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Protein Intake Conditions the Diuresis Seen after Relief of Bilateral Ureteral Obstruction in the Rat (41912)

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Abstract. Natriuresis and diuresis occur in experimental animals after release of bilateral ureteral obstruction. Accumulation of urea and/or other natriuretic factors during the interval of complete obstruction may play a role in the ensuing postobstructive diuresis. The present experiments examine the potential role of dietary protein intake in conditioning the magnitude of the postobstructive diuresis after unilateral release of bilateral ureteral obstruction of 24-hr duration in the rat. Rats were fed isocaloric diets containing high (40% casein) or low (6% casein) protein for 4 weeks prior to obstruction. Rats fed a high protein diet had greater urine flows and fractional excretion of sodium and potassium after relief of obstruction than rats fed a low protein diet. Increased excretion of urea accounted for only part of the greater diuresis seen in rats fed a high protein diet. Hence, greater accumulation of other natriuretic factors during the period of obstruction in rats fed a high protein diet must play a role in the increased diuresis seen in this group of animals after release of obstruction.

In man and experimental animals, natriuresis and diuresis are known to occur after relief of bilateral ureteral obstruction (1–7). The same phenomenon does not occur after relief of unilateral ureteral obstruction (7–9). The mechanisms underlying this postobstructive diuresis remain unclear despite extensive research in this area. It has been suggested that expansion of the extracellular fluid volume during the period of bilateral ureteral obstruction may play a role in the natriuresis observed after relief of the obstruction (3). However, studies in rats have demonstrated that the increase in sodium excretion seen following release of obstruction is not merely a physiological response to volume expansion (10). Because the natriuresis and diuresis occur in the face of a marked decrease in glomerular filtration rate (GFR), they must represent a decrease in reabsorption of fluid by one, and most likely multiple anatomical segments of the renal tubule. Accumulation of urea and/or other natriuretic factors during the interval of complete ureteral obstruction may play a significant role in the ensuing postobstructive diuresis because a natriuresis and diuresis comparable to those seen after release of acute bilateral ureteral obstruction can be produced in normal rats by crosscirculation with rats with complete obstruction of a 24-hr duration (11). Further, rats subjected to unilateral ureteral obstruction in the face of reinfusion of urine from the contralateral kidney developed a marked increase in the excretion of sodium and water similar in magnitude to that observed after bilateral ureteral obstruction (11–14). The present experiments were designed to elucidate the potential role of dietary intake of protein in conditioning the magnitude of the postobstructive diuresis after unilateral release of bilateral ureteral obstruction. We reasoned that the amount of protein in the diet preceding the development of urinary tract obstruction may condition the degree of elevation of blood urea levels and perhaps affect the generation of other natriuretic factors during the interval of obstruction. The results of the present studies indicate that rats fed a high protein diet had greater urine flows and fractional excretions of sodium after unilateral relief of bilateral obstruction than rats fed a low protein diet.

Material and Methods. *Dietary regimen and induction of bilateral ureteral obstruction (BUO).* Adult Munich–Wistar or Sprague–Dawley rats were pair-fed isocaloric diets containing either low (6% casein) or high protein (40% casein) for approximately 4 weeks. The details concerning the composition of the diets and the method of feeding have been reported previously (15). After this period of dietary treatment, bilateral ureteral obstruction (BUO) was induced as previously

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described (16) and the animals were returned to their cages and not allowed water or food for the next 24 hr.

