Session VI. Factors in Reducing Salt Intake

High Salt Intake

Sensory and Behavioral Factors

Gary K. Beauchamp and Karl Engelman

Salt (NaCl) is a ubiquitous component of diets in developed countries. A major reason for this is that people judge many salted foods as more palatable than the same foods without salt. Because recent evidence indicates that an acceptable salt substitute is unlikely, an understanding of the behavioral and sensory factors involved in maintaining high salt preference is a prerequisite to successful programs aimed at reducing intake. Although little evidence exists for a genetic determination of individual differences in consumption and preferred level of salt, more research in this area is necessary. Considerable data support the view that the optimal level of salt in the diet is determined in part by the level an individual is currently consuming; increasing or decreasing customary salt intake, as long as the salt is tasted, increases or decreases the preferred level of salt in food. Although these data are consistent with a hypothesis that optimal salt preferences are learned, other data, from both animal models and human developmental studies, suggest that salt preference has an innate component. Furthermore, early experience with low or high salt diets may have a long-term impact on preferred salt levels. Liking for salt, similar to liking for sweets, has an innate basis that can be modified by individual experience. (Hypertension 1991;17[suppl I]:I-176-I-181)

Substantial evidence from epidemiological and experimental sources links dietary NaCl (salt) intake to hypertension, as has been reviewed elsewhere.1 For this reason, reduced-salt diets are frequently recommended for individuals with hypertension2 and have also been recommended for the US population as a whole.3 Here, we discuss selected sensory factors that must be considered in developing successful programs to limit or decrease intake of salt.

Because salt is required for life, it is not surprising that regulatory systems have evolved to ensure discovery, recognition, and consumption of sufficient sodium to meet that requirement. A focal point in these systems is the sense of taste. Salty taste is one of a relatively small number of primary taste qualities, others being sweet, bitter, sour, and perhaps a few others.4

Humans express preferences for salty foods over those same foods without salt. Factors responsible for the preference are discussed below and are complex. However, as a consequence of this preference, many of the foods available for purchase already contain relatively high levels of salt. Recent research indicates that perhaps less than 5–10% of the average sodium consumption comes from what is generally termed discretionary sources (salt shakers and salt added during home cooking).5 Consequently, following advice to lower sodium intake by ceasing to use discretionary salt is likely to result in a very small overall decrease in sodium intake. For there to be a substantial decline in sodium intake on a populationwide basis, a reduction in the amount of salt in prepared foods, both at the store and sold in restaurants, and a general decrease in consumption of very high salt foods appears to be necessary.

That most adults prefer salty foods rather than pure salt itself has a methodological implication for research designed to understand factors responsible for high salt intake. To evaluate the pleasantness of the taste of salt, it must be added to a familiar food. Salt taste preference studies that use saline solutions are generally very difficult to interpret.

Salt Substitutes?

A series of recent observations leads to the conclusion that it is very unlikely that a practical salt substitute can be developed in the near future. Although there are now known to be a large and growing number of sweet-tasting substances, many of them rather similar to the taste of sucrose, believed to be the purest sweet, very few compounds taste predominantly salty (Table 1). This suggests that a...
random search for non-NaCl salty compounds is unlikely to be successful.

Recent advances\textsuperscript{6-9} in the understanding of the mechanisms of sweet and salty tastes may help to explain the very small number of substances that taste salty. Sweet receptors are thought to be protein-based and likely respond to some aspects of molecular shape. As long as a molecule has the appropriate conformation, according to this view, it will taste sweet. In contrast, it is thought that at least one component of salt taste reception involves passage of the sodium ion through sodium channels at the apical end of the taste bud. The major evidence for this comes from electrophysiological\textsuperscript{6} and biophysical\textsuperscript{7} studies in experimental animals demonstrating that the diuretic amiloride reduces salt stimulation by about 50\%. Whether an equally robust effect exists in humans is unclear,\textsuperscript{8,9} and more studies are needed to clarify this issue. This finding is in basic agreement with several earlier studies.\textsuperscript{16} However, when asked to rate how much they would like to eat each of the 29 foods varying in salt content, the sodium-depleted subjects did express a greater desire for salt than when they were not treated with diuretics to further deplete them of sodium. They did not express a strong hunger for salt; this finding is in basic agreement with several earlier studies. However, when asked to rate how much they would like to eat each of the 29 foods varying in salt content, the sodium-depleted subjects did express a greater desire for salt than when they were not treated with diuretics.

