The taste for salt in humans\textsuperscript{1–3}

Richard D Mattes

ABSTRACT Accumulating evidence indicates that the taste of salt is innately appealing to humans, although responses to salty foods are strongly influenced by environmental factors. Except in instances of severe, prolonged sodium depletion, a sodium-specific appetite has not been documented in humans. Limited data reveal no clear association between early exposure to salt and various hedonic responses to salt later in life, but recent exposure markedly alters a person’s preferred salt content of foods. Restricting exposure for 8–12 wk can enhance the appeal of reduced-sodium foods in both normotensive and hypertensive individuals. Although the appeal of the taste of salt is one factor contributing to its intake, the extent to which such a hedonic shift promotes long-term adherence to a reduced-sodium diet has not been determined. There is little evidence supporting a relation between either taste sensitivity or hedonic responses to salt and blood pressure. Am J Clin Nutr 1997;65(suppl):692S–7S.

KEY WORDS Salt, hypertension, taste, preference, chemosensory property, hedonic response, sodium, blood pressure, gustatory sense

INTRODUCTION

The basis for the high, apparently need-free, sodium chloride ingestion in populations with free access to the compound has not been established. Numerous explanations can be postulated. One possibility is that the ubiquity of sodium in the food supply makes adoption of a low-sodium diet difficult. Only 5–10\% of dietary sodium is under discretionary control (ie, table and cooking salt)\textsuperscript{(1)}, so reductions would entail substantive changes in food selection (eg, more fresh, less-processed items), storage, and preparation. Although the quantity and quality of reduced-sodium foods are increasing, the higher cost and inconvenience of a reduced-sodium diet are obstacles to dietary change.

Cultural factors also contribute to intake patterns. For example, a 10-fold difference in sodium intake was reported between two Solomon Island populations that was due to one group’s customary practice of steaming foods with fresh water whereas the other group cooked with sea water\textsuperscript{(2)}. Discretionary salt use by individuals is stable\textsuperscript{(3)} and may be dictated more by custom than by sensory influences. Covert manipulations of saltshaker pore size have not elicited changes in shaker use, and \approx 20\% of individuals salt foods before tasting them\textsuperscript{(4–6)}.

The possibility that current intake reflects a higher need should also be considered. Numerous populations with varied cultures adopt a similar, high intake, suggesting that there may be a biological basis for this behavior\textsuperscript{(7)}. Furthermore, normotensive individuals spontaneously increase intake to offset modest sodium losses caused by diuretic treatment\textsuperscript{(8)}.

The increased palatability of foods containing salt provides an additional, if not prepotent, incentive to ingest large amounts of the compound. In populations where salt availability is limited, its introduction results in a rapid and marked desire for the taste\textsuperscript{(9)}. When salt is readily available, foods characterized as predominantly salty make up a high proportion of total energy intake (\approx 40\% in one study)\textsuperscript{(10)} and acceptance of reduced-sodium foods has been slow.

The relative importance of each of these influences, as well as possible others not described, will undoubtedly vary for populations and for individuals over time. The consistent observation that palatability is a primary determinant of food selection suggests that the hedonic attributes salt imparts to foods are important determinants of its ingestion.

TRANSDUCTION OF SALT TASTE

The principal mechanism for transduction of the salty taste involves passage of sodium through a specific ion channel in the apical membrane of receptor cells\textsuperscript{(11)}. The channel can be blocked with the drug amiloride. Application of amiloride to the dorsal tongue epithelium can reduce the saltiness of sodium chloride in humans\textsuperscript{(12)}, although the consistency of this finding has been questioned\textsuperscript{(13)}. The channel is specific; lithium, which can also pass through readily, is salty whereas other cations (eg, potassium) that do not fit are not strictly salty. This specificity explains the difficulty in finding an acceptable salt substitute. Amiloride-sensitive channels account for \approx 70–85\% of the response to sodium chloride in rats but probably less in humans\textsuperscript{(14, 15)}. The mechanism for the residual function is not established but may involve chloride transport\textsuperscript{(15)}.

