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Role of Volume and Prostaglandin Synthesis in the Suppression of Renin by Saline in the Rat (42386)

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Abstract. To evaluate the contribution of plasma volume expansion per se on acute inhibition of renin release by sodium chloride infusion, renin responses to comparable plasma volume expansion with intravenous infusions of sodium chloride, sodium bicarbonate, or albumin were studied in separate groups of sodium chloride-depleted rats. In addition, urinary prostaglandin E_2 (PGE_2) excretion rate was compared in the saline- and sodium bicarbonate-infused animals to evaluate the relationship between acute changes in renin release and intrarenal PGE_2 synthesis. All three groups were plasma volume-expanded by approximately 55%. Plasma renin activity (PRA) decreased in response to saline (12.3 ± 1.0 to 6.7 ± 0.7 ng AI/ml/hr; $P < 0.01$) whereas PRA did not change with sodium bicarbonate (11.3 ± 1.4 to 10.2 ± 1.5) or albumin (9.9 ± 0.7 to 8.2 ± 1.0). The rate of PGE_2 excretion was not changed by either saline (72.2 ± 13.1 to 72.3 ± 18.7 pg/min) or sodium bicarbonate infusion (70.7 ± 8.8 to 64.9 ± 7.0). These results support the hypothesis that acute suppression of PRA by infusion of saline is not dependent upon volume expansion per se. In confirmation of earlier observations, inhibition of renin release by sodium chloride was related to chloride. Finally, the results suggest that the renal tubular mechanism for inhibition of renin release by sodium chloride is not related to overall changes in renal PGE_2 synthesis in the rat. © 1986 Society for Experimental Biology and Medicine.

Renin release is inhibited by both acute sodium chloride infusion and by plasma volume expansion with albumin. The effect of sodium chloride on renin has been attributed to a renal tubular mechanism, whereas inhibition of renin release by acute plasma volume expansion with albumin is presumably mediated by a neurally controlled volume-sensing mechanism (1, 2). Since intravenous infusion of sodium chloride also results in plasma volume expansion, it is not clear whether the inhibition of renin release that occurs with infusion of saline is partly dependent upon a volume as well as a tubular mechanism. The principal objective of the present study was to evaluate the contribution of plasma volume expansion per se on inhibition of renin release in response to an acute standardized intravenous infusion of sodium chloride. In separate groups of animals, we studied renin responses to comparable plasma volume expansion with intravenous infusions of sodium chloride, sodium bicarbonate, or albumin.

Renal prostaglandins modulate renin secretion in response to a variety of clinical and experimental conditions (3, 4). Prostaglandins have been reported to stimulate renin release both by a direct effect on the juxtaglomerular cells and indirectly via a renal tubular mechanism (5, 6). Prostaglandin synthesis inhibitors have been reported to have no effect or to inhibit the rise in plasma renin activity in response to dietary sodium chloride deprivation (7, 8). A second objective of the present study was to evaluate the relationship between acute changes in renin release and urinary prostaglandin E_2 excretion in response to acute plasma volume expansion with sodium chloride and sodium bicarbonate.

Methods. Plasma renin activity (PRA) was measured before and after acute plasma volume expansion with isotonic sodium chloride ($n = 6$), isotonic sodium bicarbonate ($n = 7$), or hyperoncotic albumin ($n = 7$) infusion in 250- to 300-g male Sprague–Dawley rats. Urinary prostaglandin E_2 (PGE_2) concentration was measured before and after infusion of sodium chloride and sodium bicarbonate. To permit measurement of renin suppression from a stimulated state, all animals were maintained on a low NaCl (<0.005 mEq/g) diet (ICN/Nutritional Biochemicals, Cleveland, Ohio) and distilled water *ad libitum* for 3–5 days prior to the acute experiment.

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On the experimental day, the animals were anesthetized with Inactin (100 mg/kg body wt (BW), ip). After tracheostomy, two jugular veins were cannulated with PE-50 tubing for the infusion of inulin and experimental solution. The right femoral artery was cannulated with PE-50 tubing to monitor direct intraarterial blood pressure and to collect blood samples. A bladder catheter (PE-50) was inserted for the collection of urine. Following insertion of the jugular catheters, all animals received a bolus of inulin (0.2 ml/100 g body wt) and then a maintenance infusion of 2% inulin in 0.15 *M* sodium bicarbonate at a rate of 1 ml/100 g body weight per hour for the duration of the experiment.

