

## REPRINTS AND REFLECTIONS

# Possible role of salt intake in the development of essential hypertension\*

LK Dahl

## Introduction

Although the practice of adding salt to food is an ancient one, there seems to be little doubt that until relatively modern times its widespread use as a condiment was uncommon. At present, the practice is a ubiquitous one and, in the United States at least, salt may be added at any stage before, during or after food-processing as well as before, during or after cooking. And sometimes salting takes place during each of these steps!

The ancient valuation of salt as a precious possession<sup>1</sup> may have contributed to the modern notion that the addition of salt to food is necessary or even beneficial.<sup>2</sup> Nonetheless, during the 20th century, there has been evidence accumulating which suggests a possible relationship between salt ingestion and hypertension in man.<sup>3</sup> In this paper we will review the sum total of the evidence, which we have been gathering since 1954.<sup>1,4–14</sup> The original papers should be consulted both for the primary data as well as for correlation with the experiences of others in this field. These studies are in line with the modern effort against subtle lethal agents as, for example, fall-out, carcinogens and atherogenic factors.

## Salt need, salt intake, salt appetite

We have dealt with these considerations at some length elsewhere.<sup>9–14</sup>

### Need

There is no doubt that some salt is required by man, and estimates of normal daily requirements for adults have ranged up to 15 g per day. For the most part, such estimates were arrived at by a circular argument in which, over any given period, the amount of salt excreted in the urine was equated with the need for ingestion of an equal amount in order to maintain metabolic balance. As opposed to such opinion there are numerous careful metabolic studies which indicate unequivocally that in people with normal renal function, salt balance can be maintained easily on daily intakes well below 1 g. Our own group has studied many individuals who were limited to 100–375 mg for periods of 3 to 12 months.<sup>15–18</sup> We have reported 3 subjects whose intake was proven to have been restricted to 250–375 mg NaCl continuously for periods ranging

from about 2 to 5 years.<sup>13</sup> We recently studied a 17-year-old girl for several months who easily maintained salt balance while her daily intake was only 10–12 mg of salt. We do not imply that such low intakes are either necessary or advisable, but we do suggest that under ordinary circumstances the adaptive mechanisms of the body are so exquisitely effective in conserving salt that intakes of only 1 or 2 g a day are more than sufficient for metabolic needs, including periods of growth.<sup>1</sup>

### Intake

There are—*were* is perhaps more accurate today—many vigorous peoples which for countless generations added no salt to their food and ate only that which was present naturally. Among such groups are the Eskimos, some of the northwest American Indians, and the Masai of Africa. Analysis of the diets among such groups or maximal estimates based on the rather constant salt content of foods in their natural state indicate maximal daily intakes of not more than 5 g, with some eating 1 g or less.<sup>1</sup> Calculations based on the analyzed sodium content of known foods indicate that salt intake rarely could exceed 4 or 5 g per day without the addition of salt to food, save in areas where drinking water had a high salinity.

Determinations of actual as contrasted with estimated salt consumption are remarkably few in all societies, and no less so in western than in other societies. Measurement of 24 hr urinary excretion is a reliable index of *minimal* salt intake. Except when sweat losses are significant it is even an accurate index of total intake<sup>1</sup> in normal people. In view of the ubiquitous sources of salt in modern diets the errors of this technique are in my opinion less than those of any other method. There are two criticisms of this means of evaluating salt consumption: 1. losses from the skin as well as inevitable losses from lack of total co-operation will cause underestimation of salt intake in some subjects; and 2. salt consumption may vary so widely from day to day that a single or even several consecutive 24 hr collections will hardly give an accurate index of a person's average intake. These criticisms are more applicable when determining *maximal* consumptions but less so when *minimal* consumptions are being investigated.

