†1597 EFFECT OF PROTEIN RESTRICTION ON GROWTH IN NORMAL (N) AND CHRONIC RENAL FAILURE (CRF) RATS. Aaron L. Friedman and Rita Pityer. University of Wisconsin, Department of Pediatrics, Madison, WI.

3 week old female Sprague Dawley rats were divided into N and CRF groups. CRF was created by heminephrectomy followed in 7 days by contralateral nephrectomy. Each group was subdivided into 3 diet groups - 6, 14 and 24 percent protein. All diets were isocaloric and contained equivalent amounts of Na, K, Cl, Ca and phosphate. Wt and Ht were measured weekly for 9 wks. (12 wks. of age). *P<.05 compared to 24%.

DIET	CRF		NORMAL	
	Wt (gm)	Ht (cm)	Wt (gm)	Ht (cm)
6%	120*	17.5*	150.5*	18.7*
14%	214	21	240*	21.3
24%	180	20.5	189	20.4

Animals on 6% diet were statistically significantly smaller than 24% diet (usual protein content of rat chow) and 14% diet, both under N or CRF conditions. Under N conditions 14% animals gained more wt than 24%; under CRF the same tendency was seen. Wt and Ht were most severely effected by the combination of CRF and protein restrictions (6% diet). We conclude that:

- (1) Severe protein restriction limits growth even further in the young animal with CRF.
- (2) Mild protein restriction without calorie restriction may be beneficial for growth in CRF.
- (3) Dietary manipulation in the young animal must be different than the adult animal to test the benefit of protein restriction as treatment of CRF.

†1598 CONCAVALIN-A (Con-A) STIMULATION OF PERIPHERAL BLOOD MONONUCLEAR CELLS (PBMC) FROM MINIMAL LESION NEPHROTIC (MLN) PATIENTS IN REMISSION RESULTS IN AN INCREASED IN VITRO $^{35}\text{SO}_4$ UPTAKE IN GLOMERULAR BASEMENT MEMBRANE (GBM). Eduardo H. Garin, Department of Pediatrics, University of Florida, Gainesville, Florida.

We have previously shown a significant increase in $^{35}\text{SO}_4$ uptake in rat GBM when glomeruli were cocultured with PBMC from MLN patients in relapse, but an uptake no different than normal controls if glomeruli were incubated with PBMC of patients in remission. We have studied the $^{35}\text{SO}_4$ uptake by GBM after PBMC from 12 MLN patients in remission have been stimulated with ConA (10 $\mu\text{g}/\text{ml}$ of culture media).

There was a significant increase in $^{35}\text{SO}_4$ GBM uptake when glomeruli were cocultured with ConA stimulated MLN PBMC (geometric mean [GM], 331 cpm/mg dry glomerular weight) as compared to glomeruli cocultured with MLN PBMC (GM, 200) ($P=0.047$); glomeruli alone (GM, 162) ($P=0.008$), and glomeruli alone stimulated with ConA (GM, 146) ($P=0.003$). No significant differences were seen between the groups when glomeruli were cocultured with PBMC from 12 normal adults (GM range 81-162).

These data show that ConA stimulated PBMC from MLN patients in remission alter the sulfate metabolism of rat GBM. The stimulation of PBMC with ConA reproduces the increase in $^{35}\text{SO}_4$ uptake observed when glomeruli are cocultured with PBMC from MLN patients in relapse. Since sulfated compounds in the GBM may play a role in glomerular permeability, these findings suggest that PBMC may play a role in the pathogenesis of this disease by altering the metabolism of GBM sulfated compounds.

1599 PNEUMOCOCCAL VACCINATION IN PATIENTS WITH ACTIVE NEPHROTIC SYNDROME (NS). Eduardo H. Garin, Carlos Lee, Douglas J. Barrett. Department of Pediatrics, University of Florida, Gainesville, Florida.

Patients with NS have a decreased antibody (Ab) levels to pneumococcal vaccine when immunized during relapse. Previous studies have not differentiated between IgM and IgG responses and thus, it is not known if the decreased Ab levels are due to an inability to respond or to Ab losses in the urine. To answer these questions we have studied the IgG and IgM Ab responses to Types 3 and 19 pneumococcal polysaccharides in 11 patients (ages 2 5/12 - 21 years; median 9 years) with active NS and fifteen normal adult controls.

Both nephrotics and controls had significant increases in serum IgM Ab to Types 3 and 19 with immunization. There was no difference in post-immunization geometric mean titers of IgM Ab to either Type 3 or 19. While normals also demonstrated a significant rise in IgG Ab to both Types 3 and 19, the nephrotic patients had IgG increased only to Type 3. Further, post-immunization geometric mean titers of IgG Ab to both Types 3 and 19 were lower in nephrotic patients compared to normals ($P<0.01$). A significant correlation was found in nephrotic patients between serum albumin and Type 3 ($P<0.01$) and 19 ($P<0.05$) IgG pneumococcal Ab concentrations. "Protective" Type 3 IgG antipneumococcal Abs were found in 4/10 nephrotic and 15/15 controls.

Patients with active NS are able to mount a normal IgM immune response to pneumococcal antigens. However, since protective IgG levels are not achieved, these data cast doubt as to the efficacy of the vaccine in patients immunized during relapse.

