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Sodium Intake of the American Male: Implications on the Etiology of Essential Hypertension

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THE AUTHOR has obtained evidence based on estimated dietary intake of sodium as well as urinary excretion of this element which indicates that the level of sodium intake is the primary etiologic factor in the development of essential hypertension in humans.[1-5] Because of this thesis concerning the cause and effect relationship between sodium ingestion and human hypertension, it has become important to establish the range of sodium eaten by a group of ambulatory, asymptomatic adult Americans as a base-line for comparison with other societies in which essential hypertension is present to a significantly greater or lesser degree than in our own. The present paper is a report on the intake of sodium, based on the urinary excretion of sodium, among 71 adult males. A summary of the data on a few of these subjects has been presented earlier in a paper dealing with the levels of sodium intake in people with and without hypertension.[4]

RATIONALE, PROCEDURE, AND METHODS

In this report, the urinary excretion of sodium has been assumed to be virtually equivalent to sodium intake. During a 10-year period of prolonged metabolic studies on ambulatory, asymptomatic individuals with good renal function, with and without hypertension, the author has found the urinary excretion of sodium to be a highly reliable index of sodium intake. This use of urinary excretion is subject to error under the following circumstances: (1) factors affecting renal function primarily or secondarily, e.g., (a) kidney disease with abnormal wasting or retention of sodium, (b) cardiac failure, cirrhosis, hypoproteinemia, (c) hormonal effects with sodium retention, e.g., premenstrual edema, steroid administration, etc., (2) extra-renal losses, in which the two commonest are through the gastrointestinal tract and the skin; obviously such conditions as (a) ptyalism, (b) gastroenterocolitis, (c) severe sweating, (d) an exuding dermatitis or a large burn with weeping, could lead to significant sodium loss. None of these was present in the subjects in the current investigation. Over a wide range of sodium intakes (from 2 to 180 meq/d) the author, as well as many other investigators (e.g.,[6,7]) has found the normal stool excretion of sodium to be about 3 meq/d so that this route of loss may be disregarded except in individuals who have been on prolonged and drastic sodium restriction. Therefore, in otherwise healthy people the chief source of unaccounted sodium loss will be through sweat. Among the 71 subjects whose urines were analyzed for sodium, 55 were in sedentary occupations (scientists, technicians, administrators) and 16 were in occupations requiring light manual work (orderlies, kitchen helpers, clerks, etc.). Studies on 68 people were done between the months of November and April during the winters of 1954–55 and 1955–56, in the course of which the weather varied from cool to cold: three collections were made in June, 1956 during a period of warm weather. It is unlikely then, that either

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TABLE I

Urine Sodium—Males Only

Case no.	Age	Weight *lb*	Work*	H.T.†	Single collection *meqNa/24 hr*	Multiple collections		
						days	*meqNa/24 hr*	*Av*
1	55	172	S	0	95	13	120, 111, 144, 164, 123, 110, 134, 122, 12, 4, 98, 114, 140, 133	126
2	43	183	LM	0	96	7	140, 143, 168, 216, 108, 107, 225	158
3	40	159	S	0	98	6	144, 178, 174, 164, 188, 192	173
4	39	159	S	0	115	7	208, 152, 183, 178, 188, 120, 71	157
5	27	144	S	0	116	7	108, 128, 215, 149, 194, 342, 161	185
6	31	178	S	0	117	—	—	—
7	54	148	S	0	117	—	—	—
8	52	155	S	+	118	—	—	—
9	33	No weight, slender	S	+	119	—	—	—
10	39	142	S	0	122	—	—	—
11	35	184	S	0	123	—	—	—
12	35	151	S	0	124	8	159, 162, 115, 111, 205, 86, 125, 111	134
13	62	136	LM	0	124	—	—	—
14	29	187	S	0	124	7	120, 104, 105, 164, 141, 179, 107	131
15	37	145	S	0	134	—	—	—
16	36	162	S	0	136	7	99, 148, 149, 134, 107, 111, 126	125
17	35	137	LM	0	137	—	—	—
18	37	142	LM	0	140	—	—	—
19	56	163	LM	+	141	7	173, 245, 157, 148, 177, 196, 159	179
20	28	119	S	0	142 242 281	7	250, 161, 153, 112, 132, 132, 154	156
21	57	157	S	0	145	—	—	—
22	61	223	S	+	145	—	—	—
23	49	152	S	0	149	7	168, 139, 165, 111, 143, 126, 125	140
24	62	154	S	+	152	—	—	—
25	31	170	S	0	154	7	147, 149, 203, 173, 298, 232, 165	195
26	40	152	S	0	159	—	—	—
27	26	167	S	0	163 275	—	—	—
28	49	153	LM	0	164	—	—	—
29	32	147	S	0	169	—	—	—
30	36	198	LM	+	171	7	224, 144, 192, 156, 238, 292, 169	202
31	26	167	S	0	172	—	—	—
32	40	172	S	0	173 147	38	134, 136, 206, 238, 208,‡ —, —, 130, —, —, —, 151, 126, 117, 130, 193, 281, 184, —, 145, 130, 197, 170, 140, 235, 157, 192, 223, 149, 192, 223, 324, 180, 201, 182, 215, 197, 219, 216, 197, 119, 102, 125, 168	180
33	45	166	S	0	178	—	—	—
34	45	179	LM	0	180	—	—	—