Similar problems have existed in estimating the exact consumption by a single individual of such common items as fat, cigarettes, or alcohol, but excesses are well recognized here, whether committed by an individual or a nation. I suggest that this be also applied to salt: single 24 hr urine collections may be inaccurate, but my own considerable experience during the last ten years with five different peoples in five different parts of the

Published in Cottier P, Bock KD: *Essential Hypertension—an International Symposium*. Berlin: Springer, 1960, pp. 53–65. Reprinted with permission.

\* This work was supported by the United States Atomic Energy Commission.

world indicates that this method is an excellent index of average salt consumption by a group as well as by individuals. It has been checked against known national and community salt purchases in Japan, where salt is controlled by a monopoly; against actual 24 to 48 hr diet collections in Japan, in the United States, and among natives of the Marshall Islands in the Pacific; and finally against known salt content of natural foods eaten by Alaskan Eskimos.

By the use of such methods my associates and I have found that Eskimos were consuming an average of less than 4 g of salt per day, Marshall Islanders about 7, white male Americans about 10, and Southern Japanese farmers and laborers about 14 g. Northern Japanese farmers were found to average 26.3 g by my Japanese friend and investigator, Dr. Fukuda of Chiba University. Studies on Eskimos, Marshallese, and Americans (including the southern negro) are still in progress and shall be periodically reported.

These group averages indicate that salt intakes may and do vary widely among different peoples. However, these figures will be misleading if they are interpreted to mean that all individuals in such communities are consuming equivalent amounts of salt, since we have observed a wide diversity in individual salt consumption if salt is readily available. Just as data based on average alcohol consumption fail to indicate either the total abstainer or the chronic alcoholic, so average levels of salt consumption fail to show that, among individual members of the communities we have studied, some people habitually eat very little salt while others consume it gluttonously.

The accompanying table (Table 1) summarizes these data up to the present time: the variable *range* of values in the several communities may be as informative as the variation in *averages*. Comparison of these data on salt intake with the earlier estimates of metabolic need indicates a very considerable excess of intake over need in some societies. The importance of this disparity is basic to the thesis of this paper.

I have often been asked whether an average difference of only a few grams of salt per day could be important. In this regard, it is critical to remember that 'grams per day' defines a *rate* as opposed to a *quantity*. Relatively small rate differentials operating over long periods of time can yield striking disparities in end result, as the fable of the tortoise and the hare aptly illustrates. From some data now in preparation for publication, our group has found by means of Na<sup>22</sup> turnover studies that the biological half-life of sodium in humans is a clear-cut function of salt intake. There is more than a two-fold difference in

turnover rate between intakes of 2 and 5 g, or between 5 and 10 g; there is a five-fold difference between the effect of 2 and 10 g, and a ten-fold difference between 2 and 30 g. The thesis that such marked differences are without physiological implication appears untenable.

## Appetite

Detailed consideration of the role of salt *appetite* as it relates to salt *intake* is beyond the scope of this paper. There is no doubt that salt appetites exist, as every reader can personally testify. The issue is whether salt appetites are innate or acquired. Among animals, the long treks to salt-licks are well known; less well known is the fact that the herbivores rather than the carnivores do this. Whether the high dietary K/Na ratio of about 20 : 1 in the herbivores as contrasted with the 5 : 1 ratio in the diet of the carnivores is important is not known.

We have never seen evidence of salt-craving in our subjects whose salt-intakes were drastically reduced for months or years. Reports by Stefansson<sup>19</sup> and Holmberg<sup>20</sup> show that the primitive Eskimos and Bolivian Indians, with whom they were associated respectively, did not like salt initially, although they could grow to like it rapidly. Within my own family, where the children were reared without added salt, there was no evidence of a salt appetite until it was induced by well-meaning friends. Furthermore, it seems clear that if an innate appetite exists it is surprisingly easy to change, in contrast to some of the better-established ones. In our patients who were on diets containing only 100 to 250 mg of NaCl per day, the addition of as little as 0.5 to 1 g to their total daily intakes evoked initial comments of 'too salty', although later adaptation occurred. In sharp contrast, among subjects on 10 to 20 g per day, the addition of 5 or 10 g went unnoticed. Thus it seems evident that salt appetite is induced rather than innate. This is fundamental, since having shown that salt *intake* bears no necessary relationship to salt requirement, it now seems highly likely that salt *appetite* is also unrelated to requirement.