After completion of all surgical procedures, the animals were allowed to stabilize for 30 min. Urine was then collected during a subsequent 30-min control clearance period, at the end of which a minimal amount of blood (600 μl) was obtained for the measurements of baseline PRA, inulin concentration, and hematocrit. Following this control period, each animal was infused over the next 60 min with either isotonic saline at a rate of 5% body weight per hour, isotonic sodium bicarbonate at a rate of 7.5% body weight per hour, or 25% human serum albumin at a rate of 0.2% body weight per hour. Preliminary experiments had demonstrated that the infusion rates for these experimental solutions result in equal expansions of plasma volume in each group (approximately 50%). Urine was collected during the latter 30 min of the 60-min infusion period for clearance measurements. At the end of the infusion period 1.0 ml of blood was rapidly drawn for the measurement of PRA, and an additional 5.0 ml of blood was then obtained for measurements of inulin concentration, hematocrit, and plasma electrolyte concentrations. Mean arterial blood pressure was monitored throughout the experiment.

Further, to compare the rate and extent of plasma volume expansion, saline and albumin infusions as described above were carried out in separate groups of rats ($n = 6$/group). Serial blood samples (150 μl) were obtained for the measurement of hematocrit at the beginning of the infusion and at 15-min intervals during the 60 min of infusion and for the 60 min following infusion.

Urinary and plasma sodium and potassium concentrations were measured with a flame photometer (Instrumentation Laboratory, Morris Plains, N.J.); chloride concentrations were measured with a Buchler chloridometer (Buchler Instruments Division, Searle Analytic, Fort Lee, N.J.). Inulin concentration was measured by the anthrone method (9). For measurement of PRA, blood was drawn into sodium EDTA tubes and the plasma was stored at −15°C until the PRA was measured by the method of Haber *et al.* (10). For measurement of urinary PGE_2 excretion, urine samples were collected in siliconized, calibrated test tubes and were stored in polyethylene vials at −80°C immediately after collection; all samples were assayed together after completion of the study. PGE_2 concentration was measured in unextracted urine by radioimmunoassay (New England Nuclear Corp., Boston, Mass.; sensitivity, defined as the mass corresponding to twice the standard deviation of the zero-binding, is 0.13 pg). The levels of PGE_2 in urine are sufficiently high to allow for dilution prior to assay, thereby minimizing potential nonspecific interferences. Successful direct assay of urine for PGE_2 has been reported and dilutions of up to 1:40 have yielded linear results (11). Cross-reactivity with $PGF_{2\alpha}$ and other PG metabolites has been found to be less than 1%. The percentage change in plasma volumes was calculated as

$$\frac{(\mathrm{Hct_c})}{(\mathrm{Hct_E})} - 1/(1 - \mathrm{Hct_E}),$$

where $\mathrm{Hct_c}$ is the hematocrit at the end of the control clearance period and $\mathrm{Hct_E}$ is the hematocrit at the end of the infusion period (12).

Statistical significance between groups and among groups was determined by analysis of variance using a three-by-two mixed, within-subjects factorial design; this design allows for direct comparisons between the independent groups and between the repeated measures within each group. A one-way analysis of variance was used when only postinfusion measurements were obtained. Differences between individual treatment means were analyzed using the Newman–Keuls multiple range test. Data were expressed as the means ± SEM and differences were regarded as significant at $P < 0.05$.