physical activity or ambient temperature led to significant unrecorded loss of sodium through excessive sweating in the overwhelming majority of subjects studied here.

Only males were selected for this program in view of the greater ease of collecting urine samples as well as the lack of need to allow for premenstrual sodium retention. It is possible that this elimination of females may have skewed the data slightly in favor of somewhat higher intakes of NaCl, since the author[5] as well as Ashe and Mosenthal[8] have found that among females there are more low-salt intake people while among males there are more high-salt intake individuals. Because this study was carried out as part of the author's investigations on the causal relationship between sodium ingestion and hypertension, 22 of these men had asymptomatic essential hypertension of varying levels. None of these hypertensive subjects however, had poor renal function as indicated by nocturia, fixed specific gravity or nitrogen retention, although some of them had mild albuminuria. In the first study, 70 men collected 24-hour urines (two subjects collected specimens for two 24-hour periods and another for three separate days). Subsequently, 27 of these men, as well as another man who had not participated in the first study, volunteered to collect 24-hour specimens for a week or

TABLE I—*continued*

Urine Sodium—Males Only

Case no.	Age	Weight *lb*	Work*	H.T.–	Single collection *me Na/24 hr*	Multiple collections		
						days	*me Na/24 hr*	*Av*
35	63	170	S	+	183	—	—	—
36	32	226	LM	+	183	—	—	—
37	48	226	S	+	185	9	241, 234, 177, 254, 210, 140, 179, 174, 163	197
38	41	189	S	+	187	—	—	—
39	33	159	S	+	187	—	—	—
40	33	170	LM	0	189	7	194, 206, 206, 169, 233, 240, 220	210
41	37	166	S	0	189	—	—	—
42	42	194	S	+	191	7	230, 296, 260, 201, 238, 163, 203	227
43	42	197	S	+	193	—	—	—
44	33	169	S	0	195	7	103, 172, 185, 184, 134, 223, 195	171
45	45	212	S	0	197	7	283, 213, 176, 145, 160, 148, 162	184
46	30	178	S	0	200	—	—	—
47	47	173	S	0	202	—	—	—
48	33	138	S	0	203	28	174, 168, 170, 147, 141, 136, 220, 222, 218, 155, 145, 232, 245, 219, 186, 221, 161, 176, 128, 155, 187, 174, 169, 218, 184, 157, 150, 198	181
49	43	198	S	0	206	7	63, 103, 96, 115, 117, 122, 195	116
50	30	169	S	0	208	—	—	—
51	45	170	S	0	210	—	—	—
52	40	163	LM	+	217	—	—	—
53	50	183	LM	+	217	—	—	—
54	53	189	LM	+	222	7	179, 222, 152, 154, 165, 214, 259	192
55	49	148	LM	0	225	—	—	—
					226			
56	53	200	S	0	236	—	—	—
57	27	156	S	0	238	—	—	—
58	62	178	LM	0	240	—	—	—
59	31	209	S	0	241	—	—	—
60	24	257	S	+	246	—	—	—
61	47	230	S	+	253	—	—	—
62	39	189	S	0	262	—	—	—
63	69	187	S	+	270	7	169, 173, 175, 203, 406, 182, 108	202
64	33	152	S	+	272	7	170, 195, 249, 309, 259, 213, 259	236
65	40	195	S	0	279	—	—	—
66	25	143	LM	0	289	7	225, 271, 294, 171, 266, 151, 276	236
67	37	239	S	0	296	8	194, 153, 134, 126, 162, 229, 307, 224	191
68	31	142	S	0	307	—	—	—
69	45	177	S	+	330	7	233, 285, 253, 219, 276, 274, 166	244
70	38	201	LM	0	364	—	—	—
71	45	203	S	+	No sample	6	105, 199, 144, 173, 119, 219	160