Sensitivity to salt, measured as thresholds for detection or recognition of the quality of sodium chloride, and suprathreshold intensity ratings for graded sodium chloride concentrations in taste stimuli are not significantly correlated with measures of salt preference\textsuperscript{(16, 17)}. Although a stimulus must be perceived

\textsuperscript{1} From the Monell Chemical Senses Center, Philadelphia.
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\textsuperscript{3} Address reprint requests to RD Mattes, Monell Chemical Senses Center, 3500 Market Street, Philadelphia, PA 19104-3308. E-mail: mattesr@cfs.purdue.edu.

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for its hedonic character to be judged, many factors influence
how that sensation will be interpreted.

**ONTOGENY OF SALT-TASTE SENSITIVITY AND
PREFERENCE**

Human neonates do not differentially ingest salt and water
solutions (18), suggesting that they either lack the ability
to detect the salty stimulus or judge it to be of equal palatability
to plain water. Studies in rats and sheep indicate that there is a
maturational lag in sensitivity to sodium chloride that is coin-
cident with evolving peripheral innervation patterns and develop-
ment of functional amiloride-sensitive channels in taste cells
(19). A similar pattern is now proposed for humans, who are
clearly able to detect the stimulus at \(\approx 4\) mo of age (18). From
4 to 24 mo of age, humans ingest saline in preference to water.
After this time, environmental factors are apparent. Children
from 31 to 60 mo of age reject saline solutions (as do adults)
yet find salty soup palatable (20). Adolescents, especially Afri-
can Americans, may prefer higher concentrations of salt in
foods than do adults (21), although definitive evidence for this
is not available.

Because environmental factors are important in shaping salt
preferences, concern has been expressed over a potential influ-
ence of early salt exposure on long-term salt preference and
intake. Few data are available on this issue. Recent exposure to
salty foods may influence short-term acceptability of items
with various sodium concentrations in 6-mo-old infants (22),
but a longer-term effect has not been observed. The most
rigorous test of this phenomenon involved supplying formula
and food containing either 4.78 \(\mu\)mol Na/kJ (2 mmol/100 kcal)
or 21.5 \(\mu\)mol Na/kJ (9 mmol/100 kcal) to infants for 5 mo
when they were 3–8 mo of age and assessing their hedonic
response to and intake of salty foods at the age of 8 y. No
significant group differences were noted for sodium excretion
or estimates of the child’s salt-use preference (23). There is
evidence that salt use during the second year of life is related
to intake at \(\approx 4\) y (24), but these data may simply reflect
tracking of family salt-use patterns rather than establishment of
a lasting salt preference. Other work indicates that the prefer-
ence for salt is not related to salt intake at 2 y of age (25).

**SODIUM APPETITE**

Salt appetite has been defined as "... a strong motivation to
seek, obtain, and ingest sodium" that is typically manifest in
cases of salt-wasting or need (26). Although the term is com-
monly used to describe salt preference, the two terms are
distinct, with the latter referring to the desire for items with a
salty taste in the absence of sodium need. Herbivores actively
seek out sodium chloride in preference to nonsodium salts,
presumably because of the low amount of sodium in their diet
(27). It has been argued that the diet of ancestral humans was
largely vegetarian and that the neural, hormonal, and behav-
ioral mechanisms that evolved to ensure adequate sodium
ingestion now underlie a purported salt appetite in humans
(27). Support for such an appetite stems from a case study in
which a strong craving for salt was exhibited by a child with
undiagnosed Addison disease (28) and several reports of indi-
viduals with sodium-wasting pathologies self-medicating by
ingestion of licorice, which possess mineralocorticoid proper-
ties (29, 30). However, salt craving occurs in only \(\approx 15\)% of
patients with Addison disease (31, 32) and experimental so-
dium depletion in humans is not accompanied by a strong and
consistent craving for salt (33). One explanation for these
discrepant findings may lie in the timing, duration, and severity
of a sodium depletion provocation. Accounts of manifest salt
appetite generally involve conditions in which an individual
experienced a lengthy period of marked sodium depletion early
in life. Animal studies indicate that such conditions may result
in enduring alterations in neurophysiologic taste responses
(34).