### Determinants of Human Salt Preference

For heuristic purposes, the following presents a broad discussion of the reasons for high salt intake into physiological, genetic, psychological, and developmental categories. We recognize that, in reality, these categories of explanation overlap and are not mutually exclusive.

#### Physiological Factors

Most adults in the United States consume 6–12 g NaCl/day (100–200 meq), an order of magnitude or more above published requirements.\textsuperscript{10} Although it would thus appear unlikely that this level of consumption actually reflects some sort of physiological need, that has in fact been proposed,\textsuperscript{11,12} despite the fact that some people live on as little as 20 meq/day or less without apparent harm.\textsuperscript{13} At the extreme, the Yanomamo Indians are reported to consume approximately 1 meq sodium/day, as determined by urinary excretion (extra loss in sweat is possible), and other unacculturated peoples also have been reported to chronically consume very low sodium diets. Indeed, there is exceedingly little human evidence that even an extremely low sodium diet and sodium depletion are followed by a greatly increased desire for salt or what has been termed “salt appetite,” as distinguished from the “salt preference” (the choosing of salty food even when sodium is not needed) observed in animals and humans who are sodium replete.

In a recent study,\textsuperscript{15} we placed human volunteers on a very low sodium diet (5–10 meq/day) and treated them with diuretics to further deplete them of sodium. They did not express a strong hunger for salt; this finding is in basic agreement with several earlier studies. However, when asked to rate how much they would like to eat each of the 29 foods varying in salt content, the sodium-depleted subjects did express a greater desire for salty foods, and in taste tests they tended to prefer higher levels of salt in food while depleted. These data, albeit suggestive, do not conclusively demonstrate a physiologically determined salt appetite in adult humans since a psychological explanation is also possible: extremely low sodium diets are bland and unpalatable when contrasted with the previously experienced food, and it may not be the salt taste these subjects desire so much as the more flavorful food associated with saltness. However, since preference for sweet foods and sucrose in food declined during depletion, a simple desire for more orosensory stimulation cannot explain these results. This issue needs further investigation. A finding that increased salt preference among sodium-depleted subjects is due to psychological factors would further argue against the need hypothesis.

Related work\textsuperscript{17} has shown that normal subjects, when administered a hydrochlorothiazide diuretic, exhibited a compensatory increase in sodium intake.
Subjects expressed no obvious desire for increased salt, and in fact, it was not possible to determine the source of the increased intake. Interestingly, this effect was not observed with the diuretic amiloride, so it would seem that simple sodium loss could not account for the observed results.

Sodium depletion is generally seen as the adequate and necessary condition for salt appetite or enhanced salt preference. Depletion of other nutrients may also stimulate avid salt consumption. Tordoff et al. found that calcium-deficient rats increased voluntary sodium intake. It is possible that mineral deficiencies in general may stimulate salt intake because there are no specific tastes for other minerals and because, in nature, salty tasting substances are almost always associated with other minerals. Perhaps in an analogous fashion, protein deficiency could stimulate salt appetite since protein-rich animal foods contain relatively high amounts of sodium. Torii et al. and Kimura have found that very low protein diets or diets imbalanced in amino acids stimulate dramatic increases in salt consumption in rats. This is attributed to the effects of altered protein diets on fluid balance.

It is generally believed that individuals in cultures in which a primarily vegetarian diet is consumed need and crave more salt than do primarily meat-eating peoples. The traditional explanation has been that meat-eaters obtain substantial amounts of sodium in meat. Although it is certainly true that meat diets generally contain more sodium than vegetable diets, there is little evidence that the low sodium diets that might exist in vegetarian cultures would lead to an elevated desire for salt. Vegetarian diets in native populations are not only low in salt but may also be low in protein; perhaps protein content, rather than sodium content, of the diet underlies an increased desire for salt, or perhaps both protein and sodium content influence this desire.

Genetic Factors

Genetic factors have been proposed to account for differences between groups and individuals in their salt requirements, intakes, and preferences, but the evidence favoring that explanation is not yet strong. For example, it has been speculated that blacks may have a different mechanism than do whites for handling sodium. In particular, they may be less efficient in dealing with excess salt. This could contribute to the high prevalence of hypertension among blacks when salt is readily available. There is a single report that black adolescents and adults prefer higher levels of salt than do whites, although recent studies with children did not confirm this. Additionally, it is problematic whether genetic differences would necessarily underlie a race difference in preference.