* S—Sedentary; LM—Light manual. † HT—Hypertension. ‡ Collection not obtained on this day.

longer. Therefore, in these 28 adult males, urine collections were made for from 6 to 38 days (median 7 days; average 9.1 days) and with few exceptions the collections were made on consecutive days. Each man had detailed verbal and written directions on the procedure to be followed. If a sample was lost for any reason, the subject was instructed to discard the entire collection and resume on a full 24 hour basis. Similarly, he was told not to deviate from his usual behavior and activities, and the test was to be stopped only in the event of illness. Urine was voided directly into chemically clean capped bottles specially prepared in the author's laboratory. Most of the men in this series were unaware of the problem under study but included here are the data from five colleagues in the Brookhaven Medical Department who knew of the general interest of the author: all five denied any conscious change in their use of salt or salty foods during the period of these collections. Of the 67 men classified according to salt (NaCl) intake by the author's technic[2] there were 11, 29, and 27 distributed among the low, average and high salt-intake groups, respectively. This distribution corresponds closely with that of the series of 1,124 unselected adult males from whom the 67 were derived.[5]

Sodium concentration in the urine was

measured on a Baird Model DB2 flame photometer using an internal lithium standard. From the measured 24-hour urine volume, and the concentration of sodium therein, the 24-hour urine sodium output was calculated.

RESULTS

The primary data on individual patients are shown in Table I.

In Table II the data for the single and multiple collection groups have been summarized.

TABLE II

Urinary Excretion of Sodium, meq/Day

	Mean	Median	Range
70 Subjects; single collections (74* samples)	186	183	95–364
28 subjects; multiple collections (256 samples)	178	180	63–406

24-hour excretion of sodium in urine (meq/d) in 70 ambulatory male subjects in whom single 24-urine collections were obtained (* in two subjects two 24-hour specimens, and in one subject three daily specimens were collected) compared with excretion values for 28 subjects in whom 24-hour samples were obtained for from 6 to 38 days. (See Table I for primary data.)

The range for the two groups was similar, namely from about 60 to 400 meq Na/day (approximately 4 to 24 g of NaCl). However, the average and median levels of daily urinary sodium excretion for both groups were bunched closely about 180 meq/day—equivalent to about 10 g of NaCl.

These findings suggest that, relative to urinary excretion levels, one group fairly mirrors the other. However, from Table I it is apparent that single 24-hour urine collections often could have been misleading as to the more usual level of sodium ingested, which is shown by variations from day to day of from 2 to nearly 4-fold in some subjects on whom multiple collections were made. Nonetheless in the series with 28 members, among those men with low and high average daily excretions of sodium, it was common to find fairly constant low and high daily levels, respectively. By contrast, subjects whose average excretion was about the group mean tended to vary widely from day to day from low to high levels.