Studies documenting neurophysiologic alterations in humans
deprived of salt in infancy have not been undertaken, but one
natural experiment suggests that early deprivation can result in
long-term changes in responsiveness to the taste of salt. In the
late 1970s an infant formula, developed in response to public
concerns about sodium intake, was deficient in chloride. It
provoked an endocrine response characteristically associated
with salt appetite in animals. A recent follow-up study of 169
adolescents who consumed this formula during infancy re-
vealed a heightened preference for salt relative to the prefer-
ence of their nearest-aged siblings (35). In general, salt appetite
is extremely rare in humans and may appear only under ex-
treme conditions.

**SENSORY AND METABOLIC INFLUENCES ON SALT
PREFERENCE**

Long-term adherence to a diet reduced in sodium can lead to
a hedonic shift wherein normotensive (36–41) and hyperten-
sive (42, 43) persons develop an increased acceptance of foods
with reduced sodium contents. This downward hedonic shift
generally requires \(\approx 8–12\) wk to occur (40, 41). Although a
similar change was reported after adherence to a diet for only
2 wk (44), most data indicate that there is an initial transient
increase in preferred saltiness (45–47). The shift is not associ-
ated with a change in ability to detect or judge the saltiness of
foods (40, 41, 44). The mechanism underlying the diet-induced
hedonic shift is primarily based on sensory exposure to the
salty taste rather than altered sodium metabolism or amount
consumed. When sensory exposure is restricted through pro-
scriptions on discretionary (table and cooking) salt use but
intake is essentially unchanged, a hedonic shift occurs (40).
Alternatively, if intake is reduced substantially but sensory
exposure is maintained through free access to discretionary
salt, hedonic ratings remain unchanged (36). Increased sensory
exposure, independent of intake, can elicit a shift toward a prefer-
ence for higher concentrations of salt in foods (48).

Not all studies tracking salt preference in individuals adher-
ing to a reduced sodium diet reveal a significant hedonic shift.
A nonsignificant trend only was noted in one trial with un-
treated hypertensive men (49). This discrepant finding may be
explained by the greater variance in hedonic responses to salty
stimuli shown by prehypertensive and hypertensive adults (50),
which may, in turn, reflect differences in hypertension etiol-
ogy. Recently, we began exploring the relation between salt
sensitivity and hedonic responses to dietary sodium restriction
in normotensive persons. This population was selected because
a hedonic shift would presumably aid in adherence to a re-
duced-sodium diet, and salt-sensitive individuals would be a primary target for dietary intervention.

To date we have collected a complete set of data from 15 healthy normotensive adults. Each subject adhered to a reduced-sodium diet for 4 d followed by a high-sodium diet (customary diet plus 10 g NaCl as enteric tablets [Eli Lilly, Indianapolis]) for 7 d on two occasions separated by 2 wk. Salt sensitivity was diagnosed after each trial on the basis of a rise of sitting mean arterial pressure of \( \geq 5 \) mm Hg from the last day of the low-sodium period to the last day of the high-sodium period. Further, classification was attempted only for individuals who, on the basis of sodium excretion in urine, ingested \( \geq 50\% \) more sodium during the high-sodium period than during the low-sodium period and whose intake was \( \leq 100 \) mmol lower and higher than their own baseline intake during the low-sodium and high-sodium periods, respectively. Eight subjects (five salt-sensitive subjects and three salt-insensitive) met these criteria, were classified the same way on both trials, and completed a 4-mo period during which sodium intake was restricted according to guidelines for a reduced-sodium diet published by the American Dietetic Association (51).

