Obesity, Sodium Intake, and Blood Pressure in Adolescents

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SUMMARY It has been postulated that increased dietary sodium associated with greater food intake by obese people is a mechanism for the relationship between obesity and blood pressure (BP). We have evaluated this hypothesis by exploring the interrelationships of measures of obesity, sodium intake, and BP in 248 "normal" adolescents, 16 to 17 years of age. As an index of sodium intake, the sodium excretion in three overnight urine collections was used. As a more specific index of saltiness of diet, we used a ratio of sodium excretion to calorie intake, with calories estimated from 3-day diet records and dietary interview. Body weight and other measures of obesity showed a positive relationship with systolic blood pressure (SBP), but not with diastolic (DBP). Measures of overnight sodium excretion were positively correlated with body weight and calculated body fat percentage, suggesting that heavier people indeed ingest more sodium. This may result not from Increased intake of food per se, but from increased saltiness of diet, since calorie intake did not increase with body weight. No significant relationships were found between BP and concurrent measures of sodium excretion or diet saltiness. (Hypertension 2 (suppl 1): I-78-I-82, 1980)

KEY WORDS • obesity • sodium intake • blood pressure • hypertension • adolescents

THE existence of an association between obesity and hypertension has been known for many years.1 Even within the normotensive range of blood pressure (BP), a positive association has been demonstrated between body weight and other measures of obesity and BP, both in adults2 and children.3 As a potential mechanism for this relationship, an increased dietary sodium intake associated with greater food intake by obese individuals has been postulated.4

We have studied body weight and more direct measures of obesity, sodium excretion and BP in "normal" post-pubertal adolescents, with special reference to the potential role of sodium intake as a mechanism for the relationship between obesity and BP.

Methods

Members of the Boston cohort of the Collaborative Perinatal Study4 were invited for a heart disease risk factor evaluation at an outpatient clinic. Letters were mailed to all members of the cohort born between 1959 and 1962 with the following exceptions: letters were not sent to blacks because of their very small number in the original cohort; letters were also not sent to members of the cohort who had died, had congenital anomalies, or had not returned for a physical examination and BP measurement at 7 years of age. Of approximately 900 persons who were sent and presumably received letters (i.e., not returned by the post office), 380 responded; of these, 254 who were between 16.0 and 17.9 years of age at the time of their clinic visit make up the subjects for this study.

All subjects were asked to collect overnight urine specimens on the two mornings before and the morning of their visit "to aid in the heart evaluation," and to record the times of each urine collection period. Each collection was considered complete if the urinary creatinine excretion was at least 0.4 mg/kg/hr for males and at least 0.3 mg/kg/hr for females. The 248 subjects (130 males and 118 females) included in the present analyses had at least one complete overnight collection. Three complete collections were furnished by 207 subjects (84%), two by 30 (12%), and one complete collection by 11 (4%). When more than one complete collection was available, the average of the two or three values was used.

At the clinic, measurements were made of height, weight, triceps skinfold thickness, and BP. Height was measured with the subjects not wearing shoes; a beam balance scale was used to measure weight. Triceps skinfold thickness was measured with Holtain calipers at a point midway between the acromial process and the olecranon. Measures of obesity that were derived included the Quetelet index (wt/ht²), and an estimate of the body fat percentage based on the formula of
Rathbun and Pace, with an estimate of body density based on the formula of Seltzer and co-workers. The latter has been demonstrated to give a reasonable estimate of actual body densities obtained by underwater weighing of adolescent boys and girls.

Concentrations of electrolytes and creatinine in the urine collections were used to calculate measures of overnight sodium excretion for each individual, including the total sodium in the overnight specimen, the sodium-to-creatinine and sodium-to-potassium ratios, and, for subjects with dietary records (see below), a sodium-calorie index (the overnight sodium excretion in milliequivalents divided by the calculated calorie intake in kilocalories for the previous day multiplied by 1000).