DISCUSSION

Among standard texts the average intake of sodium in adults is said to be about 5–15 g of NaCl per day but primary references are notable by their absence.[9–11] Studies in which various kinds of control[12] are imposed during the collection of samples clearly are not applicable. Except for an occasional report of an individual subject who was allowed an unrestricted salt intake prior to the onset of an experiment, the only pertinent study which the author could find was that of Ashe and Mosenthal.[8] These authors measured the 24-hour urinary excretion of chloride in 1,000 ambulatory adults of New York City, who had no disease other than hypertension. Although for the most part only a single 24-hour collection was analyzed on each person, an unstated number had several such collections during the more than 15 years covered by the study. No primary data were given in the paper, but the following ranges of urinary excretion (calculated as NaCl) were found: (a) 4 g or less—50 subjects; (b) 4 to 8 g—416 subjects; (c) more than 8 g—534 subjects. In contrast to the present author,[4] they found no differences in the amount of salt eaten by the 437 individuals with, and the 563 without, hypertension. They concluded however, that among females there were more "low-salt eaters" and among males more "high-salt eaters," a conclusion with which the author is in agreement.[3]

The urinary excretion data reported here are in line with the commonly quoted estimates of daily sodium chloride intake of adult Americans as ranging from about 5 to 15 g. Although stool and skin losses were not measured in these subjects, as noted earlier it seems unlikely that under the conditions of this study the results would have been changed materially. Direct comparison between these data and those reported by Ashe and Mosenthal is made difficult by the absence of primary data in their paper, but it seems worth while to compare the two series briefly. The 24-hour urine sodium chloride excretions may be divided into three groups according to the schedule used by Ashe and Mosenthal. By such a classification it is possible to say that at one time or another in the present study, 2, 36 and 62 per cent of the

71 subjects excreted sodium in amounts which could place them in groups (a), (b), or (c), respectively. This distribution does not differ significantly ($P > 0.1$) from that of the 1,000 subjects reported by Ashe and Mosenthal. It is of interest however, that among the 330 separate 24-hour collections included in the current study, only one specimen had a level of sodium below the equivalent of 4 g NaCl/d. And, while 36 per cent of the subjects had sodium excretions at one time or another equivalent to 4–8 g NaCl/day, the 77 samples in this range comprised only 23 per cent of the total. Thus it may be unwise to assume that the two series were similar. As noted earlier, this disparity might be accounted for in these 71 subjects by the exclusion of females who have somewhat lower salt intakes than males. Another possibility deserving of consideration is that people are eating more salt (NaCl) today.

The excretion of amounts of sodium in the urine in these subjects indicates a gross positive metabolic balance for the ion, the metabolic need for which is not apparent since the daily requirements can ordinarily be satisfied by 1–2 g of NaCl,[13] or even less. It has been inferred that ingestion of salt at levels found in our society is beneficial by virtue of its effect on adrenal cortical hormone secretion, thereby providing a "stimulating" action which makes the organism more responsive to the increased demands of our complicated society.[14] On the basis of nearly 10 years' experience with drastic (2–6 meq/day) prolonged sodium restriction in the study and treatment of individuals with hypertension as well as in the study of the effects of drastic salt restriction on normotensive control subjects, the author has found no evidence to suggest the validity of such an inference.[6,15–18] The regulation of the immediate response to heightened demands upon the organism is largely controlled by the adrenal medullary hormones, epinephrine and nor-epinephrine.[19] The author has reported the results of studying the pressor response of nine subjects, eight with hypertension, to nor-epinephrine before and after sodium restriction.[20] Only one of these nine individuals, a 45-year-old female with essential hypertension, showed a clear-cut decrease in the vaso-pressor response to nor-epinephrine after salt limitation. Since the capacity for constricting blood vessels and raising the blood pressure is a very basic one for survival, the lack of change following sodium restriction in eight of the nine subjects suggests no lessened capacity for response on the part of these subjects. Furthermore, among the men studied for from 6 to 38 days in the current report there were individuals who, by dietary history and pattern of urine excretion, routinely ate only half as much salt as some of their contemporaries: there was no evidence of mental, physical, or psychologic superiority of those on the higher salt intake. The author has had under continuous metabolic observation a 69-year-old widow (#5504) who was admitted in September of 1953 to the Brookhaven Laboratory Research Hospital for study and treatment of her essential hypertension of 12 years' duration. It was established that this woman had normal renal and cardiac function, judged by the usual clinical and laboratory studies. After six weeks of control observations during which time she received 180 meq Na/day, her daily sodium intake was sharply reduced to 4 to 6 meq/d on October 26, 1953. After almost four years of such limitation, this woman remains the same active, ambulatory, intelligent (and somewhat aggressive) female that she was prior to sodium reduction, although her blood pressure has been normal for several years. Her daily activities are numerous and ordinarily include two walks, each of one to three miles in length, on the laboratory grounds. From some studies in preparation for publication, it has been found on a number of individuals that as compared with the observations during control periods when the daily sodium intake averaged 180 meq/d (10 g of NaCl), prolonged and drastic (6 meq/d) sodium restriction was unassociated with mental effects discernible by a skilled psychiatric analyst upon detailed interview. Psychologic tests performed by a trained professional psychologist showed no objective changes; electroencephalograms were unaffected by this degree of sodium limitation.