## Evidence that salt ingestion may be related to hypertension

### A) Experimental hypertension

In several forms of experimental hypertension, the simultaneous ingestion of excess salt appears to be necessary. Grollman and his associates were the first to demonstrate that various sterols were hypertensogenic only if additional salt was provided.<sup>21</sup> The effectiveness of desoxycorticosterone acetate in producing hypertension with the addition of extra salt is well known.<sup>22,23</sup> Salt feeding with fluid restriction by means of hypertonic saline as the sole source of liquid has been used to produce hypertension in the chick,<sup>24</sup> rat<sup>25</sup> and rabbit.<sup>26</sup> Finally, Meneely and his collaborators have shown that chronic ingestion of excess sodium chloride *alone* will produce a hypertension in rats which mimics human hypertension morphologically.<sup>27-30</sup> We have been using this same technique in our laboratory for some years and have confirmed the potential of chronic salt-feeding to produce hypertension. I suspect that inability to confirm Meneely's work may be caused by failure to engage in long-term, chronic experiments. To investigators whose previous experience has been with the

**Table 1** Average daily salt intake (based on 24 hr urine excretion) in several societies

| Group                                | Year       | Sex  | Salt Intake   |             |
|--------------------------------------|------------|------|---------------|-------------|
|                                      |            |      | Average (g/d) | Range (g/d) |
| Alaskan Eskimo                       | 1958, 1960 | both | 4             | 1-10        |
| Marshall Islander<br>(Pacific Ocean) | 1958       | both | 7             | 1.5-13      |
| United States<br>(Brookhaven)        | 1954-1956  | male | 10            | 4-24        |
| Japan                                |            |      |               |             |
| Hiroshima (South. Japan)             | 1958       | male | 14            | 4-29        |
| Akita (North. Japan)                 | 1954       | both | 26            | 5-55        |

rapidly evolving varieties of experimental hypertension, that which follows chronic salt-feeding will seem negligible or non-existent because of its slow onset. Nonetheless, from extensive personal experience I would like to state unqualifiedly that hypertension ultimately will result in most rats that are chronically fed extra salt. Its onset may occur at different intervals following the onset of salt-feeding; its character may vary from mild to severe; however, once present, it rarely if ever disappears if salt-feeding is continued. It is usually slowly progressive, but may reach only a modest level and remain on this plateau for the remainder of the animal's life; in contrast we see animals whose disease is marked by early onset, rapid severe elevation of blood pressure and death within a few months. In all of these respects it must be conceded that it resembles the picture of the human disease more closely than the commoner varieties of experimental hypertension.

Hence, chronic salt-feeding in rats can produce a picture resembling human essential hypertension. Furthermore, the evidence is unequivocal that, as the *amount* of salt ingested daily is increased, both *incidence* and *severity* of the hypertension will be increased.

## B) Human hypertension

Let us now turn to man. Here, salt restriction has long been used as part of the therapeutic regimen against established hypertension. Much of the early work either failed to separate sodium from chloride or frankly ascribed the results to chloride. In 1945 Grollman and his associates clearly demonstrated that it was the sodium restriction which was important.<sup>31</sup> This idea is today so widely accepted and used that dilation would belabor the obvious.

