896 IMMUNE RESPONSES FOLLOWING NATURAL STING BY HONEY BEE (HB) AND YELLOW JACKET (YJ) INSECTS. Mark Ballow, James Brakoniecki, Louis Mendelson, Kenneth Sparks, and Edward Chu (Spon. by Arnold J. Altman) Univ of Connecticut Health Center, Dept. of Pediatrics, Farmington, CT.

Serum was obtained from 17 individuals within 48 hours (baseline immune status) and 3-4 weeks after HB or YJ sting. None had a prior systemic reaction or had received desensitization therapy. Specific IgG and IgM antibodies (Ab) to YJ venom and HB phospholipase A (PL-A) were measured by ELISA. Specific IgE (RAST) and total serum IgE (PRIST) were also measured. Of the individuals stung by HB (3), none had a physical reaction or measurable IgG, IgM or IgE specific Ab. Only one person developed an immune response: IgG Ab to HB-PL-A. Fourteen people were stung by YJ. One individual who had high baseline IgG, M and E Ab to YJ had a systemic reaction. Four individuals with preexisting IgM Ab (geo. $\bar{x}$ · 310 ELISA units vs non-stung controls, 75.9 units, $p<0.001$) had a large local reaction. Two of these 4 also had preexisting IgG Ab; 3/4 further increased their serum IgG Ab to YJ venom 3 weeks post-sting (avg 4.3-fold). The IgM did not change. Nine people who had no physical reaction to YJ sting had no preexisting IgG or IgM Ab to YJ venom. The serum IgG Ab increased in 4/9 (avg 12.1-fold) 3 weeks post-sting. Only 1/9 had an increase in the serum IgM and IgE Ab to YJ. Elevated preexisting IgM and IgG Ab may predispose individuals to large local reactions. IgG and IgM specific immune responses to HB and YJ venoms can occur in individuals following natural sting.

897 PNEUMOCOCCAL VACCINE (PnVx) IN SICKLE CELL DISEASE (SCD): IgG AND IgM ANTIBODY RESPONSE. Douglas J. Barrett and Arthur J. Ammann. Departments of Pediatrics, University of Florida, Gainesville, and University of California, San Francisco.

The IgG and IgM class-specific antibody response to PnVx in SCD patients was investigated since pneumococcal infection after PnVx occurs in some SCD patients and since patients with functional asplenia (including SCD) may not be able to form normal levels of protective antibody and/or switch from IgM to IgG class of antibody in response to certain antigens. Both normals and SCD patients formed IgG as well as IgM antibody by ELISA to each pneumococcal type tested. The level and predominant class of antibody varied between serotypes.

Mean Fold-increase ($\log_2$) in Antibody Titer to PnVx Serotypes:

Serotype:	3		6		18		19		23	
Ig Class:	G	M	G	M	G	M	G	M	G	M
SCD (n=11)	2.8	3.4	1.4	1.9	2.7	2.4	1.2	2.4	2.1	3.3
Normal (n=9)	3.3	4.2	2.6	2.3	3.0	4.4	2.0	3.0	3.0	3.8

Although statistically significant differences were not found, slightly lower responses were noted in SCD patients, especially to type 6, a serotype frequently associated with PnVx failure. Further testing in patients with PnVx failure will be necessary to determine if inadequate IgG or IgM response contributes to lack of protection in these instances.

898 SUPPRESSOR/HELPER CELL FUNCTION IN CHILDREN WITH AUTOIMMUNE DISEASE. Douglas J. Barrett, Arthur J. Ammann, Diane Wara and Elia M. Ayoub. Departments of Pediatrics, University of Florida, Gainesville and University of California, San Francisco.

Immunoregulatory cell function was tested prior to therapy in 3 children with systemic lupus (SLE) and 4 with mixed connective tissue disease (MCTD). Diagnoses were made by the American Rheumatism Association's clinical criteria and elevated anti-DNA with low C_3, C_4, and/or CH_{50} in SLE or elevated anti-ENA of RNP specificity in MCTD. Suppressor/helper function was evaluated by comparing pokeweed mitogen-induced IgG and IgM secretion in individual cultures of patients' lymphocytes (PBLs) with that produced in co-cultures of PBLs of patients and normals.

SLE patients produced more IgG (mean ± S.E. = 10,100 ± 6100 ng per 2 x 10^6 cells) in individual cultures than did 10 normals (1,450 ± 2,100 ng); IgM production was similar in the two groups. Co-cultures of SLE PBLs with normals suggested normal immunoregulation in one patient, loss of suppressor function in one, and excess helper function in the other. Three of 4 MCTD patients produced normal amounts of IgG (3980 ± 1780 ng) and normal immunoregulation was found in co-culture. One MCTD patient had markedly low IgG (220ng) and IgM (70ng) production and this patient excess suppressor function in co-culture.

Our results suggest that children with SLE may produce more IgG in response to pokeweed mitogen than normals and a variety of immunoregulatory abnormalities may be found in children with active, untreated autoimmune disease.