The level of sodium intake found among these Americans may be compared with that of

other societies in which hypertension is more or less frequent. As noted earlier[2] a virtual absence of hypertension has been found among different primitive ethnic groups who also differ widely in environment: e.g., the Greenland Eskimos,[21] Australian aboriginal,[22] mountainous Chinese tribes,[23] and the Cuna Indians of Panama.[24] It was of interest to find that among all of the truly primitive groups for which data were available, a common factor was a low intake of sodium, estimated from listed foods or actual analyses to contain about 1 to 2 g (2.5 to 5 g as NaCl) a day and sometimes less. By contrast, there is considerable evidence that the West Indian Negroes have a much higher incidence of hypertension than either whites or Panamanian Indians.[25,26,27] In discussing this problem in 1953 with Prof. G. P. Murdock of the Yale University Department of Anthropology, I was informed that an associate in his department had recently returned from a field trip to Jamaica, British West Indies, and had spontaneously reported that the Negroes of that area ingested large amounts of salt, chiefly through the consumption of salted pork and fish. Indeed this anthropologist reported that salt imagery played a prominent role in the songs, stories, and jokes of these people. Thus, although precise estimates of salt intake are presently lacking in these people (the author is currently organizing an expedition to determine the precise range of salt intake in these people), the evidence suggests that a high-salt intake is present, and in all probability, considering the restricted diet of these people, such an intake can be assumed to begin in early childhood at a time when, at least in animals, the organism is more sensitive to the hypertensive effects of a high sodium intake.

SUMMARY

As an index of sodium intake, complete 24-hour urine collections were made on 71 ambulatory, working, male adults for periods ranging from 1 to 38 days. The mean and median 24-hour sodium excretions were about 180 meq/day—equal to approximately 10 g of sodium chloride. The minimal and maximal daily excretions of sodium were 63 and 406 meq which are equivalent to about 4 and 24 g of sodium chloride, respectively.

Such levels of sodium excretion indicate intakes of comparable magnitude, since these healthy subjects were presumably in sodium equilibrium. There is increasing evidence that high intakes of sodium are harmful: the West Indian Negroes who probably have high intakes of sodium from early childhood develop hypertension much more frequently than do the white people. Furthermore hypertension is virtually absent among races of people known to have a low-sodium intake. All of this evidence is in agreement with the author's thesis that sodium is one major etiologic factor in the development of essential hypertension in humans.

