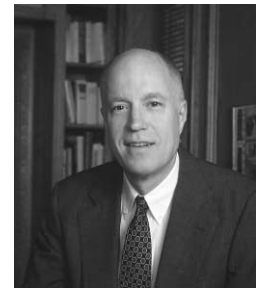
High Salt Intake, Its Origins, Its Economic Impact, and Its Effect on Blood Pressure

A number of years ago, when dining, I would cover the food on my plate with a heavy dose of salt before taking a single bite. After a while, I noted my blood pressure was a bit elevated and I abruptly stopped adding salt at the table. Within a month I found that I did not miss the added salt one iota. Unfortunately, salt added at the table accounts for only about 15% of the salt most Americans consume. Why is it that salt, so beneficial as a preservative and added to food for thousands of years, should only in recent years be discovered to be so harmful? MacGregor and de Wardener[1] in 1998 published a splendid book entitled *Salt, Diet & Health: Neptune's Poisoned Chalice: the Origins of High Blood Pressure*. The information that follows is taken entirely from their work, which describes how humans became addicted to salt, how it played an important economic and historical role, and how it became recognized as being so deleterious to our health.

SALT ADDICTION

Salt, of course, means sodium chloride, which is 40% sodium and 60% chloride. For 5,000,000 years our ancestors ate a diet to which no salt was added! Humans, like other mammals, relied on the small amounts of salt naturally present in food to provide enough to regulate the amount of fluid in our bodies. Powerful mechanisms for conserving salt within our bodies were developed. The addition of salt to food began 5,000 to 10,000 years ago, and then the problem of getting rid of the excess was produced. Our consumption of salt today is 10 to 20 times greater than 5,000 to 10,000 years ago. Because it had been geared to conserve salt, the human body found it difficult to dispose of this relatively sudden, in evolutionary terms, increase in salt intake. The result was a general rise in blood pressure. Those who had the greatest difficulty in getting rid of the excess salt had the greatest rise. Systemic hypertension is the major cause of stroke, of aortic dissection, a major contributor to aortic aneurysm, and an accelerator of atherosclerosis.

Humans are genetically programmed to eat about 1 gram (g) of salt daily, not the 10 we now average daily in the USA. The rise in salt intake was due to several factors but mainly to salt's ability to preserve food.



The rise was then reinforced by an acquired addiction for salt which is now maintained, in large part (80%), by the consumption of processed foods. Until 8 to 4 million years ago, fruit was the main food of mammals. Later, when the human and ape lines diverged, the human line, our ancestors, began to eat a modest amount of meat until 1.8 to 1.6 million years ago when *Homo erectus* began to consume more meat as evidenced by the large accumulations of animal remains in archaeological sites. These hunter-gatherers lived in areas where there were large numbers of grazing animals. Their diets eventually consisted of 50% meat and 50% plants. If the wild animals our ancestors hunted had the same contents of minerals as in present-day cows and sheep, the intake of salt in paleolithic times was <1 g/day. Because the hunter-gatherers also consumed large quantities of vegetables and fruits, the potassium content of their diet was approximately 16 times greater than their salt intake. Potassium, in contrast to salt, lowers blood pressure. Our potassium intake now is considerably less and lower than that of salt.

The consumption of salt began to rise when the combined effects of overhunting, climate changes, and population growth led to the introduction of agriculture. During the first few 1,000 years after the advent of agriculture, the intake of meat declined and the proportion of vegetable food in the diet increased by up to 90%. These early farmers probably consumed about the same amount of salt as did their hunter-gathering ancestors (<1 g/day). Plants contain very little salt, so herbivores, in contrast to carnivores, may suffer from salt deficiency and will travel miles to salt licks. But, the amount of salt consumed by herbivores from salt licks is modest and there is no evidence that their total salt intake exceeded that of carnivores, the salt needs of which are satisfied by the salt contained in meat and blood. (Carnivores do not search for salt but they visit salt licks in hopes of making a meal of salt-seeking herbivores.)