Hedonic responses to 10 foods (bread, cheese, chicken broth, crackers, canned green beans, margarine, peanuts, potato chips, tuna, and vegetable juice) that are commercially available in a “regular” and a “reduced-sodium” form were obtained by using a 20-point category rating scale before diet initiation and monthly during the diet period (Figure 1). A significant hedonic shift occurred wherein the reduced-sodium products became more acceptable over time (\( F_{4,24} = 4.42, P = 0.008 \)). Although the regular-sodium versions were rated as more pleasant at baseline than their reduced-sodium counterparts (\( P < 0.001 \)), by the end of the dietary period no significant difference was reported. Additional testing will be required to determine whether salt-sensitive individuals have a delay in their shift. These preliminary data indicate that ratings of salt-insensitive individuals for regular and reduced-sodium items were similar by month 2 whereas this did not occur until month 3 for salt-sensitive individuals. The data document that a hedonic shift occurs in salt-sensitive individuals. Whether this shift contributes to long-term dietary adherence in this group has yet to be established.

**SALT TASTE AND HYPERTENSION**

Sodium chloride, the compound implicated in the onset and manifestations of hypertension, is the prototypical salt-taste stimulus. On the basis of the premise that sodium reception mechanisms on the tongue mirror activity elsewhere in the body, it has been postulated that measures of taste function will provide information useful for predicting, diagnosing, and managing hypertension.

Early studies focused on taste sensitivity as measured by thresholds. No evidence of a difference in detection thresholds (the lowest concentration that can be detected in a given medium) was obtained in comparisons of normotensive and hypertensive persons (52–54). Several studies revealed elevated recognition thresholds (the lowest concentration of sodium chloride that evokes a recognizable salty taste) in hypertensive subjects (52, 55–57), but other studies did not note a difference (53, 54). Further, no correlation between recognition threshold and blood pressure was observed in a study of 95 subjects aged 11–16 y (17). An explanation for these discrepant findings is not apparent.

The ability to distinguish between and assign saltiness intensity ratings to graded suprathreshold concentrations of sodium chloride has also been examined. Comparisons of ratings from normotensive and undifferentiated hypertensive individuals have generally revealed no differences (50, 58). One study noted a slight nonsignificant diminution of intensity ratings for high-salt concentrations by hypertensive subjects (59). Psychological functions describing the increase of saltiness with concentration are nearly superimposable (50). This was noted in an investigation involving patients with low renin activity and mild hypertension and control subjects (60) and in another assessing responses of salt-sensitive and salt-insensitive normotensive persons (61). Thus, there is little evidence for impaired salt perception in individuals with hypertension.

In studies exploring the association between hedonic ratings for salty foods and blood pressure, no association was observed in adolescents (17) or in college students (62). Salt-sensitive and salt-insensitive normotensive black adolescents also provided comparable hedonic responses to salted stimuli and, when given free access to discretionary salt, prepared samples with similar sodium concentrations (61). A heightened preference was reported for hypertensive patients consuming an extremely low-sodium diet (9 mmol/d) compared with normotensive control subjects during the first 4 d of a 7-d trial (63). The extreme conditions of this study, the lack of a consistent effect, the use of saline ingestion (a uncommon dietary item) as the index of preference, and the observation that the patients ingested very low amounts of salt when given free access after the trial raise questions about extrapolating the findings to free-living individuals.

Equivocal findings were also obtained from a study in which heightened hedonic responses for saline solutions were obtained from hypertensive patients relative to control subjects with only one of the two response scales used (60). No significant differences were observed in several other investigations using various foods and multiple response formats (eg, ratings

![Figure 1: Mean (± SE) hedonic ratings of 10 foods sampled in regular-salt (REG-Na) and reduced-salt (RED-Na) forms by five salt-sensitive (SS) and three salt-insensitive (SI) subjects before the initiation of a reduced-sodium diet (0 mo) and at monthly intervals during a 4-mo diet period. Hedonic ratings were obtained on a 20-point category scale with 20 labeled “like extremely” and 1 labeled “dislike extremely.”](https://academic.oup.com/ajcn/article-abstract/65/2/692S/4655402/1)
of foods prepared with graded sodium concentrations, subject adjustment of salt concentrations in foods, and questionnaire ratings of high- and low-sodium foods) (50, 59). Taken together, the data do not reveal a heightened preference for salt by persons at risk for hypertension (ie, normotensive with high-normal blood pressure or salt sensitive) or with manifest hypertension.