Studies in awake animals. Twenty-four hours after the onset of BUO, rats (eight pairs of Munich–Wistar rats, and six pairs of Sprague–Dawley rats fed either low or high protein-content diet) were anesthetized lightly with ether for removal of the ligature from one ureter and for the insertion of cannulae into the femoral artery, tail vein, and bladder. The animals were then placed in Plexiglass holders and a period of 1.5 to 2 hr was allowed for complete recovery from anesthesia. A priming dose of inulin (Fisher Co., St. Louis, Mo.) designed to produce plasma levels of 75 to 100 mg/dl and *para*-aminohippurate (PAH, Merck Sharp & Dohme, West Point, Pa.) calculated to produce plasma levels of 1 to 2 mg/dl were infused in 0.6 ml of saline, through the tail vein over a 3-min period, followed by a sustaining infusion delivered at 39 μl/min and containing inulin (20 μg/ml) and PAH (1 μg/ml) so as to maintain plasma levels of these compounds constant. Following an equilibration period of at least 60 min, and approximately 4.5 hr after the onset of release of ureteral obstruction, three consecutive collections of urine and blood specimens were made for the determination of whole kidney inulin (C_{In}), PAH clearances (C_{PAH}) (17) and urinary excretion of sodium, potassium, and urea.

Effects of inhibition of angiotensin II formation. To determine whether or not differences in angiotensin II production on the two different diets may influence the postobstructive diuresis, we examined the effects of administration of captopril, an inhibitor of angiotensin I converting enzyme, in six Munich–Wistar rats fed a low protein diet and six Munich–Wistar rats fed a high protein diet. In these experiments, after unilateral release of BUO, three control clearance periods were obtained under awake conditions, followed by the administration of captopril [SQ14225 (batch 230003), kindly supplied by the Squibb Institute of Medical Research], 10 mg/kg body wt, iv, over a 5-min period. Subsequently, three additional clearance periods were obtained. Urine samples from all clearance periods were collected in chilled, weighed tubes for the determination of urine flow rate and concentration of inulin, PAH, sodium, potassium, and urea.

Analytic determinations. Inulin concentrations in plasma and urine were determined by the method of Fuhr *et al.* (18). PAH content of blood and urine was quantitated by a modification of the method of Smith (19). Urea in blood and urine was measured by the method of Marsh *et al.* (20) as adapted for the Technicon Autoanalyzer. Sodium and potassium in serum and urine were determined by flame photometry using an Instrumentation Laboratories Model 143.

Calculations and statistics. Clearances of inulin and PAH, filtered loads, and fractional excretions of Na and K were calculated using standard formulae. An unpaired Student *t* test was used when comparing data of rats fed a low protein with those fed a high protein diet. A paired Student *t* test was used when comparing data obtained in the same rats before and after captopril administration.

Results. *Studies in awake animals.* Table I summarizes mean values for blood urea nitrogen and whole kidney function measured in 14 pairs of rats fed a low or a high protein diet. At the time of study, body weights averaged 276 ± 25 g in rats fed a low protein diet vs 285 ± 22 g in those fed a high protein

TABLE I. EFFECTS OF UNILATERAL RELEASE OF BILATERAL URETERAL OBSTRUCTION ON RENAL FUNCTION IN RATS FED A LOW OR A HIGH PROTEIN DIET

	BUN (mg/dl)	C_{In} (ml/min)	C_{PAH} (ml/min)	FF (%)
Low protein (n = 14)	57.6 ± 3.9	2.87 ± 0.38	11.7 ± 1.34	25.6 ± 2.4
High protein (n = 14)	117.3 ± 5.1	1.53 ± 0.19	6.37 ± 0.70	25.9 ± 2.3
P	<0.001	<0.005	<0.01	NS

Note. C_{In} = inulin clearance; C_{PAH} = clearance of *para*-aminohippurate; FF = filtration fraction [(C_{In}/C_{PAH}) × 100]. Data reported for C_{In} and C_{PAH} are per kg body wt.

diet. These values are not significantly different from each other. Arterial blood hematocrits were not statistically different between the two groups (42.7 ± 1.2% for low-protein-fed rats and 45.6 ± 1.0% for high-protein-fed rats). By contrast, blood urea nitrogen values were, on average, twofold greater in high-protein-fed rats than in low-protein-fed rats. Whole kidney clearances of both inulin and PAH were significantly greater in rats fed a low protein diet than in animals fed a high protein diet. Calculated values for filtration fraction (FF) were essentially identical in both groups of rats.