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The increased intake of salt by humans initially was the discovery that meat and other foods could be preserved by placing them into concentrated salt solutions. Nomads hunted and ate the meat within a few hours of the kill. When the wide herds migrated, the nomads moved with them. In contrast, the farmers were tied to their fields, making it difficult for them to acquire fresh meat. The need arose to preserve food during the winter, an essential element for survival. Preservation was achieved by soaking meat in brine. Salt permeates food and makes bacterial life impossible. Salt preservation was used in Egypt by at least 2,000 BC. Additionally, highly salted food suppresses the salt taste buds in the mouth so that natural foods become insipid and unappetizing. The result was that salt would then be added to unsalted food to bring it up to the same concentration as that of the preserved food.

The addiction for salt also must have been exacerbated by its increasing availability. The change from a nomadic to an agricultural way of life gave rise to settled communities, between which trade began to flourish. Salt became a precious article of commerce. About 1,000 years ago, salt intake in the Western world had risen to about 5 g/day. It continued to rise until the 19th century when, in Europe, it was about 18 g/day. In the 16th century in Sweden, daily salt intake rose to 100 g/day due to the high consumption of salted fish. The worldwide reduction to an average of 10 g/day during the 20th century is probably the result of the introduction of refrigeration.

Throughout the world there are still many isolated populations which continue to eat a low salt diet, 0.05 to 2 g/day. They are more fit and have little to no cardiovascular disease compared with populations consuming an average of 10 g of salt per day. Numidian nomads and certain Bedouins, who feed on fish and roasted meat, never eat salt with food. Eskimos who have not been in contact with Western civilization have a strong dislike for salt and avoid foods in which saltiness is detected.

Addiction to salt has been introduced subsequently to many of these isolated tribes and also, inadvertently, to a colony of chimpanzees, the species phylogenetically closest to humans. In the wild, chimpanzees eat a vegetable and fruit diet (low salt, high potassium). For many years a colony of chimpanzees in San Antonio, Texas, in addition to fruit and vegetables, ate 1 to 2 kg of biscuits per day and they provided 6 to 12 g of salt per day, the same high intake as in humans. (These biscuits also provided a potassium intake of 6 to 11 g/day.) The high content of salt given the chimpanzees was based on the prevailing assumption that because the average intake of salt in humans is around 10 g/day, that this should be normal for chimpanzees, our nearest relatives who are near our weight. The chimpanzees then were given biscuits identical to the original ones except that they contained a low salt content (0.5 g/day). They refused the low salt biscuits and rapidly lost weight. Thus, these chimpanzees had become so addicted to the taste of high salt biscuits that they found the taste of low salt biscuits so repellent that they preferred to do without!

In 1853, Lehmann, a physiologist, asserted that humans had no need to add salt to natural food. He had noted that most animals in freedom or captivity did well on natural food without the addition of salt. He admitted that some herbivores (e.g., cow and deer) ate salt eagerly when they were offered it or when they came across it in salt licks, but he believed that there was no proof that they needed it. At the time, Lehmann was a voice crying in the wilderness, but today most veterinarians have similar views.

Addiction to a high salt intake in modern humans is induced early in life. Newborn infants are either indifferent to or avoid moderate to high concentrations of salt, but by 2 or 3 years of age children commonly prefer salty foods over the same foods without salt. It appears that the ability to taste salt develops in infants after 4 months of life. It is uncertain whether this preference for salt develops because of maturation in the infant's ability to detect salt, which they then find pleasurable, or whether, as is more probable, the normal high salt content of food given to children conditions them to its taste. Some investigators have reported that a preference for a salty food in infants can be induced by only 1 exposure to the salted food. It is hardly surprising, therefore, that as these children get older, their addiction for salty foods, fanned by commerce, worsens. Urinary salt excretion (virtually all salt consumed is excreted in the urine) was reported in 4- to 6-year-old children in 1986 in the UK. The mean 24-hour salt excretion was 4 g; their mean body weight was 21 kg. Although difficult to compare salt excretion in young children with that of adults, when compared for an equivalent muscle mass, salt excretion was 3.5 times greater in the children than the average excretion in adults, an indication of an enormous salt intake in the children.

