968 LYSIS OF VARICELLA ZOSTER VIRUS (VZV) INFECTED CELLS BY ANTIBODY-DEPENDENT CELLULAR CYTOTOXICITY (ADCC). Steven L. Shore, G. David Cross, and Theresa L. Cromeans. Immunology Division, Center for Disease Control, Atlanta.

Human foreskin fibroblasts acutely infected with VZV were exposed to VZV antibody-positive human serum and human mononuclear cells (MC) in a 6-hr ^{51}Cr release assay. Specific lysis could be detected by 60 min and was proportional to the effector cell:target cell ratio. The reaction was temperature dependent, proceeding optimally at 37°C. Only VZV antibody-positive serum mediated the reaction, and uninfected targets were not lysed. MC alone had little or no lytic activity. Median antibody titers of 1000 were noted in the serum of normal adults without a history of zoster. The antibody was of the IgG class as indicated by its adsorption with S. aureus containing protein A, its presence in high titer in immune serum globulin and zoster immune globulin, and its quantitative placental transfer. A requirement for effector cell IgG-Fc receptors was shown by blocking of ADCC by protein A and inhibition of effector cell activity by heat-aggregated gamma globulin. Lymphocytes were more effective as killer cells than were monocytes or polyorphonuclear leukocytes. ADCC was able to lyse cells earlier in the VZV infectious cycle than antibody-dependent complement-mediated lysis. These results are the first demonstration of ADCC against VZV-infected cells and suggest a mechanism whereby IgG antibody aids in prophylaxis of infection or reduces the severity of exogenous reinfection.

969 PHYSIOLOGIC MODIFICATION OF THE ACTIVITY OF THE ALTERNATIVE PATHWAY OF COMPLEMENT (APC) IN THE NEONATE: A POTENTIAL MECHANISM FOR DEFECTIVE OPSONIZATION. Roger E. Spitzer, Ann E. Stitzel, Ellen C. Blinder, Julia A. McMillan, Larry Consenstein, Leonard B. Weiner and David A. Clark. SUNY, Upstate Medical Center, Department of Pediatrics, Syracuse, NY.

Under ordinary conditions, C3 and factor B interact constantly in order to generate C3b and prime the APC. This interaction is closely regulated by the activity of two other serum proteins, β1H and C3bINA. In the older child and adult, this regulation results in a serum ratio of C3 + B/β1H + C3bINA (determined by RID) which is surprisingly constant at 1.06 + 0.08 (mean + S.D.; n=153). In the newborn, serum levels of all 4 of these proteins are normally reduced. Despite this physiologic hypocomplementemia, umbilical cord blood shows a much higher ratio of these components with nearly 70% (n=200) of neonates being greater than 1.3. These data suggest that the activity of β1H and C3bINA in regulating the interaction of C3 and factor B is more potent in the newborn. Measurement of specific functional β1H activity revealed little difference between the adult and newborn in the number of β1H molecules required to inactivate 1 effective C3b molecule. By contrast, specific C3bINA functional assays demonstrate that the newborn requires 50-75% fewer C3bINA molecules than the adult in order to inactivate 1 effective C3b molecule. Thus, the neonate appears to have an altered form of C3bINA which is 2-3 times more potent than its adult counterpart. This altered molecule could allow for less C3b deposition on an offending agent and, therefore, less efficient opsonization.

970 EFFECT OF DIETARY IRON ON CELLULAR IMMUNE FUNCTION. Robert M. Suskind, Solo Kuvibidila, and B. Surendra Baliga. U. of South AL, Dept. of Peds., Mobile, AL.

The effect of inadequate dietary iron uptake on cellular immune responses was studied in weanling female mice C57BL/6, which were fed either control (C), iron deficient (DEF) or pairfed (PF) C diets. The DEF mice showed a significant decrease in the delayed cutaneous hypersensitivity reaction to dinitroflurobenzene (DNFB) as measured by the inflammatory skin response ($p<.001$) and ^{125}I-dUR incorporation into sensitized ears ($p<.001$). When a single dose of imferon was given to DEF mice prior to recall, the ^{125}I-dUR incorporation was restored to greater than normal levels ($p<.001$), a response which was DNA, RNA, protein synthesis dependent (Table 1).

TABLE 1:

	C	PF	DEF	+Imferon
Inflammed Area cm^2	2.7±0.2*	2.6±0.2	1.5±0.3	---
Ratio of CPM (^{125}I-dUR)Rt/Lt	7.8±1.4	---	3.2±0.7	17.4±2.2

In vitro splenic lymphocyte response to T-cell mitogens Con A and PHA was significantly decreased in DEF cells ($p<.001$) (Table 2).

TABLE 2: ^{3}H-Thymidine Incorporation (CPM) 1x10^3

	C	PF	DEF	Repleted
Con A	43.1±2.1	42.8±2.7	16.2±2.4	23.4±1.2
PHA	36.5±2.6	46.1±2.1	24.3±1.7	28.0±2.1

When the cytotoxic capacity of T-lymphocytes or peritoneal cells to kill ^{51}Cr labelled P815 target tumor cells were compared, the cells from the DEF group had a killing capacity which was 50% of the C. The above data strongly supports the contention that iron deficiency significantly depresses the in vivo and in vitro CMI response.