899 IMMUNODEFICIENCY IN CONGENITAL HEART DISEASE (CHD): Douglas J. Barrett, Willa H. Drummond, Ernest A. Keil, Drew Weigner and Elia M. Ayoub. Department of Pediatrics, University of Florida, Gainesville.

DiGeorge Syndrome (DGS) consists of the association of CHD, immunodeficiency, and hypoparathyroidism. Since recent data suggest that DGS is more common than usually appreciated, we prospectively tested immune function during the first 3 months of life in 22 infants referred for evaluation of CHD and 7 age-matched controls. CHD patients as a group had T-cell rosettes (TCR) lower (mean ± SE = 48% ± 5) than age-matched controls (64% ± 4) or older normals (71% ± 2). Eight of the 22 CHD infants had TCR below 2 standard deviations from the mean for age-matched controls (<40%). Six of 21 CHD infants had low peak phytohemaglutinin (PHA) responses (<40,000 cpms); however the mean peak PHA response for CHD patients (78,000 cpms) was not different from age-matched controls (86,100 cpms) or older normals (98,000 cpms). All CHD patients had B-cell numbers comparable to controls (8-20%). When grouped by cardiac abnormality, 8 of 12 infants who had lesions previously described in DGS (interrupted aortic arch, aortic atresia/hypoplasia, truncus arteriosus, tetralogy of Fallot, VSD), had low TCR or PHA, with 4 of the 8 having both low TCR and PHA. In contrast, 4 of 10 infants with CHD not associated with DGS had some evidence of immunodeficiency, with boarderline low TCR only in 3 of the 4.

These findings suggest that about 75% of patients with congenital heart disease, especially those with aortic arch abnormalities or truncus arteriosus, may have laboratory evidence indicative of depressed immune function.

900 CHEMOTACTIC RESPONSES OF VARIOUS DIFFERENTIATIONAL STAGES OF NEUTROPHILS FROM CORD AND ADULT BLOOD. A. Boner, B.J. Zeligs, J.A. Bellanti. Georgetown Univ. Sch. Med., Wash., D.C.; Univ. Verona, Verona, Italy.

The chemotactic response (CTR) of cord blood neutrophils (CN) was compared to adult blood neutrophils (AN) in an agarose plate assay using various concentrations of zymosan activated serum (ZAS) as chemoattractant. The total numbers of cells responding (mean ± SEM) are:

	% ZAS				
	1%	10%	25%	50%	100%
CN(CTR)	3 ± 1	12 ± 4	32 ± 10	138 ± 42	187 ± 43
AN(CTR)	8 ± 5	87 ± 18	226 ± 30	435 ± 76	394 ± 61

The CTR for AN > CN at all % ZAS. A method was developed to measure mean migrating distances (MMD) of each differentiational stage of CN and AN. The MMD (± SEM) in mm for band (B); bi-lobed (BL); multi-lobed (ML) neutrophils were 0.58 ± 0.07, 0.71 ± 0.06, 0.64 ± 0.04 for AN vs 0.41 ± 0.02, 0.43 ± 0.05, 0.40 ± 0.04 for CN at 100% ZAS. The MMD decreased with 50%, 25% ZAS but all MMD of AN > CN. The ratio of the MMD of PMN (BL + ML) to bands from AN was 1.2 ± 0.08 indicating an increase in CTR with neutrophil differentiation. This could contribute to the differences in CTR of CN vs AN since 31% of CN are bands vs only 8% of AN. Random migration and adherence assays showed no differences in CN vs AN. These results show that the CTR of neutrophils increase with the age of the host as well as maturation of the responding cells. NIH Grant 1P50AI-15321-03.

901 ANTIGEN-SPECIFIC INDUCER ACTIVITY IN HUMAN LEUKOCYTE DIALYSATES BINDS TO ANTI-V_H BUT NOT ANTI-V_L CHAIN ANTIBODY WHILE ANTIGEN SPECIFIC SUPPRESSOR ACTIVITY BINDS TO ANTI-V_L CHAIN ANTIBODY. W. Borkowsky & H.S. Lawrence, NYU Med. Ctr., NYC.

We have reported on Leukocyte Migration Inhibition (LMI) as a reliable in vitro assay of antigen-specific activity in human leukocyte dialysates (DLE) containing Transfer Factor. We have shown that treatment of nonimmune leukocytes with specific DLE renders them immune to the antigen and DLE treatment of immune leukocytes renders them nonimmune to the antigens to which the DLE donor was sensitive. These properties are designated "Inducer DLE" (I-DLE) and "Suppressor DLE" (S-DLE) respectively (J.I. 4/79). Specific activity of I-DLE binds to specific antigen-immunoabsorbent but not to specific antibody-immunoabsorbent (J.I. 2/81). We now suggest that I-DLE AND S-DLE are separate moieties since 1) the antigen specific activity in I-DLE binds to an affinity purified anti-V_H antibody but not to anti-V_L chain antibody (prepared by Givol) while S-DLE binds to the anti-V_L chain antibody; & 2) that S-DLE does not bind antigen; preliminary results suggest that it binds IgG. This suggests that antigen specific activity in I-DLE may represent a dialysable fragment of antigen binding sites of immune T-cells; I-DLE and S-DLE may be representative of a network system.