Table II presents the mean values for serum concentrations of sodium and potassium, urine flow rates, and excretion of sodium, potassium, and water in the urine after unilateral release of BUO in rats fed a low or a high protein diet for 4 weeks prior to BUO. Serum concentrations of Na and K were not significantly different between the two groups of rats. Urine flow and fractional excretion of water were significantly greater in the rats fed a high protein diet. Although the absolute excretions of sodium and potassium were not significantly different between the two groups of rats, the fractional excretions of both sodium and potassium were significantly greater in the rats fed a high protein diet. As would be expected, the filtered loads of both sodium and potassium were significantly lower in the rats fed a high protein diet due to the marked decrease in GFR in this group of animals when compared to rats fed a low protein diet.

In a group of six low-protein-fed and six high-protein-fed rats, the effects of captopril infusion on inulin and PAH clearances were examined after obtaining initial control clearance periods. The results are given in Table III. As can be seen, captopril administration resulted in significant and comparable increases in C_{In} and C_{PAH} in both low- and high-protein-fed animals so that whole kidney filtration fraction, FF, remained essentially constant in both groups. Captopril administration produced similar increments (18 μl/min) in urine flow in rats fed a low or a high protein diet. However, the increment in urine flow rate from baseline was approximately 67% in low-protein-fed rats and only 30% in rats fed a high protein diet.

TABLE II. SERUM CONCENTRATIONS OF SODIUM AND POTASSIUM, UREA NITROGEN, AND EXCRETION OF ELECTROLYTES, WATER, AND UREA AFTER UNILATERAL RELEASE OF BILATERAL URETERAL OBSTRUCTION IN RATS FED A LOW OR A HIGH PROTEIN DIET

	Serum (meq/liter)		Urine flow (μl/min)	FL_{Na} (μeq/min)	$U_{Na}V$ (μeq/min)	Fe_{Na} (%)	FL_K (μeq/min)	U_KV (μeq/min)	FE_K (%)	FE_{H_2O} (%)
	Na	K								
Low protein (n = 14)	143 ± 0.8	4.3 ± 0.13	42 ± 5	104.8 ± 11.7	4.23 ± 0.60	4.84 ± 1.05	3.18 ± 0.39	1.50 ± 0.13	53.3 ± 6.0	6.63 ± 4.69
High protein (n = 14)	142.9 ± 1.0	4.3 ± 0.14	64 ± 7	60.8 ± 9.2	5.58 ± 0.56	11.4 ± 1.63	1.80 ± 0.26	1.44 ± 0.17	88.6 ± 7.5	18.10 ± 2.22
P	NS	NS	<0.025	<0.01	NS	<0.01	<0.01	NS	<0.01	<0.01

Because there was an increase in GFR in both groups of rats, the greater urine flow after captopril did not result in a significant increase in fractional water excretion. Captopril administration increased the absolute excretion of sodium ($U_{Na}V$) in both groups of rats. However, only the rats fed a low protein diet demonstrated a significant increase in fractional excretion of sodium (FE_{Na}) after administration of captopril.

Figure 1 compares the fractional excretion of sodium and water and the absolute excretion or urea after unilateral release of BUO in rats fed a low or a high protein diet for 4 weeks preceding the onset of obstruction. Fractional excretion of sodium and water was approximately two- and threefold greater in rats fed a high protein diet as compared to those fed a low protein diet. Absolute urea excretion was approximately twofold greater in rats fed a high protein than in those fed a low protein diet. This increase in the absolute excretion of urea in rats fed a high protein diet occurred despite a 50% reduction in GFR in these rats as compared to those fed a low protein diet.

Discussion. In experimental animals (7, 10, 12, 16, 21–23) the release of BUO results in a dramatic increase in sodium and water excretion in the urine. In the present studies the same phenomenon was observed. Since the animals in the present study were fasted in the interval between obstruction and its release, it is clear that the changes in sodium and water excretion are not due to extracellular fluid volume expansion. Despite a severe reduction in GFR, and hence in the filtered load of sodium, following release of bilateral ureteral obstruction, absolute excretion of sodium was greater than normal (7, 8, 10, 12, 16, 21–24), and fractional sodium excretion rose from a level of less than 1% found in normal rats to almost 5% in the rats fed a low protein diet and to over 11% in those fed a high protein diet. The amount of water excreted also increased. Thus, this change must represent a decrease in the reabsorption of sodium and water by one and most likely multiple anatomical segments of the renal tubule.