The last 80 subjects to be studied complied with a request to keep a complete record of all food intake for the 3 days prior to the clinic visit. Subjects were asked to maintain their usual diet on these 3 days (two of which were weekdays) and to record the type and amount of all foods and beverages ingested. The records were reviewed with the subject at the time of the clinic visit to ascertain the completeness of reporting and to help validate portion size by referring to food models. The daily calorie intake was estimated from such records by utilizing the Quick Input of Food program that has been developed by the Department of Nutrition at the Harvard School of Public Health and the Department of Computer Medicine at the Beth Israel Hospital in Boston. This program has had extensive validation in both children and adults by comparisons with results from other diet records analyzed by nutritionists and by comparisons with other established computer programs for estimating calorie intake (J. Witschi, personal communication, 1979).

The BP was measured with a Narco Physiograph automatic device that records Korotkoff sounds on a strip chart. (Pressures with this device have been shown by Hohn and co-workers to closely approximate simultaneous pressures obtained with sphygmomanometer and cuff, and to adequately reflect intraarterial pressures.) The machine was calibrated before each clinic session by means of a mercury sphygmomanometer. Cuffs of appropriate widths with 30-cm-long inflation bags were used. At the beginning of BP monitoring in each subject, the amplitude of pen excursion was adjusted to reflect the onset of Korotkoff sounds (K₁) and their disappearance (K₄) as detected by simultaneous auscultation by an experienced observer. An XY digitizing table and a desk computer were utilized to measure the systolic blood pressure (SBP) and diastolic (K₂) blood pressures (DBP) from the strip chart. (Measurement of the fourth Korotkoff sound (K₄) is not possible from such tracings.) All recordings of BP were done between 8 and 9:30 a.m. with the subjects in a fasting state.

For the purpose of this publication, relationships will be reported with the resting supine SBP and DBP. They represent the average of five recordings taken at 1-minute intervals after the subject had been resting quietly alone in a room for 5 minutes.

### Results

Distributions of weight and of more direct measures of obesity, according to gender, are given in table 1. Males were not only heavier but had larger values for Quetelet index; females showed much higher values for triceps skinfold thickness and for body fat percentage.

Mean overnight sodium excretion was 43.4 mEq for males and 30.8 mEq for females, with average collection periods of 9.2 hours for both. There was large variability for both genders, with average values for individual subjects ranging from 4 to 144 mEq for males and from 6 to 207 mEq for females.

The average daily calorie intake for the 42 males with diet records ranged from 1116 to 6027 kcal, with a mean of 3001 kcal. The 38 females with diet records had an average calorie intake of 2369 kcal, with individual averages for the 3 days ranging from 950 to 3705 kcal per day.

For SBP, the mean and sd were 121.5 ± 9.0 mm Hg for males and 112.7 ± 8.7 mm Hg for females. For DBP, the corresponding values were 59.0 ± 8.3 and 58.8 ± 8.8 mm Hg. Only four males and no females had a SBP exceeding 140 mm Hg, and no subject of either gender had a DBP exceeding 80 mm Hg; thus, this is an essentially normotensive group of adolescents.

The SBP showed significant correlation with body weight and more direct measures of obesity in both males and females, as shown in table 2. For males, the correlation coefficients with SBP were slightly greater for the triceps skinfold thickness and estimated body fat percentage than for body weight, while for females, the correlation coefficient was slightly higher for body weight than for the other measures of obesity. When Quetelet index, triceps skinfold thickness, and body fat percentage were added singly to a regression model relating SBP to gender and body weight, none had a significant coefficient.

None of the measures of obesity showed significant correlation with DBP in either gender; the only one approaching significance was triceps skinfold thickness in males (r = 0.15, p = 0.08).