* M ± SEM.

● **971** IMPAIRED PRODUCTION OF IMMUNE (PHA-INDUCED) INTERFERON IN NEWBORNS IS DUE TO A FUNCTIONALLY IMMATURE MACROPHAGE. Stephen Taylor, Yvonne Bryson (Spon. by James Cherry), UCLA Sch. Med., Dept. Pediatrics, Los Angeles.

Previously demonstrated deficiencies of immune PHA-induced interferon (IF) by leucocytes of normal newborns could be the result of a functionally immature T cell, macrophage (macro) or both. We therefore studied PHA-induced (IF) production by macros and T cells from 23 cord and 13 adult bloods. After Ficoll-Hypaque (FH) separation of mononuclear (mono) cells, macros were separated by adherence and cultivated for 7 days. T cells (separated by E-Rosettes) were then added, macro-T cultures were stimulated with PHA, and supernatants harvested at 48 hrs. IF was assessed by a microassay using Trisomy 21 cells and encephalomyocarditis virus challenge. PHA stimulated macro-T cultures of autologous and nonautologous cells (9 adults) showed enhanced IF production (GMT 121±1 SD) as compared to FH mono cells (GMT 42±1 SD). Combinations of adult and cord cells were PHA-stimulated. No IF was detected in supernates from PHA-stimulated FH cord cells alone or macro-T cord combined cultures. Similarly combined cord macro and adult T cells produced minimal IF (GMT 14±3 SD). Cord T cells with adult macros showed enhanced IF production (GMT 195±2 SD). These results indicate that the cord macrophage is primarily responsible for the poor IF PHA induced response since the newborn T cell functions normally in the presence of adult macros. Thus a combination of impaired macro IF production and increased susceptibility of neonatal macro to viral replication may explain the enhanced severity of neonatal viral infections.

972 DEVELOPMENT OF APPARENT GRAFT VS HOST (GVH) REACTION FOLLOWING A MATERNAL PLATELET TRANSFUSION IN AN IMMUNOCOMPETENT INFANT. Jeffrey E. Thompson, James A. Stockman, III, Frederick R. Davey, Robert K. Kanter and Roger E. Spitzer. SUNY, Upstate Medical Center, Depts. of Peds. and Clin. Path., Syracuse, New York.

An otherwise healthy term newborn received non-irradiated platelets in the first day of life for isoimmune thrombocytopenia. At 10 days of age the child developed an ultimately fatal disease characterized by rash, pneumonitis and bone marrow depression. Although no peripheral lymphocyte chimerism was detected, skin biopsy revealed a vacuolated basal cell layer with pyknotic nuclei, dermal layer lymphocytic infiltrate and edema, epidermal spongiosis, occasional infarcts, and many eosinophilic bodies with satellitosis. Bone marrow biopsy and autopsy findings were also most consistent with GVH. Immunologic evaluation revealed an adequate number of neutrophils. Complement levels were normal. The child's IgM and IgE levels were normal and her B cells could produce IgM after PWM stimulation. Peripheral lymphocytes had both maternal and paternal HLA antigens. Rosette techniques revealed 40% T cells and 10% B cells, both within the low normal range for age. In the one way MLR, the patient's cells responded well to various donors except to the mother. PHA stimulation was also within normal range. Thus there was no evidence to suggest a defect in the child's immune system. The appearance of a GVH reaction in an apparently immunocompetent infant raises the question of irradiating all blood products containing viable lymphocytes before administration to newborns.

973 Serum Zn and Cu Concentrations During Infection in the Premature Infant. E.E. Tyrala, J.I. Manser, N.L. Brodsky, C. Crawford. Temple University School of Medicine, St. Christopher's Hospital for Children, and Albert Einstein Hospital(North), Philadelphia, Pa. (Spons.- V.H. Auerbach)

Serial serum zinc (Zn) and copper (Cu) concentrations were measured in 4 intravenously fed, non-Zn, non-Cu supplemented premature infants all of whom had culture proven bacterial septicemia in the 3rd to 4th weeks of life. (Mean G.A. = 28.7 wks., mean birth wt.=1115gms.). Mean serum Zn and Cu concentrations in the infected group were compared to 9 non-Zn and non-Cu supplemented, age matched, IV nourished controls.

Age		2 wks. (Pre-infect.)	3-4 wks. (Inf.)	5-6 wks. (Post-infect.)
Zn	Infected	88	32	56
µg/dl	Control	91	81	78
		ns	p<.01	ns
Cu	Infected	31	53	34
µg/dl	Control	43	56	52
		ns	ns	ns

The data suggests that the premature infant, similar to the adult, (J Infect Dis 126:77, 1977), is capable of sequesting Zn as a host response to infection from early in gestation. Serum Zn concentrations may be helpful in monitoring onset of infection and adequacy of antibiotic therapy in the newborn. No statistically significant changes in Cu in response to infection were seen.