One study suggested that diuretic treatment increased salt-taste sensitivity in hypertensive patients (64), and work with amiloride, a potassium-sparing diuretic, reveals that topical application can reduce salt-taste responsiveness (12). However, subsequent investigations have failed to support these findings and indicate that diuretics and β-blockers also do not alter salt-taste intensity or preference responses (8, 36).

SALT PREFERENCE AND SALT INTAKE

Interest in the association between salt preference and hypertension stems from an assumption that hedonic responses to salt are related to sodium intake. Although manipulations of sodium intake can modify salt preference (35–40, 43–46, 48), under stable intake conditions the association is not strong. Of the eight studies comparing sodium intake and hedonic ratings, five noted a significant association (16, 60, 65–67), one reported a trend (62), and two observed no relation (10, 68). The lack of consensus may be related to the methods used. Intake has usually been assessed by insensitive methods (eg, 24-h urinary excretion and unvalidated questionnaires) and preference has been poorly characterized. Preference is a multidimensional attribute: preferred concentrations of sodium in foods, hedonic ratings for foods with a salty taste, and preferred frequency of ingestion of salty items all contribute unique information. Combined, these different measures hold significant predictive information (16).

SALT PREFERENCE AND MODIFICATION OF SALT INTAKE

Maintaining the palatability of foods constituting a reduced-sodium diet is important for long-term adherence. Different approaches can be used to facilitate the adoption of a reduced-sodium diet. One strategy entails increased use of reduced-sodium versions of normally ingested products that have been engineered to maintain their sensory profile. The number, diversity, convenience, and organoleptic quality of such items have increased markedly over the past decade, so little disruption of dietary practices would be required. This does not necessitate elimination of discretionary salt use because this source usually contributes little to total intake and is not markedly increased when other sources are reduced (1, 47). Indeed, the vigorous defense of discretionary use suggests that attempts to eliminate it might compromise diet adherence (39). Consumer addition of other spices and flavors may enhance the appeal of reduced-sodium foods, but the success of this approach is highly dependent on the product and individual tastes.

An alternative approach is to exploit the ability to adjust salt-taste preferences. Whether the shift that occurs after 8-12 wk of limited sensory exposure to the salty taste effectively promotes long-term dietary compliance has not been determined. Retraining the palate would likely entail a substantive change in food selection wherein fewer convenient prepared foods would be included. The hedonic shift can be reversed by renewed exposure to the taste, so vigilance is required, and factors other than taste influence dietary habits. This tactic would require a proscription against discretionary salt use that could be difficult for some individuals.

Melding these two approaches appears to be problematic because use of reduced-sodium foods engineered to retain their original salty taste would prevent a hedonic shift. Matching the strategy with the needs and desires of the individual will likely be most effective.

PHYSIOLOGIC EFFECTS OF SALT TASTE

The perception of a taste stimulus, such as sodium chloride, not only elicits a sensory response, but also activates a wide array of physiologic processes. These rapid, neurally mediated, cephalic-phase responses include stimulation of salivation, secretion of gastric acid and increased gastric motility, release of pancreatic digestive enzymes and hormones, enhanced thermogenesis, and alterations of cardiovascular and renal function (69–73). Sodium chloride may elicit these responses through its specific taste or via an influence on a food’s palatability. The physiologic significance of these responses and the particular role of sodium chloride as an elicitor of them has not been established. Consideration of this issue will be necessary before the full range of health implications of reducing sodium chloride in foods can be determined.

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HUMAN SALT PREFERENCE