### Table 1. Distributions of Measures of Obesity

<table>
<thead>
<tr>
<th>Measure</th>
<th>Males (n = 130)</th>
<th>Females (n = 118)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(mean ± 1 SD)</td>
<td>(mean ± 1 SD)</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>68.8 ± 11.6</td>
<td>58.1 ± 9.8</td>
</tr>
<tr>
<td>Quetelet index (g/cm²)</td>
<td>2.35 ± 0.97</td>
<td>2.18 ± 0.35</td>
</tr>
<tr>
<td>Triceps skinfold thickness (mm)</td>
<td>10.6 ± 5.4</td>
<td>16.8 ± 5.5</td>
</tr>
<tr>
<td>Body fat percentage</td>
<td>18.8 ± 8.4</td>
<td>29.1 ± 6.3</td>
</tr>
</tbody>
</table>

*Calculated from the triceps skinfold thickness according to formulae of Rathbun and Pace and Seltzer and co-workers.
The relationships of indices of sodium excretion to body weight and body fat percentage in males are given in table 3. Total sodium in the overnight urine increased with weight, and the overall linear correlation coefficient was 0.23 (p = 0.01). A significant relation with body weight was not seen for the sodium-to-creatinine ratio, but the sodium-to-potassium ratio (which would not be affected if urine collection were incomplete) increased with body weight (r = 0.19, p = 0.04). An index of saltiness of the diet, relating total sodium in the overnight urine to calorie intake on the preceding day, also showed an increase with body weight (r = 0.46, p = 0.004). (Number of calories showed a slightly negative relationship with body fat percentage, although it was not significant statistically.)

Analogous results for females are given in table 4. Again, total sodium in the overnight urine increased with body weight and body fat percentage. The sodium-to-creatinine ratio, but not the sodium-to-potassium ratio, related significantly with body weight and body fat. While positive relationships between the sodium-calorie index and both body weight and body fat percentage were noted (r = 0.19 and 0.20 respectively), neither of these relationships was statistically significant.

None of the indices of sodium excretion/intake related significantly to SBP or DBP for either gender. Controlling for gender, we find that partial correlation coefficients with SBP were −0.05 for overnight sodium excretion, −0.06 for sodium-potassium ratio, and 0.08 for sodium-calorie index, with none of these statistically significant.

**Discussion**

To what extent increased BP is related to increased body weight per se, and to what extent to excess body fat, has not been settled. Some studies have indicated a higher correlation of the effects of high BP with skinfold thickness than with weight, but others have found BP to be more strongly related to body weight than to body fat. Our own data show that the relation of SBP to body weight is similar to the relation of BP to more direct indices of obesity (such as body fat percentage), which are measured or estimated with much less precision. This could be taken to suggest that adiposity is more important as a BP determinant. In pursuit of the mechanism of the relationship between obesity and BP, we elected to consider both body weight and body fat percentage.

The lack of correlation between measures of obesity and DBP in our data was somewhat surprising, since a positive correlation has been reported by others. As an explanation for higher BP among the obese, Dahl et al. suggested an increased sodium intake inherent in higher calorie diet. Our results suggest that heavier and more obese individuals do, indeed, ingest larger amounts of sodium, as inferred from their greater sodium excretion. However, calorie intake did not increase significantly with weight or body fat:

**Table 2. Correlation Between Measures of Obesity and Systolic Blood Pressure**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Males (n = 130)</th>
<th>Females (n = 118)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p-value</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>0.19</td>
<td>(0.03)</td>
</tr>
<tr>
<td>Quetelet index (g/cm²)</td>
<td>0.17</td>
<td>(0.05)</td>
</tr>
<tr>
<td>Triceps skinfold thickness (mm)</td>
<td>0.20</td>
<td>(0.02)</td>
</tr>
<tr>
<td>Body fat percentage*</td>
<td>0.24</td>
<td>(0.007)</td>
</tr>
</tbody>
</table>

*Calculated from the triceps skinfold thickness according to formula of Rathbun and Pace and Seltzer and co-workers.