TABLE I. GFR, BLOOD PRESSURE, ELECTROLYTE EXCRETION, AND URINE FLOW BEFORE AND AFTER INFUSION

Group	ABP (mm Hg)		GFR (ml/min)		V (μl/min)		$U_{Na}V$ (μEq/min)		$U_{Cl}V$ (μEq/min)		U_KV (μEq/min)		U_{PGE2} (pg/μl)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
NaCl	130	123	1.13	1.74	6.0	53.9	0.71	7.48	0.26	4.83	1.55	2.57	18.10	1.55
	±4	±6	±0.18	±0.20**	±1.4	±13.8**	±0.29	±2.94**,†	±0.10	±1.83**	±0.48	±0.85	±7.4	±0.45**
$NaHCO_3$	125	119	1.11	1.81	6.8	270.9	0.65	30.81	0.33	3.01	1.31	4.85	21.23	0.23
	±8	±5	±0.17	±0.23**	±2.1	±31.0**,‡	±0.32	±2.65*,‡	±0.15	±0.61*	±0.37	±0.43**	±8.04	±0.05**
Alb	114	115	1.35	2.09	4.3	28.4	0.30	1.78	0.11	0.18	1.02	3.27		
	±3	±4	±0.21	±0.19**	±0.8	±6.5**	±0.21	±0.48*,†	±0.03	±0.06†	±0.26	±0.36**,†		

Note. Values are means ± SEM.
* $P < 0.05$ compared to preinfusion.
** $P < 0.01$ compared to preinfusion.
† $P < 0.05$ compared to other groups.
‡ $P < 0.01$ compared to other groups.

Results. Mean arterial blood pressure (ABP) did not differ among the groups before or after volume expansion and did not change within any of the groups during expansion (Table I). Glomerular filtration rate (GFR) increased significantly ($P < 0.01$) in each of the three groups and did not differ among groups before or after infusion. Urine flow rate (V) and sodium, chloride, and potassium excretion ($U_{Na}V$, $U_{Cl}V$, U_KV) were not different among groups before expansion. Urine flow and electrolyte excretion increased during volume expansion in all three groups, except that chloride excretion remained at baseline levels in the albumin-infused group, and potassium excretion remained at baseline in the saline-infused group. The greatest increases in flow and sodium excretion were observed in animals infused with sodium bicarbonate.

The degree of plasma volume expansion (%VE) was not significantly different among the three groups (Table II). In the two additional groups receiving the same saline and albumin infusions, serial blood samples at 15-min intervals showed that hematocrit fell linearly and to the same extent during infusion (Fig. 1). In the 60 min following infusion, hematocrit continued to fall in the albumin-infused group whereas hematocrit tended to increase in the saline-infused group. After volume expansion, plasma sodium and potassium concentrations (P_{Na}, P_K) did not significantly differ among groups. Plasma chloride (P_{Cl}) concentration was significantly greater ($P < 0.01$) in sodium chloride-infused animals than in the other two groups; chloride concentration was also significantly greater ($P < 0.01$) in albumin-infused animals than in the sodium bicarbonate group.

Before volume expansion, PRA did not differ among groups (Fig. 2). Volume expansion with sodium chloride resulted in significant suppression of PRA ($P < 0.01$); however, in animals infused with sodium bicarbonate or albumin, PRA was not changed by comparable volume expansion. PGE_2 excretion was not significantly different in the sodium chloride or the sodium bicarbonate groups before volume expansion. During plasma volume expansion PGE_2 excretion rate did not change significantly within either group since urine flow rate increased and urinary PGE_2 concentration decreased (Fig. 3, Table I).

TABLE II. VOLUME EXPANSION AND PLASMA ELECTROLYTES AFTER INFUSION

Group	% VE	P_{Na+} (mEq/l)	P_{K+} (mEq/liter)	P_{Cl-} (mEq/liter)
NaCl	55 ± 2	143.0 ± 1.7	3.6 ± 0.2	101.2 ± 3.5*
$NaHCO_3$	57 ± 6	142.7 ± 0.8	3.4 ± 0.2	76.4 ± 1.8*
Albumin	54 ± 3	139.6 ± 0.7	3.2 ± 0.1	88.3 ± 1.1*

Note. Values are means ± SEM.
* $P < 0.01$ compared to other groups.

