1579 A SIMPLE ESTIMATE OF GLOMERULAR FILTRATION RATE (GFR) IN PREMATURE INFANTS (PT) WITH A GESTATIONAL AGE (GA) LESS THAN 35 WEEKS. Luc Brion, Alan R. Fleischman, George J. Schwartz. Albert Einstein College of Medicine, Dept. Peds., Div. Neonatol. and Nephrol., Bronx, New York.

PT are at higher risk than full term infants (FT) for renal failure. The assessment of GFR which is necessary for the administration of fluid, drugs and solutes requires accurate urine collections. A formula has been devised previously to estimate GFR (ml/min/1.73m^2) without urine collection: GFR=kL/Pcr, where L is the body length (cm), Pcr is the plasma creatinine concentration (mg/dl) and k is a constant specific for each age group. This study was designed to establish the validity of such a formula in PT < 35 wk of gestational age.

58 creatinine clearances (Ccr) were performed in 36 patients with GA 24-34 wk ($\bar{x}$ =29), birthweight 0.6-2.1 kg (1.1), postnatal age 1-21 weeks (3), postconceptional age 28-48 wk (32). The value of k was 0.32 ± 0.02 (SE), calculated from individual studies of k=GFR Pcr/L, which is lower than 0.46 ± 0.02 for FT ($p < 0.01$). The regression of GFR vs 0.3L/Pcr yielded a slope and intercept that were not statistically different from 1 and 0, respectively (r=0.80). The mean and mode of GFR-0.3L/Pcr were not statistically different from 0. Almost 2/3 of the values were between -6 and +6 ml/min/1.73m^2. There was no difference between Ccr and 3 simultaneous inulin clearances (0.4 ± 1.3 ml/min/1.73m^2, $p > 0.5$). We conclude that GFR can be reasonably estimated in PT by 0.3L/Pcr in the neonatal ICU. We have shown the value of k (PT: 0.3, FT: 0.45, child: 0.55, adolescent boys: 0.7) has a linear relationship to muscle mass and we speculate that k should increase in the first year of life in parallel with increments in the proportion of muscle mass to body weight.

1580 EFFECT OF PROTEIN INTAKE ON RENAL FUNCTION IN THE DIABETIC MOUSE. Ben H. Brouhard, Lavenia LaGrone. University of Texas Medical Branch, Department of Pediatrics, Galveston, Texas.

Increased protein intake may have a deleterious effect on progressive renal disease. To ascertain whether chronic protein-loading would adversely affect diabetic nephropathy, genetic diabetic mice (db/db) were fed normal (27%) and high(50%) protein diets. Every 2 weeks after the start of the diets urine was collected for creatinine and albumin and blood was drawn for glucose and creatinine. Creatinine clearance (C_{cr}, ml/min) and albumin excretion (U_{alb}, mg/24 hr) were calculated ($\bar{x}$±SEM):

	CONTROL(C)		DIABETIC(D)	
	27% protein diet			
	2 wks(n=10)	22 wks(n=5)	2 wks(n=10)	22wks(n=5)
C_{cr}	.22±.05	.33±.05	.74±.09[a,b]	.38±.05
U_{alb}	---	22.7±3.5	20.6±2.5[a]	40.6±5.2
	50% protein diet			
C_{cr}	.36±.05	.47±.05	.39±.05[a,b]	.18±.05
U_{alb}	12.3±3.7[a]	34.3±2.4	49.9±15.3[b]	163±68

a $p<0.05$ 2 vs 22 wks
b $p<0.05$ C vs D

The D mice fed 50% protein had significant decreases in C_{cr} at both 2 and 22 wks compared to the 27% group ($p<0.05$). Thus a high protein diet may accelerate the nephropathy in this model of diabetes.

1581 EARLY EFFECTS OF A NEPHROTOXIC CEPHALOSPORIN ON RENAL CORTICAL AND MITOCHONDRIAL CALCIUM (CA) CONTENT AND MITOCHONDRIAL FUNCTION. Marc Browning (spon. by W.B. Lorentz). Bowman Gray School of Medicine, Wake Forest University, Department of Pediatrics, Winston-Salem, N.C.

Cephaloglycin (CG) is one of several nephrotoxic drugs in the cephalosporin family of antibiotics. The mechanism of injury produced by these drugs is uncertain. Since CA has been implicated in the damage sustained by the kidney following a variety of insults, the effects of CG on CA accumulation in the cortex (CTX) and cortical mitochondria (MITO) and the relationship of mitochondrial oxygen consumption to tissue CA content were studied. The CA content of whole renal CTX and renal cortical MITO was measured two hours after a toxic dose of CG (200mg/kgIV) in control (CTRL) and experimental (EXPT) rabbits. Mitochondrial oxygen consumption supported by succinate in the presence of ADP or CA was measured from the same preparations. Data are expressed as nanomoles/mg protein and as means±SD, n=6-10, *$p<.05$ or better compared to CTRL.

	CA content		Respiratory control ratio	
	MITO	CTX	ADP	CA
CTRL	10.7±5.9	9.9±8.0	4.1±0.9	3.6±0.5
EXPT	73.8±63.3*	35.2±31.3*	2.2±1.7*	2.3±1.4*

CG leads to early accumulation of CA in the renal CTX and cortical MITO. There is a parallel decline in mitochondrial respiratory function. These results suggest that CA may play an important role in the proximal tubular necrosis and mitochondrial dysfunction caused by CG.

