIC MENINGITIS IN AGAMMAGLOBULINEMIA. David S. Hodes, Dolores V. Espinoza, Piper L. Weldy and Thomas D. Rabin, Columbia University College of Physicians and Surgeons, The Babies Hospital, Department of Pediatrics, N.Y., N.Y. 10032.

Temperature sensitive (ts) mutant viruses are associated with persistent infection (PI) of cells *in vitro*. An example of viral PI in human disease is chronic echovirus meningitis in agammaglobulinemic patients. We investigated temperature sensitivity in a strain of echovirus type 11 (ECHO 11) causing such illness. An isolate obtained early in the patient's course was ts, showing depressed replication at 40°C compared with 37°C (yield ratio, 400C/37°C=10⁻¹-10⁻³). Surprisingly strains of ECHO 11 that had not been associated with PI were even more ts (yield ratios 400C/37°C=10⁻⁴-10⁻⁶). Temperature shift experiments showed that the ts step occurred late in the growth cycle of all the virus strains. Cells doubly infected with different combinations of the virus strains failed to show enhanced replication at 40°C (lack of complementation). These results suggest that all the tested strains of ECHO 11 have a ts lesion in the same gene but that in the persistently infecting strain, the lesion is "leaky." An isolate obtained later in the patient's illness was more ts (yield ratio 400C/37°C=10⁻⁵-10⁻⁶). Again no complementation with the other ts strains was shown. These studies indicate that the relationship between the temperature sensitivity of the infecting echovirus strain and the development of chronic meningitis in the agammaglobulinemic patient is more complex than suggested by *in vitro* models.

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EXPRESSION OF CELL MEDIATED IMMUNITY TO INFLUENZA IN BREAST FED INFANTS. Ian R. Holzman, Frederick L. Ruben, Robert G. Brackett and Philip Fireman, Univ. of Pittsburgh School of Medicine, Departments of Pediatrics and Medicine, Pittsburgh, PA, and Warner-Lambert, Detroit, MI.

The suggestion of fewer viral infections in breast fed infants has raised the possibility of a role for colostral lymphocytes (COL) in the transfer of viral cell mediated immunity (CMI) via breast feeding. *In vitro* lymphocyte stimulation (LT) assay with influenza antigens was used to assess CMI. We studied 14 healthy mother-infant pairs 1-6 days after delivery and 8 pairs were followed up 1-4 months later. Peripheral blood lymphocytes (PBL) and COL were tested for LT using ether extracted influenza antigens (A/Tex, A/Brazil, and B/Hong Kong). Lymphocyte responsiveness to PHA was confirmed. A pos. influenza response was defined as a stimulation ratio ≥2.0. After delivery 12/14 PBL from mothers and 5/13 COL were pos. to one or more influenza antigens; at follow-up 8/8 maternal PBL were pos. After delivery 7/14 infant PBL were pos. and of 8 infants not yet breast fed, 5 were pos. At follow-up 3/8 infants were pos., all 3 of whom had been negative after delivery. No respiratory infections were recognized in any of the study infants. CMI to influenza was demonstrated in many maternal PBL and COL samples after delivery and in all PBL at follow-up. While 3 infants acquired CMI to influenza after breast feeding, 5 infants had CMI after delivery and prior to breast feeding. These studies suggest that breast feeding may confer CMI to the infant but also imply that another maternal mechanism for an earlier acquisition of CMI exists.

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PASSIVE IMMUNIZATION OF INFANTS AGAINST HAEMOPHILUS INFLUENZAE TYPE B (HIB) BY MATERNAL VACCINATION. Richard A. Insel, Marvin S. Amstey, Porter Anderson, Michael E. Pichichero, and David H. Smith, University of Rochester, Departments of Pediatrics and Obstetrics and Gynecology, Rochester, New York.