Although there were a number of carefully controlled metabolic studies which had established the usefulness of sodium restriction in hypertension, widespread acceptance of this fact came only after the recent development of effective, relatively non-toxic natriuretic agents such as chlorothiazide. Here, too, salt ingestion seems to modify the response, since a high salt diet appears to limit or even block the hypotensive response to chlorothiazide.<sup>32</sup>

In our experience, which is now much more extensive than the early report,<sup>15</sup> addition of salt to the diets of individuals who have responded to its restriction generally results in a return of the elevated blood pressure. Furthermore we have found a few reports indicating that the addition of salt to the diet of *normotensive* individuals for short periods of time has resulted in significant elevations of pressure. McQuarrie<sup>33,34</sup> reported that some diabetic children rapidly became hypertensive when salt was presented, and McDonough and Wilhelmj<sup>35</sup> made similar observations on a normal young adult male.

Failure to observe a rise in blood pressure on short-term salt-feeding is not surprising to me for two reasons: 1. If it be granted that excess salt ingestion plays a primary role in the etiology of human hypertension, then the paucity of essential hypertension before the 4th decade would suggest that the salt effect must operate over a considerable span of time, possibly starting well before maturity is reached. Both our own experience as well as that of Meneely's group<sup>27-30</sup> indicates younger animals are more susceptible and that the development of significant disease ordinarily requires a third or more of the animals' expected life-time. 2. Even if salt-feeding

is continued throughout the life of a colony, some 20% of the rats remain normotensive: this variability in response to salt-feeding, exhibited by a relatively inbred species like the Sprague-Dawley rat, must operate to a much higher degree in an animal with the mixed ancestry of man. It is intriguing to speculate on the possibility that a susceptibility to salt could be bred in or out of isolated communities depending upon the original make-up of the members.

It will be recalled that earlier in this paper we stated unless drinking water had a high salinity it was unlikely that on the average more than 4 or 5 g of NaCl per day would be ingested from foods not salted by man; ordinarily it would be less, particularly among people who were largely vegetarians. Since hypertension appeared to be *uncommon* among groups which did not add salt to their food, we thought that it might be *common* among groups which did add it and that the higher the salt consumption, the higher the incidence of hypertension. This would have been in agreement with our experimental animal data as well as those of Meneely and collaborators.<sup>27-30</sup> The remainder of this paper will be devoted to exploring the studies which we have made to test this hypothesis.

Preliminary to epidemiological investigations, we considered a pilot method of estimating average salt intakes in an effort to judge whether this hypothesis warranted the time and expense of a formal study. In our milieu, salt shakers are ubiquitously available. Therefore some inferences might be made from an individual's use of salt at the table. We arrived at the following 3 classifications: 1. Low intake—did not add and never had added salt to food at all; 2. Average intake—added salt to food only if, after prior tasting, it was insufficiently salty for the palate; 3. High intake—added salt to foods routinely without prior tasting for degree of saltiness.

We were aware then, as we are now, of the defects in such a classification: 1. It is qualitative and not quantitative; 2. No allowance is made for addition of salt to food prior to its arrival at the table; 3. No provision is made for differences in sensitivity to saltiness among different individuals or even the same individual at different times. In spite of all these reservations the pilot technique proved to be a useful one in our hands. Nevertheless there was no suggestion then, nor is there now, that it is a generally applicable technique, for it clearly is dependent upon salt-eating practices in a community. We have evidence that salt-adding customs may be very different in the northern area, where the original study was made, from those in the deep south of the United States, where a small rural community is now under study. This technique would have been misleading in Japan, where salt consumption is high, but where I found that the salt ordinarily was added to foods and sauces prior to reaching the table.

It would be unfortunate if this pilot study were interpreted to mean that we were or are interested in salt-shakers. On the contrary, let me state categorically that what we *are* interested in is *actual salt consumption*, whatever its source and however it gets into the gastro-intestinal tract.