The mechanism(s) responsible for this decrease in sodium and water reabsorption is not entirely clear although the role of several

TABLE III. EFFECTS OF CAPTOPRIL ON RENAL FUNCTION AFTER UNILATERAL RELEASE OF BILATERAL URETERAL OBSTRUCTION IN RATS FED A LOW OR A HIGH PROTEIN DIET

	n	Body wt (g)	BUN (mg/dl)	C_{In} (ml/min)	C_{PAH} (ml/min)	FF	FE_{Na} (%)	Fe_K (%)	Urine flow (μl/min)	FE_{H_2O} (%)	$U_{Na}V$ (μeq/min)
Low protein	6										
Baseline		336 ± 21.2	53.7 ± 4.7	1.87 ± 0.19	9.62 ± 1.46	.22 ± 0.04	3.30 ± 0.81	61.63 ± 10.43	27.0 ± 3	4.53 ± 0.86	2.74 ± 0.71
Captopril				2.65 ± 0.22	13.34 ± 1.74	.24 ± 0.04	5.20 ± 1.03	41.32 ± 5.64	45.0 ± 5	5.32 ± 0.87	6.20 ± 0.95
P^a				<0.001	<0.005	NS	<0.005	<0.05	<0.001	NS	<.01
High protein	6										
Baseline		338 ± 16.2	109.8 ± 8.1	1.13 ± 0.16	5.73 ± 1.20	.23 ± 0.04	14.00 ± 2.98	88.36 ± 7.84	60.0 ± 5	18.57 ± 3.81	6.47 ± 0.73
Captopril				1.87 ± 0.34	7.94 ± 1.63	0.24 ± 0.02	12.80 ± 3.42	59.60 ± 9.62	78.0 ± 5	16.42 ± 4.54	8.74 ± 0.44
P^a				<0.02	<0.05	NS	NS	<0.025	<0.025	NS	<.02
^{b}		NS	<0.001	NS	NS	NS	<0.01	NS	<.001	<.01	<.01
^{c}				NS	NS	NS	NS	NS	<.001	<.05	<.05

Note. Values are expressed as means ± SEM, *n* = number of rats; NS = no significant difference ($2p > 0.1$). Data for C_{In} and C_{PAH} are per kg body wt.

[a] Calculated from paired data for the values of baseline vs captopril.

[b] Calculated from unpaired data for the values of low protein vs high protein baseline.

[c] Calculated from unpaired data for the values of low protein vs high protein captopril.

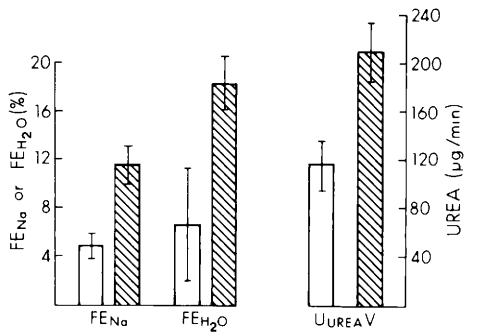

FIG. 1. Fractional excretion of sodium (FE_{Na}) or water (Fe_{H_2O}) and absolute excretion of urea ($U_{urea}V$) after unilateral release of bilateral ureteral obstruction of 24-hr duration in rats fed a low protein (□) or a high protein diet (▨). Values are the means ± SEM of results obtained in 14 pairs of rats fed a low or a high protein diet. Values for each animal represent the mean of three clearance periods obtained between 2 and 4.5 hr following relief of obstruction. Fractional excretion of sodium and water and absolute excretion of urea were significantly greater ($P < 0.01$) in rats fed a high protein diet when compared to those fed a low protein diet.

