● 1480 LYMPHOCYTE SUBPOPULATIONS IN LIPOID NEPHROSIS (LN)

Andrew J. Aronson, Mark S. Schiffer, Daniel Levitt, (Spon. by Lawrence M. Gartner) University of Chicago School of Medicine, La Rabida Children's Hospital, Chicago

Previous studies in LN have suggested an immunologic pathogenesis. We have characterized B and T cell subpopulations from peripheral blood lymphocytes(PBL) of 10 patients with LN. B lymphocytes displaying different isotypes were detected by staining with fluorescein(Fl) and Rhodamine(Rh) conjugated, affinity-purified goat anti-human μ, δ, γ, or α. T lymphocytes were evaluated using monoclonal mouse anti-human T cell reagents(OKT3 = total T, OKT4 = inducer T, OKT8 = suppressor/cytotoxic T). Plasma cells(PC) were stained after fixation with Fl or Rh goat anti-human μ, γ, or α.

	MEAN % PBL									
	SURFACE MARKERS							CYTOPLASMIC Ig		
	μ	δ	γ	α	T3	T4	T8	μ	γ	α
relapse	21.9	12.5	0.6	1.7	50.6	32.4	17.9	3.6	0.2	3.3
remission	10.2	8.2	1.7	0.8	61.2	37.4	23.7	0.3	1.3	0.8
normals	8.5	7.1	1.7	1.2	63.5	42.2	20.7	0.8	0.9	1.1

Before therapy all patients had elevated percentages of $sIgM^+$ B lymphocytes and IgM and IgA PC in blood. After treatment percentages of PC and B lymphocytes returned to normal. Distribution of T cell subpopulations were normal in pre- and post-treatment populations.

We conclude that children with LN possess elevated numbers of B cells and PC which may indicate spontaneous activation of the humoral immune system. After treatment normal B and PC numbers are seen. Such elevations of B cells are not due to an abnormal distribution of T cell subpopulations.

1481 THE EFFECT OF TRANSITION FROM CHRONIC Na DEFICIT TO Na EXCESS IN THE DEVELOPING RAT.

Abraham Aviv, Tatsuharu Kobayashi, and O. Robert Levine, New Jersey Medical School, Dept. of Pediatrics, Newark, New Jersey.

Littermate male S-D rats were maintained on either low Na (LS) or normal Na (NS) intake between ages 3-7 weeks and then switched to high Na intake between ages 7-9 weeks. The LS group showed retarded growth between 3-7 weeks of age, and a massive weight gain within a week after the switch to the high Na intake. The ECF compartment, determined by the distribution (4 hour phase) of injected ^{22}Na, and the exchangeable ^{23}Na (Exc. Na) were measured before the transition to the high Na diet, as well as at 8 and 9 weeks of age. The ECF (table; ml/kg BW; $\bar{x} \pm$ SEM) at age 7 weeks was reduced in the LS group. Both the NS and LS groups manifested expansion of their ECF a week after the switch to the high Na intake. This compartment was, however, greater in the LS group. At age 9 weeks, despite a continuous high Na intake, the ECF of the NS rats returned to its original volume, whereas that of the LS group was still increased. Similar changes were noted in Exc. Na. These alterations were not shown in adult rats. It is concluded that in the developing rat chronic Na deficit alters the adaptation to Na excess in later life. Since the kidney is a major regulator of the ECF and body Na, this phenomenon may relate to modification of the renal handling of Na.

	7 weeks	8 weeks	9 weeks
LS	273.6 ± 3.6 (6)	343.9 ± 2.6 (8)	323.6 ± 3.7 (7)
NS	305.5 ± 4.9 (6)	323.6 ± 6.1 (8)	304.7 ± 4.7 (7)
P Values	< 0.001	< 0.02	< 0.01

1482 INTERACTIONS OF HUMAN, CULTURED KIDNEY CELLS WITH THE COMPLEMENT (C) SYSTEM.

Patricia J.Baker, Byron P. Croker, Stephen G.Osofsky. Duke Univ. Med.Ctr., Depts. of Pediatrics and Pathology, Durham,N.C.

Previously we have shown that C activation takes place when heat-killed, cultured human kidney cells are incubated in normal human serum. C activation initiated by 0.5 x 10^7 cells / 50 μl permitted moderate C4, C2, C3 and C5 consumption of hemolytic activity without detectable loss of C1 and no reduction in C6 activity. At 2 x 10^7 cells / 50 μl serum, some loss of C1 and C6 hemolytic activity was noted. C4 and C2 consumption could not be prevented by blocking primary C pathway through prior EGTA chelation of serum.

Both living and heat-killed kidney cells were incubated with normal serum and then examined for surface bound C components using immunofluorescence techniques. The heat-killed kidney cells were strongly positive for C3 which was distributed in a diffuse, speckled pattern over the entire cell surface . Dead cell suspensions also showed weak IgG and C1q immunofluorescence but were negative for surface albumin, C5 and β1H. In contrast, living cell suspensions treated in a similar manner showed only occasional cells immunofluorescent positive for C3, IgG or C1q and all cells were negative for albumin, C5 and β1H. Viability stains run concurrently revealed that the few C3 positive cells in living cell suspensions belonged to the small, nonviable cell subpopulation. These data indicate that dead kidney cells can initiate limited C activation resulting in preferential C3b opsonization of dead, but not living cells.