With the use of the pilot technique we obtained information which was suggestive of a relationship between salt consumption and hypertension. Upon completion it indicated that further expense and effort were warranted in exploring this area. During the years 1953-1956, Dr. Robert A. Love, at Brookhaven Laboratory, kindly queried for me all employees

upon whom he made a physical examination as to their customary salt-adding habits, according to the classification above. The incidence of hypertension among these 3 groups was significantly different from random distribution ( $p < .001$ ). Those classified as having been on low intakes throughout their lives showed significantly less hypertension ( $p < .01$ ) and those classified as having been on high intakes showed significantly more hypertension ( $p < .02$ ) than would have been predicted by chance alone.<sup>8</sup> We have assessed the validity of this method of estimating salt intake in a series of 28 males who were willing to collect 24 hr urines for from 6 to 38 days and found that the average of those classified as 'Low' was significantly lower ( $p < .01$ ) than those who had been classified as 'High'.<sup>7</sup> More importantly, the non-hypertensives appeared to be eating significantly less ( $p < .01$ ) salt than the hypertensives. We have pointed out<sup>36</sup> that in this relatively young group of males ( $40.3 \pm 10.6$  years) even the non-hypertensive individuals classified as being in the 'Low' category were consuming on the average about nine and a half grams per day. On the basis of such intakes I expect some of these men to develop hypertension later in life.

These data indicate the probability of a *group* developing hypertension. It would be most fallacious to surmise that a *person* who consumed a given amount of salt would carry the same probability as the *group*. An individual obviously would have, as a rule, either a higher or a lower probability than the group, depending upon the type of distribution defining his particular group.

We were encouraged by these results to explore actual salt intake among groups in which the prevalence of hypertension is variable. These data are summarized briefly in Table 2, which is an extension of Table 1, as well as in the accompanying graph (Fig. 1). These data suggest in conformity with the animal data that with higher average salt consumption there is higher prevalence of hypertension. It will be of interest to compare these data with those from other areas as they become available. The small number of Eskimos that have been studied obviously requires the amplification in which we are now engaged. However, most experience with Eskimos suggests that hypertension was uncommon or rare among those who were on a truly native diet and therefore did not add salt to the food.<sup>37-40</sup>

The recent report by Scott *et al.*<sup>41</sup> is in contrast to the earlier reports on Eskimos in that hypertension was found to be as prevalent among them as among men in the United States. This report may be of great pertinence to the present work: these Eskimos were not primitives any longer, since they were all

sufficiently civilized to join the Alaska National Guard. No details on their salt intakes are available, however.

In Japan, hypertension is a common disease. Average levels of salt intake are high, and interestingly, appear to decrease from north to south.<sup>42</sup> In association with this, both the incidence of hypertension and the incidence of cerebrovascular accidents decrease from north to south. In Fig. 2, we have summarized the known data on average salt intakes and death rate from cerebral hemorrhage in Japan. Since 1951, cerebrovascular complications of hypertension have been the leading cause of death in Japan, as well as one of the leading causes of death in that country during the 20th century.<sup>42</sup>

What of factors other than salt? The highly sophisticated members of this symposium will be aware that other possible factors have been omitted from this discussion. Among several, one which is most intriguing to us is the likelihood of an interaction between salt and susceptible tissues, that is, the interaction of environment with heredity: the environmental factor in this instance is represented by dietary salt levels.