factors has been clarified. It is unlikely that this decreased reabsorption is due to elevated ureteral pressure since an acute increase in ureteral and hence intratubular pressure has been shown to increase the reabsorption of sodium and water (13, 25–29). Further, postobstructive diuresis follows the release of bilateral but not unilateral ureteral obstruction (7–9). The number of kidneys obstructed does not condition this alteration in sodium and water handling. A postobstructive diuresis occurs after release of ureteral obstruction when the opposite kidney has been severely damaged or surgically removed (13, 23). Thus, it is the period of anuria which precedes the release of ureteral obstruction that determines whether or not a postobstructive diuresis will occur. How anuria initiates this phenomenon is only partially understood, but the implication is that a natriuretic factor(s) must accumulate during this interval.

The circulating factors responsible for decreased reabsorption of sodium and water during postobstructive diuresis are not clearly defined. Urea, acting as an osmotic diuretic, has been implicated (1). Early attempts to establish such a role for urea were unsuccessful (7, 22). These early attempts failed for at least two reasons: (1) urea infusion induced an osmotic diuresis in the contralateral kidney which contracted the extracellular fluid volume and acted to increase salt and water reabsorption; (2) the studies were done in anesthetized rats, and renal blood flow of the kidney subjected to unilateral obstruction is particularly sensitive to the effects of anesthesia. When urea is infused into awake rats following the release of unilateral ureteral obstruction and particular care is taken to replace all water and solute lost from the contralateral kidney, a postobstructive diuresis does occur (13). In this setting approximately 9% of the filtered sodium is excreted in the urine.

In the present study the greater fractional excretion of both sodium and water in the rats fed a high protein diet was accompanied by an absolute urinary excretion of urea which was approximately twofold greater in these rats than in those fed a low protein diet. It is possible, therefore, to use these values and those for urine flow rate to make some qualitative predictions regarding the role of urea in the postobstructive diuresis observed in these rats after unilateral release of BUO. Urine flow was 64 μl/min after release of obstruction in rats fed a high protein diet as compared to 42 μl/min in those fed a low protein diet. At the same time the excretion of urea was approximately 90 μg/min greater in rats fed a high protein diet than in those fed a low protein diet. If one assumes isoosmotic excretion of urea after relief of obstruction, the difference in urea excretion between low- and high-protein-fed animals would account for approximately 5 μl/min or roughly 25% of the increment in urine flow seen (22 μl/min). Thus, urea plays a role in the postobstructive diuresis and natriuresis observed after unilateral release of bilateral ureteral obstruction. However, the greater depression in GFR and/or the level of protein intake may have conditioned the greater retention of other natriuretic substances besides urea in rats fed a high protein diet. Although increased urea excretion appears to play a role by acting as an osmotic agent, the contribution of other factors to the natriuresis and diuresis observed in rats fed a high protein diet has to be considered.

Of interest is the finding of significantly lower clearances for inulin and PAH after unilateral release of BUO in rats fed a high protein diet than in those fed a low protein diet. We have reported preliminary evidence (30) to indicate that such changes are due to greater vasoconstriction of the preglomerular circulation in rats fed a high protein diet. The different degree of vasoconstriction in the two groups of rats after BUO is mediated by thromboxane A_2, whose renal production seems to be influenced by the amount of dietary protein. The vasoconstriction observed after BUO is also mediated by angiotensin II (31). Inhibition of angiotensin II formation by captopril resulted in significant increases in the clearances of inulin and PAH in both groups of rats (see Table III). The increments in C_{In} and C_{PAH} were not significantly different between the two groups of rats. Thus, differences in angiotensin II "production" or "activity" do not seem to occur as a consequence of changes of dietary protein in the two groups of rats.

The results of the present studies indicate that rats fed a high protein diet had greater urine flows and fractional excretion of sodium and potassium after unilateral release of bilateral ureteral obstruction than rats fed a low protein diet. Increased excretion of urea accounted for only part of the greater diuresis seen in rats fed high protein diets. Hence, greater accumulation of other natriuretic factors during the period of obstruction, in rats fed high protein diets, must play a role in the increased diuresis seen in this group of animals after release of obstruction.

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