**Table 3. Indices of Overnight Sodium Excretion and Diet Saltiness in Relation to Body Weight and Body Fat Percentage in Male Subjects**

<table>
<thead>
<tr>
<th>Index</th>
<th>Overall corr coef</th>
<th>p</th>
<th>Body weight (kg)</th>
<th>Overall corr coef</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Na (mEq)</td>
<td></td>
<td></td>
<td>&lt;60 n = 28</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>60-69 n = 46</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>70-79 n = 29</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>80+ n = 27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na/Cr</td>
<td>0.74</td>
<td>0.007</td>
<td>0.07</td>
<td>0.4</td>
<td>0.82</td>
</tr>
<tr>
<td>Na/K</td>
<td>3.1</td>
<td>0.19</td>
<td>0.04</td>
<td>0.4</td>
<td>0.90</td>
</tr>
<tr>
<td>Calories†</td>
<td>(3084)</td>
<td>0.12</td>
<td>0.4</td>
<td>(2875)</td>
<td>0.14</td>
</tr>
<tr>
<td>Na-calorie index‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>0.46</td>
<td>0.004</td>
<td>11</td>
<td>0.56</td>
</tr>
</tbody>
</table>

*Calculated from triceps skinfold thickness according to formula of Rathbun and Pace and Seltzer et al.

†Average ratio = (overnight Na excretion + daily calorie intake) X 1000.
percentage, while the ratio of sodium excretion to caloric intake generally did. The latter relationship was highly significant with body weight and body fat percentage among males, while among females it did not reach statistical significance. This difference may be explained by greater day-to-day variability in caloric intake by the females. The range of calories over the 3 days averaged 58% of the mean calories for females, 47% for males. Greater variability could lead to instability of the sodium-calorie index; for example, a large salt load on one day may be excreted over a several-day period, and if fewer calories are ingested on subsequent days, the calculated sodium-calorie index would be spuriously high and not reflect the actual diet saltiness for these days. Overall, it appears that the increased sodium excretion by our heavier and more obese individuals was not due to higher caloric consumption but possibly due to an increased saltiness of diet. More direct measures of diet saltiness were not available from this population to test this premise further.

The possibility exists, also, that under-reporting of food intake by more obese individuals explains the positive relation between the sodium-calorie index and body weight and body fat. That this was not a major problem among our subjects is supported by several factors: 1) the calorie assessment was done from dietary records kept by the subjects, rather than by recall; 2) the subjects were not being evaluated for obesity, and there was no mention of calories in the request to record food intake; 3) food items and portion sizes were recorded each day and in great detail; 4) the relationship between the saltiness index and weight (and body fat) was stronger among the males, whereas “dieting” and a propensity to under-reporting might be expected to occur more among females; 5) the pattern of no increase in caloric intake with greater body weight and adiposity is consistent with previous studies by Johnson and others, which have shown that obese young people tend to consume the same or even fewer calories than their lean peers.

The positive association between sodium excretion and body weight/fat could also arise from obese persons eliminating a greater proportion of dietary sodium intake via the kidneys, as would be the case if they engaged less in heavy physical exertion and thereby excreted less sodium through sweating. Data on exercise habits were recorded on 112 of our males and 106 females, and average monthly energy expenditure from strenuous activity was estimated by utilizing a modification of the Leisure Time Physical Activity Questionnaire of Taylor and associates. This measure of leisure-time energy expenditure was uncorrelated with both body weight (for males, \( r = 0.03 \); for females, \( r = 0 \)), and body fat percentage (for males, \( r = -0.09 \); for females, \( r = -0.07 \)). It appears that reduced leisure-time physical activity was not a major reason for the observed positive relation between urinary sodium excretion and body weight and body fat percentage in our subjects.

One other possible explanation for greater overnight sodium excretion by heavier and more obese individuals would be a longer duration of sleep on their part, and thus a longer urine collection period. However, the recorded periods of collection did not relate significantly to either body weight or body fat percentage for either gender.