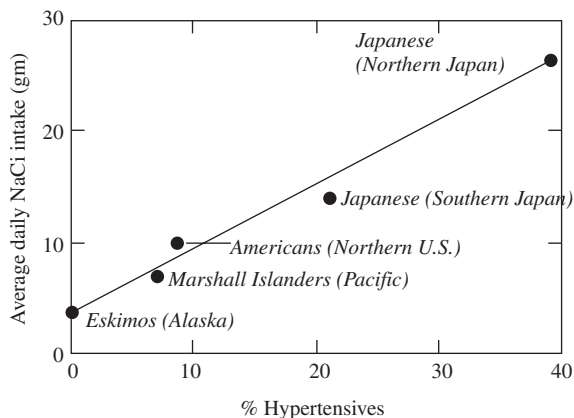
There is considerable evidence which suggests that hereditary factors operate in human hypertension. This has been well reviewed by Bechgaard,<sup>43</sup> Platt,<sup>44</sup> Soby, <sup>45</sup> Schroeder,<sup>46</sup> and Pickering,<sup>47</sup> among modern authors. In an earlier publication we discussed this possibility citing rheumatic fever as a disease in which hereditary susceptibility is important, but a concomitant streptococcal infection<sup>8</sup> is necessary for development of the disease. There are many similar examples of which only a few will be cited. The case with which certain families develop hay-fever after exposure to rag-weed suggests an inherited susceptibility to this allergen, but in the absence of rag-weed pollen hay-fever will not develop. Parkinson-like syndromes have been seen in miners inhaling manganese dusts but, in spite of the specific chemical agent, illness of a given individual indicated to Rodier<sup>48</sup> that it would develop subsequently more readily in relatives than in a general population that was similarly exposed. Furthermore, the susceptibility to Parkinsonism which develops as a toxic complication in about 7-10% of individuals chronically treated with chlorpromazine<sup>49</sup> was recently linked to prevalence of 'spontaneous' Parkinsonism in the individuals' families.<sup>50</sup>

These considerations lead us to evaluate the concept of a *lethal dose* as it might relate to salt intake and the development of hypertension. An LD<sub>50</sub> dose of a toxic or infective agent is premised on the established fact that biological responsiveness is inconstant even within highly inbred organisms. One might cite the variable response of members of the same strain of

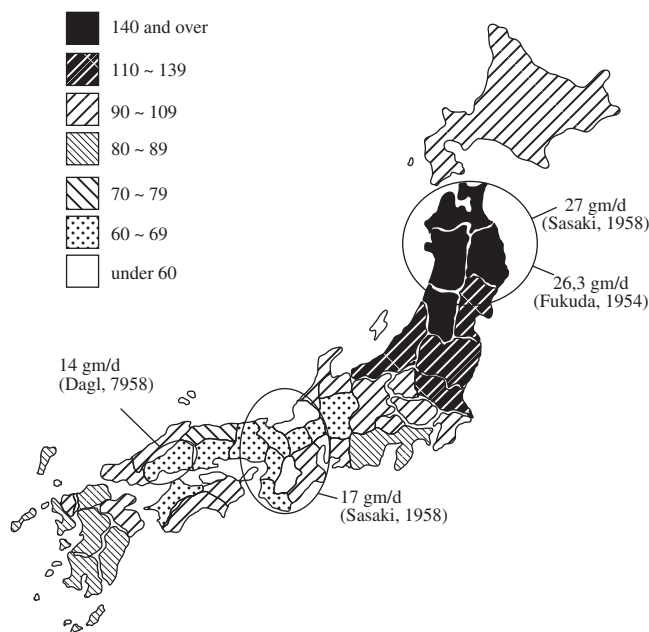
**Table 2** Salt intake (measured by urinary salt excretion) compared with prevalence of hypertension in five geographic areas

| Group                        | Year       | Sex  | No.  | Age (average) | Salt Intake   |             | % HT<br>(140/90 or over) |
|------------------------------|------------|------|------|---------------|---------------|-------------|--------------------------|
|                              |            |      |      |               | Average (g/d) | Range (g/d) |                          |
| Alaskan Eskimo               | 1958, 1960 | both | 20   | 38            | 4             | 1-10        | 0                        |
| Marshall Islanders (Pacific) | 1958       | both | 231  | 41            | 7             | 1.5-13      | 6.9                      |
| United States (Brookhaven)   | 1954-1956  | Male | 1124 | 36            | 10            | 4-24        | 8.6                      |
| Japan                        |            |      |      |               |               |             |                          |
| Hiroshima (South. Japan)     | 1958       | male | 456  | 43            | 14            | 4-29        | 21                       |
| Akita (North. Japan)         | 1954       | both | 5301 | 45            | 26            | 5-55        | 39*                      |

\* Systolic and diastolic pressures reported separately. This number (39%) based on diastolic pressure of 90 mm Hg or more.