Two twin studies have failed to find a heritable component to salt taste preference. These studies cannot be considered definitive, however. In one, sensory testing was brief and perhaps inappropriate because it used salt solutions rather than foods to assess preference. In our twin study, the use of very small numbers of twin pairs rendered the negative results difficult to interpret. Consequently, a genetic explanation for individual differences in salt taste preference remains a possibility, especially in view of species and strain differences that have been observed in nonhuman animals. More work is needed both in human populations and in animal models.

Psychological Explanations

Experimental studies have demonstrated that if salt intake is reduced by 30–50% over a period of time, the optimal level of salt in foods declines. The amount of salt required to optimize food flavor is decreased to about 65–75% of the prediet amount. Importantly, this change is gradual, taking probably 1–2 months to occur.

For several reasons, it is most likely that this change is due to the altered (decreased) experience of tasting salty foods rather than a physiological response to the change in the amount of sodium consumed. First, the gradual nature of the effect, as noted above, would suggest that experience plays the major role. Second, we found that if salt consumption was increased by requiring subjects to add approximately 10 g salt to their food, preferred levels of salt in food (but not in aqueous solution) increased. However, if the same amount of additional salt was consumed as tablets, and thus not tasted, there were no changes in taste preferences. Apparently, a sensory adaptation to different levels of salt accompanies a change in salt intake. To some extent, people like the level of salt they taste rather than, or in addition to, choosing the taste level of salt they like.

The third reason for believing this change in optimal salt level after dietary change to be a psychological phenomenon comes from another study, in which we reduced salt in the diet of subjects by 50% but allowed them continued ad libitum use of table salt. Under these conditions, their use of added salt increased almost fourfold but did not approach compensation for the amount removed. In fact, they only replaced 20% of the decrement; thus, there was an overall decrease of approximately 40% in sodium intake (Figure 1). In this instance, we suggest that salt was added "to taste"; this finding further implies that although the salt content of their regular food was in a range that the subjects found palatable, it was unnecessarily high, probably because it was dispersed throughout the food, rather than all being on its surface, and easily accessible to the taste receptors. This study suggests a novel technique to reduce salt intake, in that individuals were able to use as much salt as they wished while still reducing total salt intake. For this to be practical, however, it will be necessary for there to be wide availability of all categories of lowered sodium foods. In terms of control of salt preference, the importance of this research is that there were no changes in taste...
preference, even though salt intake was decreased 40%. We suggest that subjects, by adding salt to the outside of their food, obtained sufficient salty-taste experience to prevent any preference changes.

In sum (Figure 2), these studies support the conclusion that changes in salt taste preferences after halving or doubling salt intake are mediated by hedonic/cognitive expectations based on current dietary experience and not by physiological changes associated with alterations in the amount of NaCl available to the body. Although more extreme depletions may raise salt preferences as described above, the extent to which this is due to depletion rather than the blandness of the foods is unknown.

Developmental Factors

It is in infancy and childhood that the strongest claims for an experiential influence on salt taste have been made. However, there are conflicting and confusing data concerning the early development of salt preference and how experience may serve to modify or perhaps permanently establish preference.

Newborn infants either are indifferent to moderate to high concentrations of saline solution relative to water. By the time children are 2-3 or more years of age, preferences for salty foods over those same foods without salt are common. The juxtaposition of these two observations has led investigators to conclude that salt preference is learned, although that conclusion does not necessarily follow. We found that a preference for salt solutions over plain water, although not evident in infants less than 4 months of age, was evident in infants 4-23 months of age. This was observed in three separate cross-sectional studies, all using a brief presentation paradigm in which volume consumed served as the dependent variable. Harris and Booth also reported that a preference for salted cereal over plain cereal was found in infants at approximately the same age. We have hypothesized that this developmental change in response to salt represents, in part at least, postnatal maturation of the ability to taste salt, an interpretation consistent with a substantial body of evidence from animal models.

