1633 RENAL HANDLING OF CALCIUM IN THE EARLY NEWBORN PERIOD. Sharon R. Siegel, UCLA Medical Center, L.A. 28 male, clinically well preterm and fullterm infants with an Apgar score >7 at 5 min. were studied. The purpose was to determine whether an impaired renal conservation of calcium(Ca) exists; the relationship of urinary Ca excretion to Ca intake and serum Ca levels; and the relationship of urinary Ca excretion to sodium(Na) intake and urinary Na excretion. Timed urine specimens were collected between 24 and 48 hrs. of age for Ca, phosphate(P), Na, creatinine(cr), and cyclic AMP (cAMP); blood was drawn at the end of the urine collection period for cr, Ca, P, and Na. Ca was measured by atomic absorption spectrophotometry, P by the method of Fiske and Subbarow, Na by flame photometry, and cAMP by radioimmunoassay. Urinary Ca excretion is positively correlated to gestational age(G.A.) ($r=0.583$, $p<.01$), serum Ca levels ($r=0.512$, $p<.01$), Ccr ($r=0.712$, $p<.001$), and urinary cAMP excretion ($r=0.717$, $p<.001$). Urinary Ca excretion is independent of Na and Ca intake, and urinary Na and P excretion. The mean %fractional Ca excretion in babies ≤32 wks. is <0.5% compared to >2.5% for Na. Serum Ca levels are positively correlated to G.A. ($r=0.803$, $p<.001$) and serum P levels ($r=0.85$, $p<.001$). In conclusion: In the well newborn infant, 1)there is no impaired conservation of Ca ≤32 wks. G.A. as for Na, 2) neither Na nor Ca intake appears to effect urinary Ca excretion, and 3) probably neither excess urinary excretion of Ca nor serum P levels should contribute to early neonatal hypocalcemia.

†1634 IN VITRO PROSTACYCLIN PRODUCTION IN THE HEMOLYTIC UREMIC SYNDROME FOLLOWING THE ADDITION OF NORMAL SERUM. Richard L. Siegler, Jean B. Smith, Mike B. Lynch, S. Fazal Mohammad. University of Utah Medical Center, Departments of Pediatrics and Pathology, Salt Lake City.

We have previously reported that the serum of many children with the Hemolytic Uremic Syndrome (HUS) is unable to stimulate cultured endothelial cells to generate normal amounts of Prostacyclin (PGI_2), a potent inhibitor of platelet aggregation and thrombus formation.

Small, uncontrolled, non-randomized case studies suggest that the intravenous infusion of normal plasma, or the use of plasma exchange, is beneficial in treating HUS by virtue of replacing a "missing" factor needed for normal PGI_2 production.

We therefore measured the ability of normal serum to enhance the ability of HUS sera to stimulate cultured endothelial cells to produce PGI_2, as assessed by the radioimmunoassay measurement of its stable metabolite, 6-keto $PGF_{1\alpha}$. The results (mean±SD) of the paired (normal sera:HUS sera) mixing experiments (n=7) are as follows:

	HUS Sera	1:3 Mixture	1:6 Mixture
6-keto $PGF_{1\alpha}$ (ng/ml)	.50±1.7	.69±.23	.69±.19

The in vitro addition of normal sera to HUS sera in a 1:3 volume ratio resulted in a significant increase ($p=0.01$) in PGI_2 production. The 1:6 mixture values did not achieve significance, however ($p>0.1$, paired t test). These mixing experiments support the "missing factor" hypothesis.

1635 IN VITRO PROSTACYCLIN PRODUCTION IN THE HEMOLYTIC UREMIC SYNDROME FOLLOWING THE ADDITION OF NORMAL SERUM. Richard L. Siegler, Jean B. Smith, Mike B. Lynch, S. Fazal Mohammad. University of Utah Medical Center, Departments of Pediatrics and Pathology, Salt Lake City.

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	HUS Sera	1:3 Mixture	1:6 Mixture
6-keto $PGF_{1\alpha}$	.50±.17	.69±.23	.69±.19

The in vitro addition of normal sera to HUS sera in a 1:3 volume ratio resulted in a significant increase ($p=0.01$) in PGI_2 production. The 1:6 mixture values did not achieve significance, however, ($p>0.1$, paired t test). These mixing experiments support the "missing" factor hypothesis.

1636 THE EFFECTS OF VITAMIN E ON THE PRODUCTION OF PROSTACYCLIN IN THE HEMOLYTIC UREMIC SYNDROME. Richard L. Siegler, Jean B. Smith, Mike B. Lynch, S. Fazal Mohammad. University of Utah School of Medicine, Depts. of Pediatrics and Pathology, Salt Lake City.

