A Perspective on Reducing Salt Intake

D.M. Hegsted

Epidemiological, clinical, and experimental evidence links excessive salt consumption to hypertension; there appears to be no evidence that it is beneficial. I conclude that it should be public policy to advise and help Americans to reduce their salt intake. Because even mild hypertension increases risk, the overall problem does not appear to be amenable to treatment, although treatment for those with clinical hypertension will always be needed. There appears to be little likelihood that identification of those “at risk” will be successful, nor does it appear that we have the capacity at this time to conduct successful preventive field trials. It is difficult for the individual to modify his diet alone. The successful strategy is to modify the food supply by changing public demand. The public responds to dietary advice if acceptable and identifiable products are available. Because most of the salt is in commercially prepared foods and because their consumption will increase in the future, the major responsibility for lowering salt consumption will fall on the food manufacturers. They are beginning to respond, and there appears to be ample opportunity for them to reduce the salt content of foods markedly. Our temporary objectives, however, should be modest, because unrealistic objectives only discourage those who attempt to follow them. (Hypertension 1991;17[suppl I]:I-201–I-204)

There is a story of two men who came to a river and saw drowning people floating by. One tried as hard as he could to drag them ashore, but there were so many he could save only a few; the second headed upstream to find out who was pushing them in.

As I understand the evidence, any increase in blood pressure, whether at the individual level as in the Framingham data, or at the population level increases risk of cardiovascular disease. Thus, a very large proportion of Americans are at risk. This is not simply an aging phenomenon because it does not occur in some populations, but it must be due to environmental causes complicated by large differences in individual susceptibility. There is no way that traditional medicine—the physician, the patient, the pill—can deal with the situation.

We often hear how difficult it is to modify dietary habits. We know that this is true, but this difficulty entails changing the eating habits when there is relatively little guidance concerning the better choices and when others among and around them are consuming another diet. If we take a broader perspective, we can note the tremendous changes that have occurred in the American diet in the last few years. Modern food technology really developed after World War II; since then thousands of new foods have been introduced and continue to be introduced.

It is not that what Americans eat cannot be changed, but to successfully effect a change, the food supply must be modified. The foods we want people to eat have to be readily available, identifiable, and acceptable to the consumer.

The current food supply, however, is little more than a happenstance: it consists of whatever the various producers in the food chain can provide and sell at a profit, and it has developed with, for all practical purposes, no nutritional guidance or control. It surprises me that anyone would conclude that this is the best food supply we can achieve or that the American diet is somehow sacrosanct and should not be changed. However fragmentary our knowledge may be, there is every reason to believe that some planning is better than none at all.

So the first point is that the American food supply and what Americans eat can be modified. The food supply obviously responds to consumer demand, and consumer demand can be changed, as for example the increasing public awareness of fat and cholesterol in the past few years. Consumption of eggs, butter, and whole milk has fallen substantially, and even beef consumption has decreased. We are eating more vegetable oils, unsaturated margarines, low fat milks, and chicken.1 We should also recognize that we are only in the preliminary stages. For many years, much of the food industry thought they could ignore dietary recommendations such as the dietary guidelines of the US Departments of Agriculture and Human Services.2 Only in the last 2 years have the beef industry, for example, and agricultural producers in

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Salt Sensitivity

Obviously, there is great interest in salt sensitivity. Identification of those who are more or less sensitive would no doubt be helpful in trying to determine how the body copes with salt. Yet I see little, so far, that appears very helpful. In short-term tests some individuals show greater and lesser responses in blood pressure to a salt load. However, I see no data to indicate that the individuals whose blood pressure rises after a salt load are, in fact, those who, over many years, will develop hypertension. Indeed, it seems possible that the opposite might be true. The response in blood pressure after a salt load is presumably an attempt of the body to cope with excess salt. Perhaps those who do show an immediate response to a salt load are those best able to deal with high salt intakes and are protected from the long-term consequences.

All of those who have attempted to evaluate the serum lipid responses to changes in dietary fat and cholesterol have observed large differences in the response of individuals to the same diet. There is substantial literature on “responders” and “nonresponders.” But in all of the 30 years or so that such studies have been done, there is, as far as I know, only one group that has attempted to evaluate the reliability of those individual responses. The data are not encouraging. Katan et al studied similar dietary changes in the same individuals about a year apart. The correlation coefficient of the individual serum cholesterol responses in the two tests was statistically significant (r=0.4), indicating some consistency in response, but this means that only about 16% of the variance could be attributed to true individual differences. The cause of these differences in response from one time to the other is not known. My guess is that the reproducibility of tests of “salt sensitivity” may not be much better. If we are going to use such tests, I suggest that we make a serious attempt to ensure that they do, in fact, differentiate between individuals and that they are really useful.