Even if there is a postnatal maturation of salt taste perception and preference, experience with salt could modulate this preference in infants just as it does with adults. Other investigators have reported a correlation between one measure of exposure to salted food and relative preference for saltiness in that food. However, these data are not conclusive as they are based on very few infants. In our recent studies, no significant correlations between a measure of salt exposure and preference have been found. Further longitudinal studies on the development and early modification of salt taste preference are needed. In particular, the issue of the relation between level of exposure to salt and salt preference should be explored at several ages in order to understand the origin of the very high salt preferences we have documented in some young children.

Sodium depletion early in life may have profound effects on later salt preference. A body of evidence, primarily derived from studies with rats, now strongly indicates that the salt taste system is plastic during its development. Salt taste perception, as measured electrophysiologically, pharmacologically, and behaviorally, matures postnatally. Early restric-

![Figure 1](http://hyper.ahajournals.org/)

**Figure 1.** Graph showing mean daily excretion of sodium in subjects when they were fed at the Clinical Research Center (dotted area) and when they were on their usual ambulatory diets (weeks 1 and 18). All points represent 11 subjects, except for week 18 (n=8). *Significant difference between comparison weeks. Reprinted from Reference 29 with permission.

![Figure 2](http://hyper.ahajournals.org/)

**Figure 2.** Bar graphs showing changes in sodium intake as determined by 24-hour urine values and changes in salt taste preference (most preferred concentration of salt in soup) in three experiments. Bars with asterisks indicate significant (p<0.05) change from baseline. Left panel: Moderate sodium reduction is followed by a decrease in optimal salt levels (Reference 27). Middle panel: More extreme sodium restriction with diuretic treatment induces a moderate increase in optimal salt levels (Reference 29). Right panel: An increase in sodium intake, but only if tasted, also induces a moderate increase in optimal salt levels (Reference 28). Not shown are the differing time courses for these taste changes. Effects seen on the left panel occur gradually over 1-3 months, whereas the changes seen on the other two panels are evident within days or a few weeks after the treatment.
tion of dietary sodium alters subsequent response to NaCl. Finally, an episode of sodium depletion induced over a period of 1 or several days by a very high dose of diuretic combined with salt-free diets produces permanent elevation in salt intake weeks or months after recovery; this effect appears especially robust if the depletion occurs during early development.

The mechanisms responsible for these effects likely have both peripheral and central components. Changes in the number or activity of amiloride-sensitive sodium channels could account for alteration in peripheral activity of salt-sensitive neurons. The persistent elevation of salt appetite after extreme sodium depletion, however, seems more likely to be of central origin, possibly a consequence of the elevation of mineralocorticoids and angiotensin that follows sodium depletion.

An analysis of available clinical studies suggests that early sodium depletion may have profound effects on subsequent salt appetite in humans as well. We searched the English language literature for evidence that sodium depletion (occurring in clinical entities such as Bartter's syndrome and Addison's disease) produces a craving for salt that might be homologous with salt appetite induced in some experimental animals after severe depletion. Surprisingly, little evidence for a human salt appetite was found, and when it was, the onset was almost invariably in childhood. The case described by Wilkins and Richter of a 3½-year-old boy with an obsessive salt appetite is the most famous, but several other similar cases of early childhood onset of salt appetite have also been reported. The absence of persuasive evidence for an adult onset of salt appetite suggests that, in humans too, early depletion may have especially potent effects on later salt taste perception and preference. This is an area ripe for further research.

In conclusion, humans prefer to consume substantially more salt than is necessary for the maintenance of normal physiology. In some instances, primarily in the salt-sensitive hypertensive individual, this may lead to illness, and in other instances, it may contribute to the worsening of an existing illness (i.e., congestive heart failure, hepatic cirrhosis with fluid retention, and increased hypertension for those on antihypertensive medication). Some sensory and behavioral factors responsible for this high consumption have been identified, but a full explanation of the mechanisms is lacking. To the extent that one understands the determinants of salt intake, one may be better able to provide guidance for successful reduction of dietary salt intake where this is medically indicated.

References
12. Ely DE, Thoren P, Wiehr J, Folkow B: Sodium appetite as well as 24-hr variations of fluid balance, mean arterial pressure and heart rate in spontaneously hypertensive (SHR) and normotensive (WKY) rats, when on various sodium diets. Acta Physiol Scand 1987;120:81–91
44. Sakai RR, Fine WB, Epstein A, Frankmann SP: Salt appetite is enhanced by one prior episode of sodium depletion in the rat. *Behav Neurosci* 1987;101:724–731

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G K Beauchamp and K Engelman

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