Discussion. Renin release was suppressed by acute plasma volume expansion with sodium chloride, whereas comparable volume expansion with either isotonic sodium bicarbonate or hyperoncotic albumin failed to inhibit renin secretion. The present demonstration that equivalent volume expansion with either albumin or sodium bicarbonate fails to inhibit renin release substantiates the conclusion that inhibition of renin secretion by acute sodium chloride administration is not related to volume expansion per se during the first hour of infusion in the rat. During saline and albumin infusion, arterial hematocrit decreased at a comparable rate, suggesting equivalent degrees and rates of volume expansion. In the hour following infusion, hematocrit continued to fall in the animals receiving albumin whereas hematocrit tended to increase in those receiving saline. Thus, it is possible that a neurally mediated volume-dependent mechanism for renin release may be activated during a longer time frame (i.e., after 2 hr). It is unlikely that the difference in renin responses was related to differences in renal hemodynamics since mean arterial blood pressure did not change and glomerular filtration rate increased comparably in all three groups. In addition, the present results confirm our earlier observation that inhibition of renin release by sodium in the sodium chloride-deprived rat is dependent on simultaneous administration of chloride with sodium (13, 14).

Several lines of evidence indicate that sodium chloride inhibits renin release via a renal tubular mechanism. Renal arterial infusion of sodium chloride fails to inhibit renin release in a nonfiltering kidney model (15). We have previously suggested that the renal tubular signal for renin release is related to the ab-

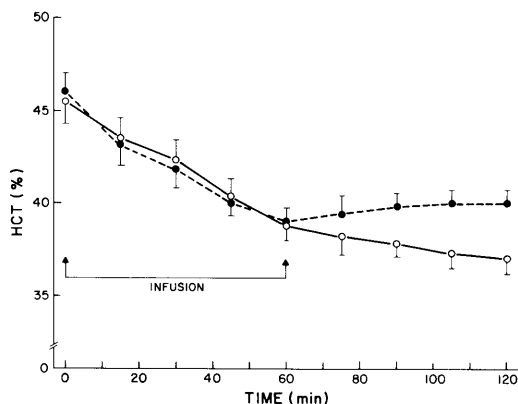

FIG. 1. Hematocrit (Hct) changes in serial blood samples taken during and after infusion of either saline (closed circles) or albumin (open circles).

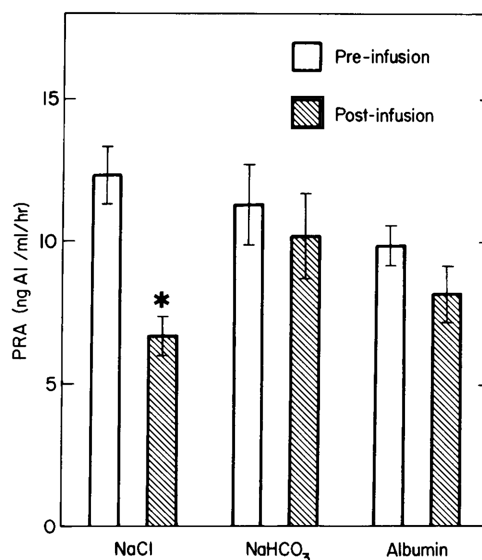

FIG. 2. Plasma renin activity (PRA) before and after plasma volume expansion with NaCl, $NaHCO_3$, and albumin. Values are the means ± SE. $P < 0.05$ compared to preinfusion.

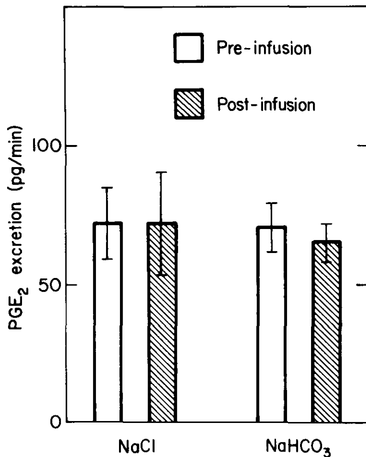

FIG. 3. Prostaglandin E_2 (PGE_2) excretion rate before and after plasma volume expansion with NaCl and $NaHCO_3$. Values are the means ± SE.