**Figure 1** Correlation of average daily salt (NaCl) intakes with prevalence of hypertension in different geographic areas and among different races



**Figure 2** Distribution of death rate (per 100 000 males aged 30-59 years) from cerebral hemorrhage, in various regions of Japan. Average daily salt intakes of farmers in 4 regions added to original. (Taken, with additions, from TAKAHASHI *et al.*, *Human Biol. (U.S.A.)* 29, 139 (1957).)

bacteria to antibiotics or of mosquitoes to DDT. Obviously much greater variability in response must be possible in any single individual human under the usual conditions of enforced outbreeding in man. In the most virulent epidemics some people survive unharmed. Many individuals who smoke 2 or more packs of cigarettes per day will survive to old age without evidence of bronchogenic carcinoma. We venture to say that the same applies to chronic excessive salt intake. The failure of some or indeed most individuals to develop hypertension following chronic excessive salt intakes is, in my opinion, comparable to the failure of most individuals to develop other common diseases in which chronic exposure to the etiological

agent is established as a requisite. It does not mean that salt is not involved but rather that not *only* salt is involved.

## Summary

We wish to reiterate what we have said before:<sup>11</sup> Among societies or groups habitually consuming low salt diets (perhaps 5 g of NaCl per person per day or less) essential hypertension will be uncommon. Among societies or groups consuming high amounts of salt (in excess of 10-15 g per person per day) essential hypertension will be common. Individual susceptibility will determine which one individual in a group will develop the disease.

## Résumé

L'hypertension essentielle se rencontre plus rarement dans les sociétés ou communautés consommant d'ordinaire des rations alimentaires pauvres en sel (par exemple 5 g ou moins de NaCl par personne et par jour) que chez celles qui en consomment de grandes quantités (plus de 10-15 g par personne et par jour). La susceptibilité individuelle reste le facteur essentiel pour déterminer l'apparition de la maladie.

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## Commentary: Possible role of salt intake in the development of essential hypertension

Niels Graudal

Salt is one of the cornerstones on which the mammalian biochemical structure is built. Total exclusion of salt from the diet leads to disaster, namely death.<sup>1</sup> Still, salt is considered by some authorities, to be toxic on a level comparable with alcohol and tobacco. Why is salt the only essential component of mammals to have obtained this unattractive status? As of 2004, the history behind this is exactly 100 years old and LK Dahl<sup>2</sup> plays an important role in this history. The purpose of the present commentary is to discuss one of Dahl's papers, 'Possible role of salt intake in the development of essential hypertension' from 1960. To do this it is important to consider the paper in the historical context in which it was written.

The modern salt saga started in 1904 with a paper by Ambard and Brochard<sup>3</sup> who showed an association between salt intake

and blood pressure in six patients. On the basis of these observations they created a salt–blood pressure hypothesis. Subsequently in 1907 the results were opposed by Löwenstein,<sup>4</sup> and from then on the salt–blood pressure hypothesis has been the basis for a dispute between supporters of the hypothesis and sceptics. What we can learn from this is that the salt–blood pressure hypothesis and the controversy dates back to the first decade of the previous century, initially based on a few case histories.<sup>3,4</sup> To begin with the chloride ion was thought to play an important role, but with the paper by Blum in 1921<sup>5</sup> the dominant element was gradually concluded to be sodium. At that time the discussion started in the USA with Allan's recommendations of rigid salt restriction for hypertensive patients.<sup>6</sup> In the following years Allan's positive results were both confirmed and disproved by several authors, but during the late 1930s the use of salt restriction faded. In 1944 and in the following years the salt controversy was renewed with the introduction of Kempner's rice diet.<sup>7,8</sup> Dahl enters the scene

Department of Internal Medicine/Rheumatology Q, Copenhagen University Hospital at Herlev, DK-2730, Herlev, Denmark.  
 E-mail: niagr@herlevhosp.kbhamt.dk