1582 ALUMINUM HYDROXIDE, VITAMIN D, DIHYDROTACHYSTEROL (DHT), 1,25-DIHYDROXYVITAMIN-D (1,25-D) ON ALUMINUM EXCRETION IN RATS. James C M Chan, Mary Jacob, Sue Brown, John Savory, Michael R Wills, Med Coll Virg, Richmond, VA; and Univ Virginia, Charlottesville, VA.

In order to study the effects of vitamin D on aluminum excretion when different forms of vitamin D and phosphate-binders are used simultaneously for therapeutic purposes, 30 Sprague-Dawley weanling rats, weighing 44 to 66 gm, were randomly assigned to five groups: (A) control, (B) aluminum hydroxide, (C) DHT at 16 mcg/kg/day, (D) 1,25-D at 16 ng/kg/day and (E) vitamin D2 at 2,000 IU/kg/day. Aluminum hydroxide (60 mg/kg/day) in the feed was provided to all except the control group. The vitamin D or metabolites were fed by stomach tube daily for a period of 10 days. At the end of the study, the mean serum aluminum concentration, as determined by flameless atomic absorption spectrophotometry, was 5.0 mcg/l; there was no significant differences in these results between groups. During the last three days of the study, 24-hour urine collections were made with the usual precautions against trace mineral contamination. The means (SEM) of aluminum excretion in mcg/100 gram body weight/day were:

Group	A	B	C	D	E
	(0.75(0.18)	1.99(0.85)	1.70(0.51)	1.47(0.27)	2.17(0.65)

Thus, urinary aluminum excretions doubled with treatment but were not significantly different between treated groups compared to control except between groups A and E, ($p < 0.05$). We conclude that at therapeutic doses of aluminum hydroxide and vitamin D or metabolites, hyperaluminemia was not observed, and urinary aluminum excretions were not significantly different between treated groups.

† **1583** PROXIMAL TUBULAR AMINO ACID TRANSPORT IS DEPENDENT ON THE PRESENCE OF EXTERNAL CHLORIDE. Russell W. Chesney, Naomi Gusowski and Shermine Dabbagh. University of Wisconsin, University Hospital, Department of Pediatrics, Madison, WI.

Taurine secretion by the flounder kidney is dependent on external Cl^- at the basal lateral membrane. Although taurine uptake by renal BBMV in mammalian species is Na^+-dependent (Rozen et al., Biochem J 180:245, 1979), little is known about the role of Cl^- on the accumulations of this β-amino acid. Of all anions tested; Cl, NO_3, SO_4, SCN, and tartarate, a typical overshoot was noted only in the presence of an NaCl gradient. Preincubation with choline Cl resulted in reduced uptake after imposition of an external NaCl (100mM) gradient. Since choline is poorly permeant, BBMV were preloaded with a series of Li^+ salts prior to incubation in 100 mM NaCl. LiCl preincubation reduced taurine uptake by 40-60%, $p<.001$; whereas preincubation in $LiNO_3$ or LiSCN did not inhibit. With $LiSO_4$ preincubation taurine uptake was actually stimulated at 15, 60 and 360 sec.; at equilibrium, (45 min) this Cl effect was not apparent. Nigericin in the presence of external KCl stimulated uptake by 30%, $p<.001$, but reduced uptake when KCl was inside, $p<.01$. Gramicidin, which makes membranes more permeant to Na, significantly reduced taurine uptake in the presence of NaCl, but had no effect in its absence. These findings are consistent with the hypothesis that internal Cl^- inhibits and that external Cl^- is required to fully demonstrate the Na^+-dependent uptake of the amino acid taurine and indicates that the anionic species present may govern amino acid transport by mammalian kidney.

●**1584** ALTERED DIETARY INTAKE OF SULFUR AMINO ACIDS (SAA) DOES NOT RESULT IN RENAL ADAPTATION OF CYSTEINE, METHIONINE AND SULFATE BY THE BRUSH BORDER MEMBRANE. (BBM) Russell W. Chesney, Naomi Gusowski, Marcia Padella and Shirley Lippincott. University of Wisconsin, University Hospital, Department of Pediatrics, Madison, WI.

Changes in the dietary SAA intake alters the rat renal BBM Na^+-dependent uptake of the β-amino acid, taurine. A low SAA diet enhances and a high taurine diet reduces taurine uptake (Chesney, Kidney Int 24:588, 1983). Neither the low SAA diet nor the high taurine diet alters the time course or concentration dependent accumulation by BBM of the sulfur amino acids, methionine (M) and cystine (C). The Km of Na^+-dependent M uptake is ∿ 190 μM and the Vmax is ∿ 1.5 nmoles/mg protein/15 sec on each diet. The Km of Na^+-dependent cystine accumulation is ∿ 500μM, and the Vmax is ∿ 490 p moles/mg protein/15 sec. for the low Km system and a Km of ∿5.6 mM and a Vmax of 4.8 pmoles/mg protein 15 sec for the high Km system regardless of diet. The Na^+-dependent uptake of sulfate (S) is also unchanged; the Km is ∿ 180 μM and the Vmax is 2.0 nmoles/mg protein/15 sec. By contrast the uptake of β-alanine, another β-amino acid which competes with taurine for uptake, is greater in BBM vesicles from animals on low SAA. The plasma levels of taurine are significantly changed by these dietary manipulations whereas the values for M and C remain unaltered. Conservation is expressed for β-alanine, a non-sulfur containing β-amino acid. By contrast M, C and S, which participate in a variety of synthetic and conjugative processes, are not conserved by the renal brush border surface following ingestion of altered SAA diets.