●1600 METABOLIC CHANGES IN ENRICHED PROXIMAL TUBULES AFTER ISCHEMIC INJURY. Karen M. Gaudio, S. Gullans, G. Thulin, T. Ardito, M. Kashgarian, N.J. Siegel, Yale Univ. Sch. of Med., Dept. of Pediatrics, New Haven, CT.

The study of tubular segments, *in vitro*, allows an evaluation of cellular and metabolic changes after acute renal failure (ARF). Previous investigators have subjected proximal tubules obtained from non-injured control animals, to *in vitro* hypoxia as a model of ischemic ARF. Since *in vitro* hypoxia may not accurately simulate *in vivo* ischemia, we have developed a method to obtain viable and stable suspensions of enriched proximal tubules (EPT) from kidneys of rats after 45 min of ischemia and 15 min reflow. In EPT obtained from non-injured control kidneys: the baseline rate of respirations (RR) was $40.8 \pm 3 \text{ nM}_2/\text{mg protein/min}$; nystatin (NYS) stimulated RR by 40% to 56.2 ± 4 ($P<0.01$); ATP levels were $7.3 \pm 1 \text{ nM/mg protein}$; and there was minimal histologic evidence of cellular injury with intact mitochondria and brush borders. EPT obtained from ischemic kidneys had a significant reduction in baseline RR, 23.3 ± 2 ($P<0.01$); decreased NYS stimulated RR, 33.5 ± 2 ($P<0.01$); diminished ATP levels, 0.9 ± 0.3 ($P<0.01$) and histomorphologic changes characteristic of ischemic injury (sloughing of brush border, apical vacuoles and swollen mitochondria). EPT subjected to anoxia, *in vitro*, demonstrated condensation of mitochondria but did not develop features of ischemic injury. These data demonstrate that: 1) EPT can be obtained from kidneys after ischemic injury; 2) cellular metabolic parameters are depressed in injured EPT which develop histologic alterations of ischemia and 3) EPT subjected to *in vitro* anoxia do not have morphologic features of ischemic ARF.

●1601 THE EFFECT OF ANGIOTENSIN II (AII) ON GLOMERULAR VASCULATURE. David I. Goldsmith, Yi-Xia Lu, Andrew S. Pomrantz, and Adrian Spitzer. Albert Einstein College of Medicine, Dept. of Pediatrics, Bronx, New York.

There is controversy in the literature regarding the effect of AII on various components of the glomerular microcirculation. Experiments were performed on adult male Munich-Wistar rats paired according to weight ($n=7$ in each group). Following anesthesia, one animal in each pair was given an infusion of AII at the rate of $0.5 \mu\text{g}/\text{kg per min}$ for 15-20 min while the other animal was given an identical volume of Ringer's lactate only. At the end of the infusion, the kidneys were fixed "in situ" with glutaraldehyde and injected with a silicone rubber compound. Following histological preparation of the tissue, measurements of glomerular tuft, arteriolar, and capillary diameters were performed with a caliper on projections of transparencies. All glomeruli chosen for examination were intact; none of the vessels were sectioned. The values are expressed in μm .

	Glom. Tuft	Glom. Cap.	Affer. Art.	Effer. Art.
Control	111.2 ± 2.4	10.4 ± 0.3	16.4 ± 0.6	18.4 ± 1.6
Experim.	106.2 ± 2.8	9.8 ± 0.4	11.0 ± 2.5	12.5 ± 1.5
p	>.2	>.2	<.05	<.02

The results indicate no significant change in glomerular and capillary diameters, but a significant change (33 and 32%, respectively) in afferent and efferent arteriolar diameters. Thus, AII given in the amounts specified, constricts to a similar extent the afferent and efferent vessels and has no effect on glomerular tuft and capillary diameters.

1602 DOES ANTIBACTERIAL PROPHYLAXIS PREVENT PYELONEPHRITIS IN GIRLS WITH RECURRENT URINARY TRACT INFECTIONS?

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Bladder washout studies (BW0) were done to localize the site of infection in 74 girls with recurrent urinary tract infections (UTIs). The UTIs were lower tract in 45 and upper tract in 29. The patients were divided into those on or off antibacterial prophylaxis (Rx) depending on whether they had received continuous antibacterial medication for more than 7 days preceding the index infection. According to these data (table), if Rx had no

Site of Infection	Antibacterial Prophylaxis	
	On Rx	Off Rx
Lower	16	29
Upper	4	25

effect on the site of infection, 8 girls on Rx would be expected to have shown upper tract bacteriuria. The occurrence of renal bacteriuria in only 4 girls was probably not due to chance alone ($\chi^2 = 4.23$; $P < .05$).

Vesicoureteral reflux was identified in 17 of the 68 girls on whom voiding cystourethrograms (VCU) were obtained. Eight of 17 girls (47%) with reflux had upper tract infections while 18 of 51 (35%) with no reflux had renal bacteriuria ($\chi^2 = .75$, $P > .05$). Six of the 17 girls with reflux were on Rx. Two of these had upper tract infections while 6 of 11 not on Rx had renal bacteriuria. More data are needed before drawing a conclusion about Rx and the site of infection in girls with reflux.