I believe that even if you can identify those who are more and those who are less susceptible, it will not be very useful from a practical point of view. As already noted, it is very difficult for any individual to modify his diet when everyone else is eating differently. Whether one looks at what we know of obesity, hypercholesterolemia, or diabetes, dietary compliance at the individual level is poor. The exceptions are when the disease is severe and clearly identified—phenylketonuria, for example. Then it is obviously worthwhile to make very serious efforts to modify the diet of the individual. The likelihood that it is possible to identify individuals who will be certain to develop hypertension some 20–40 years later is, I think, remote. And even if possible, it is most unlikely that such individuals would find that a sufficient inducement to follow a “diet” for the rest of their lives.

If there is anything we have learned in attempts to control serum cholesterol by dietary means, it is that it is a real, although moderate, increase in risk of a heart attack many years in the future is not a very strong inducement to modify one’s diet. One needs to modify the family diet or, better still, the diet of the community. Mass diseases, like hypertension, need mass approaches.

Treatment Versus Prevention

It seems rather clear to me that many believe that the usefulness of preventive efforts can be judged by the effects of salt restriction in hypertensive patients. I think it is important to note that treatment and prevention are often not the same. The best example, I suppose, would be cancer. There is abundant reason to believe that a high fat diet is involved in the etiology of many cancers, yet few believe that cancer can be effectively treated with a low fat diet. Also, we now have substantial evidence that diets high in carotene or carotenoid pigments limit cancer incidence, especially cancer of the lung. It seems unlikely, however, that dosing with carotenoids will be effective treatment.

The evidence is clear that rats made hypertensive with high salt diets develop permanent hypertension; that is, they do not respond well to low salt diets. It is
reasonably clear that hypertensive patients also fit this pattern. The point I would make is that even those who do not have hypertension may have some end-organ disease, and to a greater or lesser degree they are a changed organism. I think it is important also, to keep in mind that all public health trials will have errors that tend to invalidate the hypothesis. Such trials must be large and prolonged. There is no way that the dietary practice of individuals in such trials can be carefully monitored. If one tests the usefulness of a low salt diet, the slippage will be toward a higher salt intake than that desired and, thus, a tendency to yield a negative answer. It is not that we should not do trials, but the limitations of those kinds of trials should be recognized. I personally do not believe that we know enough about modifying foods to produce an acceptable low salt diet to warrant an effective field trial at this time.

The only long-term dietary data that we have are population data, such as that from INTERSALT. I believe that if any experimental study—be it a field trial, clinical study, or any other kind—yields results that are simply not reconcilable with the epidemiological data, we should believe the epidemiological data. We would follow the diets for the 20–50 years it takes to develop hypertension. Such a study will not be done.

In conclusion, I believe that the question “Is salt an important dietary factor in the development of hypertension?” is really not a useful one. The real questions are as follows: What is a reasonable level of salt in the American diet to shoot for? What are the best strategies for getting there? What kind of advice should we give now to people who do not yet have hypertension? I doubt that anyone really believes that a chicken breast containing nearly a gram of salt is a better choice than a similar product with one tenth that amount. Long-term changes in the diet will be produced only if we

### Table 1. Fat and Salt Contents of Commercial and Homemade Picnic Lunches

<table>
<thead>
<tr>
<th>Item</th>
<th>Commercial</th>
<th>Homemade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fat (g)</td>
<td>Salt (mg)</td>
</tr>
<tr>
<td>Fried chicken breast</td>
<td>23.7</td>
<td>787</td>
</tr>
<tr>
<td>Fried chicken leg</td>
<td>8.8</td>
<td>269</td>
</tr>
<tr>
<td>Biscuit</td>
<td>18.2</td>
<td>786</td>
</tr>
<tr>
<td>Potato salad</td>
<td>9.3</td>
<td>396</td>
</tr>
<tr>
<td>Coleslaw</td>
<td>6.9</td>
<td>261</td>
</tr>
<tr>
<td>Apple pie</td>
<td>12.0</td>
<td>412</td>
</tr>
<tr>
<td>Total</td>
<td>78.9</td>
<td>2,921</td>
</tr>
</tbody>
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can develop foods with the desired characteristics that taste good. We need a lot of effort to determine why people like what they like and how we can produce modified foods that will still be acceptable. Much of the modification of foods will have to be done by the food industry. It seems certain that they will respond if there is sufficient demand. Many have already decided that "low salt sells."

The problems involved in modifying any constituent in our diet are unique, but there are many parallels in the fat and cholesterol story with the salt story. A major difference is that the effort to lower salt intake was slow in starting. The public was well ahead of many of the experts in dealing with fat and cholesterol, and it appears to be true here also. I expect it to move in the same way.

References


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