REFERENCES

1. Dahl, L. K., and Love, R. A.: Relation of sodium chloride intake to essential hypertension in humans. *Fed. Proc.* 13: 426, 1954.
2. Dahl, L. K., and Love, R. A.: Evidence for relationship between sodium (chloride) intake and human essential hypertension. *A.M.A. Arch. Int. Med.* 94: 525, 1954.
3. Dahl, L. K., and Love, R. A.: NaCl intake as related to human hypertension. *Fed. Proc.* 15: 513, 1956.
4. Dahl, L. K.: Evidence for an increased intake of sodium in hypertension based on urinary excretion of sodium. *Proc. Soc. Exper. Biol. and Med.* 94: 23, 1957.
5. Dahl, L. K., and Love, R. A.: Etiological role of sodium chloride intake in essential hypertension in humans. *J. A. M. A.* 164: 397, 1957.
6. Dole, V. P., Dahl, L. K., Cotzias, G. C., Eder, H. A., and Krebs, M. E.: Dietary treatment of hypertension. Clinical and metabolic studies of patients on the rice-fruit diet. *J. Clin. Investigation* 29: 1189, 1950.
7. Henneman, P. H., and Dempsey, E. F.: Factors determining fecal electrolyte excretion. *J. Clin. Investigation* 35: 711, 1956.
8. Ashe, B. I., and Mosenthal, H. O.: Protein, salt, and fluid consumption of 1000 residents of New York. *J. A. M. A.* 108: 1160, 1937.
9. Peters, J. P., and Van Slyke, D. D.: *Quantitative Clinical Chemistry, Vol. I. Interpretations*, Williams & Wilkins, Baltimore, 1932, p. 766.
10. Shohl, A. T.: *Mineral Metabolism*. Reinhold, New York, 1939, p. 121.
11. Dauphinee, J. D.: in *Clinical Nutrition* (ed. by N. Joliffe, F. F. Tisdall, and P. R. Cannon), Hoeber, New York, 1950, p. 338.

12. Clark, G. W.: Studies in the mineral metabolism of adult man. *Univ. of California Pub. in Physiol.* 5: 195, 1926.
13. Falconer, M. A., and Lyall, A.: The requirements of sodium chloride. *Brit. M. J.* 2: 1116, 1937.
14. Kaunitz, H.: Causes and Consequences of salt consumption. *Nature* 178: 1141, 1956.
15. Dole, V. P., Dahl, L. K., Cotzias, G. C., Dziewiatkowski, D. D., and Harris, C.: Dietary treatment of hypertension. II. Sodium depletion as related to the therapeutic effect. *J. Clin. Investigation* 30: 584, 1951.
16. Dole, V. P., Dahl, L. K., Schwartz, I. L., Cotzias, G. C., and Harris, C.: Dietary treatment of hypertension. III. The effect of protein on appetite and weight. *J. Clin. Investigation* 32: 185, 1953.
17. Dahl, L. K., Stall, B. G., III, and Cotzias, G. C.: Metabolic effects of marked sodium restriction in hypertensive patients: Changes in total exchangeable sodium and potassium. *J. Clin. Investigation* 33: 1397, 1954.
18. Dahl, L. K., Stall, B. G., III, and Cotzias, G. C.: Metabolic effects of marked sodium restriction in hypertensive patients. Skin electrolyte losses. *J. Clin. Investigation* 34: 462, 1955.
19. Cannon, W. B.: quoted in Best, C. H., and Taylor, N. B.: *Physiological Basis of Medical Practice*, ed. 6, Williams & Wilkins, Baltimore, 1955, p. 835.
20. Dahl, L. K.: Pressor effects of norepinephrine after drastic reduction of sodium intake. *Circulation* 15: 231, 1957.
21. Thomas, W. A.: Health of a carnivorous race. *J. A. M. A.* 88: 1559, 1927.
22. Hicks, C. S., and Matters, R. F.: The standard metabolism of the Australian Aborigines. *Australian J. Exper. Biol. and M. Sc.* 11: 177, 1933.
23. Morse, W. R., and Beh, Y. T.: Blood pressure amongst aboriginal ethnic groups of Szechwan Province, West China. *Lancet* 1: 966, 1937.
24. Kean, B. H.: The blood pressure of the Cuna Indians. *Am. J. Trop. Med.* 24: 341, 1944.
25. Kean, B. H.: Blood pressure studies on West Indians and Panamanians living on the isthmus of Panama. *Arch. Int. Med.* 68: 466, 1941.
26. Saunders, G. M., and Bancroft, H.: Blood pressure of negro and white men living in the Virgin Islands of the United States. *Am. Heart J.* 23: 410, 1942.
27. Taylor, C. E.: The racial distribution of nephritis and hypertension in Panama. *Am. J. Path.* 21: 1031, 1945.