Prior to a planned conception, 24 women were immunized with purified HIB capsular polysaccharide to attempt to extend their offspring's period of passive antibody protection against systemic HIB disease. All women had an antibody rise. Geometric mean titer prior to immunization was 1.2 µg/ml and at 1, 7-10, and 19-27 months after immunization was 35 µg/ml, 23 µg/ml, and 16 µg/ml respectively as measured by radioantigen binding. Most post-immunization antibody was of the IgG class as determined by an enzyme-linked immunosorbent assay. However, there was a wide range of post-immunization titers (2.7-310 µg/ml at 7-10 months post-immunization). The low magnitude antibody responders also had low IgG antibody levels that were not boostable. The offspring of 6 women, born 1½ to 17½ months after immunization, had antibody titers markedly higher than those of age-matched infants of unvaccinated women. Their titers persisted above the estimated protective level through 6-12 months of age. Four infants, all born to low-responder women, had titers similar to natural antibody titers. These results suggest that the interval of passive antibody protection can be extended. This approach may be especially useful in populations highly susceptible to HIB and may be applicable to the prevention of other childhood and neonatal infections.

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DETECTION OF CYTOMEGALOVIRUS (CMV) BY ENZYME LINKED IMMUNOSORBENT ASSAY (ELISA). Richard F. Jacobs, Russell W. Steele, Univ. Ark. Med. Ctr. Div. Ped. Infect. Dis., Little Rock, AR.

Transfusion induced CMV infection is a significant problem in neonates and immunosuppressed patients. At present, there is no useful means to detect those units of blood that are capable of transmitting CMV. Therefore, we developed a double-sandwich ELISA to detect CMV antigen in blood; an indirect ELISA was developed to detect anti-CMV IgG antibodies. Applying these tests to 125 random blood donor samples, we found that 81 of 125 (65%) contained anti-CMV IgG. Anti-CMV antibody (IgG) titres determined by ELISA correlated well with titres determined by complement fixation ($r=0.95$, $p<0.001$) and by indirect hemagglutination ($r=0.96$, $p<0.001$). In 8 of 81 (6.4%) samples that contained anti-CMV antibody, CMV antigen was detected by ELISA; this is similar to the 4-6% rate of post-transfusion CMV infection noted by others. In addition, CMV antigen was detected in 5 of 16 selected clinical samples; each of these 5 patients had active CMV infection subsequently confirmed by viral isolation (4) or by serology (5). Of these 5 patients, 2 had congenital infection, 2 had received multiple transfusions, and one had received blood containing CMV antigen. We conclude that the ELISA technique is a sensitive technique for detecting CMV antigen. Further studies are indicated to determine its utility as a diagnostic test and as a test for screening prospective blood donors.

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ANTIBIOGRAMS - MONITORING ANTIBIOTIC RESISTANCE. R. F. Jacobs, T. Yamauchi, K. D. Eisenach, Departments of Pediatrics and Pediatric Pathology, University of Arkansas Medical Sciences and Arkansas Children's Hospital, Little Rock, Arkansas.

Monitoring of antibiotic resistance via disc diffusion or measurement of minimum inhibitory concentration is vital to optimal medical care. The compilation of these patterns of resistance and publication as antibiograms are essential in allowing physicians to intelligently administer proper antibiotic and drug regimens. We have monitored specific resistance rates plus trends of drug resistance in a children's hospital.

Of major importance is the decrease of ampicillin-sensitivity by multiple microorganisms including *Hemophilus influenzae*, *Salmonella*, *Shigella* species, and several gram-negative bacteria involved in pediatric urinary tract infections. In 1979, 78% of the *H. influenzae* isolates were sensitive to ampicillin, as opposed to 1977 when 82% were ampicillin-sensitive. Even greater changes were seen in the *Shigella* species, in 1973, 100% of *S. flexneri* were ampicillin-sensitive and by 1979 only 37% were sensitive. *Escherichia coli* has gone from 85% to 46% in the same time span. Gram-negative microorganisms have undergone similar patterns with gentamicin. *Klebsiella* and *Enterobacteriaceae* have gone from 100% sensitive to gentamicin in 1973 to only 62% and 58% respectively in 1979. Other antibiotics analyzed included: penicillins, semi-synthetic penicillins, cephalosporins, chloramphenicol, erythromycin, and trimethoprim/sulfamethoxazole. The use of pediatric antibiograms should be routine in pediatrics.