It may seem to detract from the sodium-blood pressure hypothesis that there was no association between sodium excretion and SBP in our data. Other studies have also shown no relation or only a weak correlation between either overnight or 24-hour sodium excretion and concurrent BP. However, such lack of cross-sectional association may be explainable even on the premise that high sodium ingestion is conducive to BP increase. First, a small number of overnight urine collections provide only a poor index of the current sodium excretion. Expanding on previous studies, a recent study on children by Liu and coworkers indicated that six 24-hour urine collections are required to reduce to 0.01 the probability of misclassifying a child in the highest intertertile range versus the lowest one of the distribution of sodium excretion, and even more overnight specimens would be necessary to properly characterize an individual’s usual sodium excretion. Furthermore, it is possible.

| Table 4. Indices of Overnight Sodium Excretion and Diet Saltiness in Relation to Body Weight and Body Fat Percentage in Female Subjects |
|-----------------------------------------------|-------------------------------|
| Body weight (kg) | Overall | Body fat percentage* |
| Index | <50 | 50-59 | 60-69 | 70+ | Overall |
| Na (mEq) | n = 22 | n = 55 | n = 27 | n = 14 | corr | coef | p | corr | coef | p |
| Total Na | 22.6 | 27.3 | 38.2 | 37.3 | 0.27 | 0.004 | 25.3 | 28.3 | 33.6 | 32.7 | 0.20 | 0.03 |
| \( \mathrm{Na}/\mathrm{Cr} \) | 0.78 | 0.77 | 0.95 | 0.99 | 0.21 | 0.03 | 0.70 | 0.81 | 0.94 | 0.90 | 0.19 | 0.05 |
| \( \mathrm{Na}/\mathrm{K} \) | 3.4 | 3.1 | 3.7 | 3.7 | 0.14 | 0.15 | 3.1 | 3.4 | 3.5 | 3.2 | 0.06 | 0.5 |
| (Calories) | (2213) | (2450) | (2462) | (2070) | 0.02 | 0.9 | (2338) | (2612) | (2363) | (2081) | -0.09 | 0.6 |
| Na-calorie index | 0.19 | 0.3 | 13 | 9 | 19 | 13 | 0.20 | 0.3 |

*Calculated from triceps skinfold thickness according to formulae of Rathbun and Pace and Seltzer et al.
†Value based on data from 38 subjects.
‡Average ratio = (overnight Na excretion + daily calorie intake) × 1000.
that some of our adolescent subjects, upon learning of higher levels of BP in themselves or in their families, had begun to restrict sodium intake. This could lead to a less positive, or even an inverse, relationship between current sodium intake and BP.

One should also appreciate that the sodium-BP hypothesis does not imply that it is the current sodium intake that is most important as a determinant of the current BP. The previous, perhaps long-term, exposure to sodium is probably much more relevant, etiologically, than the present intake (even if the latter is accurately characterized by a large number of urine collections or other measures). Furthermore, animal experience suggests that exposure to sodium early in life may be particularly important in determining the "track" that future BP will tend to follow. Thus, associations between BP and concurrent sodium intake may occur partly because of the relation of the latter to previous sodium ingestion. Of necessity, cross-sectional studies will underestimate the relationship between sodium and BP.

In summary, three main results emerge from this study on post-pubertal adolescents. First, sodium excretion is positively correlated with body weight and measures of adiposity; this supports the hypothesis that the relationship between body fat and BP results, to some extent at least, from an association between adiposity and sodium intake. Second, calorie intake does not show correlation with body weight or adiposity, suggesting that the above relationship between body weight/fat and sodium excretion may reflect not so much calorie intake as saltiness of diet. Third, there is little or no correlation between BP and concurrent sodium excretion, as measured by three overnight urine collections; since such measurements reflect only poorly the current intake, and even less well the previous (and presumably more etiologically relevant) sodium exposure, the lack of correlation is not viewed as evidence against the sodium-blood pressure hypothesis.

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