1483 FUROSEMIDE (F) ANTAGONISM OF INDOMETHACIN (I) EFFECT ON RENAL FUNCTION,

M.V.Betkerur, T.F.Yeh, A.K.Wilks, J.Singh and R.S.Pildes, Cook County Hosp., Dept. of Pediatr., Univ. of Ill., Chgo. Ill.

To evaluate if (F) would prevent the adverse renal effects of I.V.(I), 9 premature infants with PDA were randomized into 2 groups; 4 received I(0.3mg/kg) alone and 5, I and F (1mg/kg I.V.) simultaneously. There were no sig. differences between the groups in B.W.(mean+S.D. 1179+456 vs 1021+282gm), gest.age(31.3+2.2 vs 29.5+1.9wks), postn. age (9.0+2.9 vs 11.6+0.5d.), clinical cardiovascular status, pH, FiO_2, pO_2 and pCO_2.

Group I (hrs)	Urine Output (ml/kg/hr)	GFR (ml/min/1.73m^2)	FeNa %	C_{H_2O} (ml/min)
Pre 0-24	2.25+0.42	9.47+2.32	2.17+1.26	0.36+0.23
Post 0-12	0.77+0.54**	5.85+2.34**	1.24+0.77	0.16+0.16
12-24	0.70+0.76**	7.97+4.86	0.59+0.44*	0.12+0.08*
Group I+F				
Pre 0-24	1.65+0.53	6.14+3.52	2.30+1.09	0.17+0.12
Post 0-12	1.63+0.76	7.21+4.43	3.04+1.82	0.32+0.30
12-24	1.10+0.20	6.60+2.72	0.64+0.27*	0.16+0.09

Three in I and 4 in I+F responded with closure of PDA. There were no sig. differences in renal function between the groups. However, when compared with baseline, sig. decreases in urine output, GFR and $C-H_2O$ were seen when I was used alone but not when I+F were given simultaneously. The addition of F did not affect FeNa. These changes suggest that F may be useful in preventing oliguria by overriding the action of I.

*p <0.05 **p<0.01 (paired)

1484 RENAL FUNCTIONS FOLLOWING ACUTE HYPOVOLEMIC SHOCK IN DEVELOPING BABOONS.

Rama Bhat, Eunice John, Burt Braverman, Tonse N.K. Raju, Leonardo Malalis, Parvin Justice, Morton Schulman, Dharmapuri Vidyasagar, University of Illinois, Department of Pediatrics, Chicago, Illinois.

Renal functions, cardiac output(CO) and colloid oncotic pressure (COP)were measured during control (C) hypovolemic shock (HS) and recovery (R) in baboons (2-8 weeks). Heart rate, mean blood pressure (MBP) and tissue pH (tpH) were monitored continuously. Shock was induced by bleeding till the MBP dropped from 81 to 41 mmHg. CO, blood gases (ABG), COP, Hct were measured every 15'. GFR, PAH and osmolar clearance, FEna were measured during C, HS, R. GFR, CPAH and COSM decreased significantly during HS ($p<0.5$) but recovered to baseline following reinfusion. In newborns both Fena and Uv decreased by 85%. Whereas in the older baboons the decrease was 52 and 85% respectively. CO and MBP decreased by 50%, but recovered following reinfusion. These findings suggest that: 1) The decrease in GFR and Uv is due to decrease in CO and MBP; 2) The decrease in FEna in the presence of decreased GFR, PAH, COSM suggests an intact tubular function; 3) All the parameters recovered following infusion. In conclusion, the changes in renal function in early HS (45') are transient and reversible following reinfusion. *(p<.05)

	GFR ml/min/kg	CPAH ml/min/kg	COSM ml/min/kg	FEna% ml/min/kg	Uv ml/min/kg
C(30')	1.98±0.54	5.71±1.5	0.25±0.08	13.56±11.0	0.27±0.10
HS(45')	0.52±0.24*	0.95±0.31*	0.03±0.01	2.27±1.0	0.08±0.06
REC(45')	2.07±0.39	6.75±1.27	0.22±0.10	9.76±7.0	0.21±0.10

1485 NEPHROTIC SYNDROME IN A CHILD WITH HEMOPHILIA AND PERSISTENT HEPATITIS:A PATHOGENETIC HYPOTHESIS.

M.Borzani,G.Migliavacca,R.Longhi,(spon.by F.Sereni) Università di Milano,Clinica Pediatrica I°,Milan,Italy.

The developement of immunocomplex-related pathology during type B viral hepatitis is well known.HBsAg positive subjects can be affected by glomerulonephritis,usually of the membranous type; HBsAg-antiHBs immunocomplexes (I.C.)have been found in these patients'kidneys. Some authors believe that other I.C. may cause this pathology (Kleinknecht,1979). Our patient is a 3-years old boy polytransfused because of hemophilia A,with chronic active hepatitis, who developed nephrotic syndrome at the same time that factor VIII inhibitor was produced. The presence of the inhibitor has not been previously described as a cause for I.C. related pathology. In view of our data, we can formulate the hypothesis that factor VIII-inhibitor I.C. caused the renal disease, at the moment when the R.E.S. was saturated by the HBsAg-antiHBs complexes. It seems advisable to carefully monitor the renal function in hemophilic patients when they develope inhibitor, in particular if they also have persistent hepatitis.