There is speculation that the antioxidant Vitamin E might be helpful in treating the Hemolytic Uremic Syndrome (HUS) by virtue of its ability to inhibit lipid peroxidation and promote PGI_2 production. Even though we have previously reported normal Vitamin E levels and normal Vitamin E/Total Lipid ratios in 15 children with HUS, we decided to see if the in vitro addition of pharmacologic amounts of Vitamin E to HUS sera would stimulate cultured endothelial cells to increase their production of PGI_2. PGI_2 production was assessed by radioimmunoassay of its stable metabolite, 6-keto $PGF_{1\alpha}$. The following results (mean±SD) were obtained before and after the addition of either 5mg/dl of Vitamin E (n=8) or the Vitamin E vehicle (alcohol) (n=7) to HUS sera:

	HUS Sera	HUS Sera plus Vitamin E	HUS Sera plus Vitamin E Vehicle
6-keto $PGF_{1\alpha}$ (ng/dl)	10.7±2.9	10.1±2.4	11.2±2.2

Increasing the concentration of Vitamin E in HUS sera to approximately six times normal had no effect on the ability of HUS sera to stimulate cultured endothelial cells to produce PGI_2.

While higher doses of Vitamin E might be effective, and while the results of this ex vivo study do not necessarily apply to in vivo situations, these results, plus our earlier finding of normal Vitamin E levels in HUS patients, fail to support a role for Vitamin E in the pathogenesis or treatment of HUS.

●1637 DEMONSTRATION AND MECHANISM OF ACTION OF IgM C3NeF IN NORMALS AND PATIENTS WITH MEMBRANO-PROLIFERATIVE GLOMERULONEPHRITIS (MPGN). Roger E. Spitzer and Ann E. Stitzel, S.U.N.Y., Upstate Medical Center, Dept. of Pediatrics, Syracuse, NY.

C3NeF has only been characterized as an IgG molecule which is present in the sera of patients with MPGN. It acts by stabilizing the alternative pathway C3/C5 convertase (C3bBb). When peripheral blood lymphocytes (PBL) from newborns, normal adults, or patients with MPGN are cultured in fetal calf serum with pokeweed mitogen, however, C3NeF is elaborated as both IgG and IgM molecules. Thus, culture supernatants (after adsorption with E, EC3b, B, P) were added to sheep erythrocytes bearing C3bBb (EC3bBb). The cells were then quantitated for bound human IgG or IgM by ELISA and decayed at 30° with measurement of residual convertase activity (RCA). Cells reacted with cultures from both normals and patients contained variable amounts of IgG (8-81 ng) and IgM (5-22 ng); decay curves showed a clear decrease in slope over control. IgM and IgG isolated from the culture supernatants by NH_4SO_4 precepitation and Protein A adsorption were both active as C3NeF. When IgM and IgG C3NeF from normal or MPGN cultures were mixed, there was a substantial decrease (40-56%) in the deposition of both molecules. RCA was unchanged with the mixture of normal Ig's. In the mixture from MPGN cultures, however, there was a marked decrease in stabilization with a shift of decay curves toward normal. These results indicate that IgM C3NeF may inhibit IgG C3NeF and, therefore, be important in the control of complement activity in MPGN.

●1638 URINARY CITRATE EXCRETION IN CHILDREN WITH HYPERCALCIURIA. F. Bruder Stapleton and Leslie A. Miller. Dept. Peds. and Clinical Research Center, University of Tennessee Center for the Health Sciences, Memphis, Tennessee.

Decreased urinary excretion of citrate, an inhibitor of urinary crystal formation is typical of adult patients (pts) with hypercalciuria (HCU). We examined urinary citrate excretion in 28 children with HCU (urine calcium >4mg/kg/d on unrestricted diet), in 5 pts with unexplained calcium stones, and in 7 normal children to determine if hypocitraturia is present early in the natural history of HCU and if citrate excretion differs in HCU pts with and without calculi. 24-hour urine citrate and calcium excretion were measured during a 300 mg calcium, 2 gm sodium diet. Renal (RHCU) or absorptive (AHCU) was determined by a calcium loading test. Data are mean ± SE.

	RHCU	AHCU	Stones	Normal
n	14	14	5	7
Ages, yrs	10.3±0.6	12.3±1.3	11.2±1.5	10.1±0.9
Urine Citrate, mg/gm creat	539±34	386±56	587±105	439±49
Urine Calcium, mg/kg/d	5.4±0.4	3.0±0.4	3.3±0.8	2.2±0.4

Urinary citrate was not statistically different from normal in RHCU, AHCU or unexplained stone pts; urine citrate in AHCU was less than RHCU, $P<0.05$ and was not statistically different in RHCU pts with or without calculi (510±45 vs 569±50 mg/gm creat) or in AHCU pts with or without calculi (348±78 vs 414±81 mg/gm creat). An inverse relationship between urine citrate and age was observed in HCU pts ($r= -0.42$, $P<0.05$) but not in normal controls ($P<0.2$). Differences in urinary citrate excretion between children and adults with HCU may explain the apparent reduced risk of calculi in children.