sorptive flux of Cl^- in the thick ascending limb of the loop of Henle (13, 14, 16). In contrast, inhibition of renin release by volume expansion, per se, may be related to atrial stretch, integrity of the vagi, changes in renal nerve activity (the afferent and efferent pathways of the cardiopulmonary reflex arc), or the release of atrial natriuretic peptide (17–20). Cardiac denervation also prevents inhibition of renin release by direct left atrial stretch and by intravascular volume expansion with dextran (21). Fisher and Malvin reported that despite renal denervation and vagotomy, renin release is normally suppressed by volume expansion in the dog (22). However, since their infusate was isoncotic to plasma and contained 146 mEq/l Na and 128 mEq/l Cl (5), renin suppression may have been related to a renal tubular effect of the large volume of sodium chloride given (7.5 ml/kg in 3–5 min) rather than to volume expansion per se. Our results are consistent with the observations of Tuck *et al.* (23) that in man, saline infusion decreases PRA within 10 min of the start of infusion, whereas comparable plasma volume expansion with dextran was not accompanied by a significant decrease in PRA until 240–360 min after the start of infusion. In addition, Anderson *et al.* (24) reported that a 1-hr infusion of Ringers solution depresses PRA in sodium chloride-depleted dogs whereas equivalent plasma volume expansion with dextran had no acute effect on PRA. Thus, in contrast to the renal tubular signal, the renin response to expansion of plasma volume appears to be delayed.

When PGE_2 is administered systemically, there is no resultant change in the urinary levels of this compound; thus it seems likely that PGE_2 measured in urine originates from the kidney itself (25). The levels of PGE_2 in the urine can therefore be useful as biochemical indicators of cyclooxygenase activity and intrarenal prostaglandin biosynthesis (25, 26). Several investigators have reported a direct association between changes in renin release and changes in PGE_2 excretion (3, 4, 27). In the present study, urinary PGE_2 was not affected by infusion with either sodium chloride or sodium bicarbonate. Thus, our results do not support the hypothesis that inhibition of renin release by sodium chloride is related to alterations in overall renal prostaglandin synthesis. In addition, although it has been proposed that urinary prostaglandin excretion rate may be directly related to urine flow rate (28), in the present study, marked changes in urine flow rate (ranging from 4.3 to 270.9 μl/min) did not result in change in urinary PGE_2 excretion.

While the rate of urinary PGE_2 excretion can be used as an index of intrarenal prostaglandin synthesis, the site or sites of intrarenal synthesis cannot be determined. Prostaglandin synthesis in the kidney is localized to specific portions of the nephron, and different renal cells synthesize a distinctive profile of prostaglandins in response to various stimuli (29). In the current study, although net urinary PGE_2 excretion did not differ in animals infused with sodium bicarbonate or sodium chloride, we cannot exclude the possibility that sodium chloride and sodium bicarbonate may have had different effects on PGE_2 synthesis in different segments of the kidney. For example, Chamet-Riffaud *et al.* (30) observed that salt depletion inhibits PGE_2 production in glomeruli and has no effect on prostaglandin production in papillary homogenates; salt loading, however, stimulated PGE_2 production in papillary homogenates without effecting glomerular prostaglandin production.

Similar to our observation that inhibition of renin release by sodium chloride is dependent on chloride, Linas has recently reported that renin release is greater from isolated rat

kidneys when chloride in the perfusate was partially replaced by nitrate (31). However, in apparent contrast to our results, Linas found that urinary PGE_2 excretion was less from kidneys perfused with sodium chloride than with sodium nitrate. Indomethacin inhibited both renin release and PGE_2 excretion from sodium nitrate-perfused kidneys, suggesting that renal prostaglandins mediate the renal tubular signal for renin release. There are several differences between his *in vitro* model and our *in vivo* studies, including considerably higher *in vitro* renal blood flow, GFR, and urine flow. Additionally, other investigators have reported that salt depletion stimulates renal medullary PGE_2 synthesis (32), and our animals were on a low sodium chloride diet whereas those in the study of Linas were not.

In summary, we have demonstrated that acute plasma volume expansion by 50% with saline in the rat results in significant suppression of PRA, whereas equivalent volume expansion with either albumin or sodium bicarbonate does not. Thus, inhibition of renin release by acute saline infusion is related to a specific effect of sodium chloride rather than to plasma volume expansion per se under these circumstances. Suppression of renin release by sodium chloride was not associated with changes in urinary PGE_2 excretion, suggesting that the renal tubular mechanism for inhibition of renin release is not related to overall changes in intrarenal PGE_2 synthesis.

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