SALT AS A REVERED SUBSTANCE

The usefulness of salt for purifying and preserving food raised it to a revered substance. In some cultures, salt was used to ward off the evil eye, and this belief stemmed from the belief that the devil was actually afraid of salt. Throughout history, salt has been seen as an emblem of hospitality. The Romans considered salt to be a sacred article of food, and it was a matter of religious principle with them to see that no other dish was placed upon the table before the salt was in position. For centuries, the wealthy placed silver saltcellars in the middle of the table, and they served as a symbol of friendship and hospitality. The saltcellar was a sign that the guest had been invited in love. That it was the last article to be removed was to remind guests that while meals may come to an end, friendship is perpetual. The emblematic saltcellar increasingly became a decorative art of beauty. The rank of guests at a banquet in England was indicated by their place at the table with reference to the saltcellar. Salt also constituted the essence of things, particularly of

life itself. Christ told his disciples, "Ye are the salt of the earth," i.e., the best of the human race. And again, referring to them he asked, "If the salt has lost its savour wherewith shall it be salted?"

Salt rapidly moved from the magical to the medicinal. Salt was used extensively throughout history in the belief that it prevented and cured certain diseases. It was thought that the corruption of a corpse in the grave was due in part to worms and that salt delayed this corruption. Salt, therefore, was used to treat the living suffering from worms.

Salt also was considered a symbol of procreation and reproduction. The sea, with its wealth of fish, was believed, because of its saltiness, to be a fructifying creative element. Feeding a dog salt was believed to increase the number of puppies. Ships carrying salt were believed to cause the mice in the holes to multiply far more than on ships carrying non-salt cargoes. Eating salt was believed to cause mice to become impregnated. Salt also was connected with barrenness, presumably because of the empty isolation of salty deserts and other places where an excess of salt prevents all growth, and it was used by women to prevent barrenness. The dread of impotence on the marriage night (an embarrassment known at one time as the "ligature") could be allayed if one or both partners carried some salt in their pockets or on their clothes or the wife had salt in her shoe. Salt was considered to have an exciting influence on the nervous system, arousing passion and desire.

The veneration of salt by humans is perhaps best illustrated by the association of salt with most forms of religion. The earlier gods were worshiped as the givers of the fruits of the earth, and this included "bread and salt." Salt was an essential constituent of sacrificial offerings in ancient Egypt, Greece, Rome, and Judaism. In the Roman Catholic Church, salt was introduced as a purifying substance for baptisms in about the 4th century, and it has played a prominent part in certain rituals since.

SALT'S POLITICAL AND ECONOMIC IMPACT

The presence of salt often determined the site of settlements and their prosperity. Where salt was plentiful societies tended to be free, independent, and democratic, but where it was scarce ". . . he who controlled the salt controlled the people." The ample supply of salt along the shores of the Mediterranean and the North Sea was associated with free societies. In Rome, salt was often given to the people by the government. In contrast to the liberal societies associated with the relatively ample supply of salt, in the ancient river valley civilizations of the Nile, in Babylon, Mexico, Peru, and some part of China, the scarcity of salt led to its being monopolized by the rulers and priests. They kept the salt in heavily guarded stores and were thus able to manipulate their unfortunate salt-addicted populations, who were entirely dependent on them to satisfy their craving and their absolute need for salt to preserve meat and fish. The need for salt for preservation and the desire for its taste has always been so great that for nearly 4,000 years governments found it profitable to control its availability. The immense wealth and prosperity of several empires, including the Chinese and Venetian, were entirely based on salt. The industrial importance of salt remains embedded in the names of certain towns that were big producers of salt: Salins in France, Salzburg in Austria, Salzkotten in Prussia, Saltdean in England, Saltcoats in Scotland, and Saltville in Virginia, and towns the names of which begin with *Hal* (the Greek for salt), Halle, Hallstadt, and Hallein.

There are 2 infamous centers of industrial salt production, mentioned in the Old Testament, Sodom and Gomorrah, which lay at the southern end of the Dead Sea. The citizens of Sodom and Gomorrah indulged in certain sexual habits which were disapproved of in Heaven. God therefore decided to destroy them. One man named Lot, however, together with his wife and 2 daughters, were led to safety by an angel. They were instructed not to look back at the town they were leaving. Unfortunately, Lot's wife could not resist 1 backward glance, and she was immediately turned into a pillar of salt. This biblical incident has been depicted in many medieval stained-glass windows, engravings, and paintings.

Salt was often a cause of conflict and at other times influenced the course of a war. In earlier times there were vicious local wars for the possession of salt springs and surface deposits of salt, particularly in Central Europe. The dominance of England in salt export during the 19th century not only had a profound effect on India but also on the American Civil War. In 1861, there was no refrigeration and canning had not been invented. Salt was needed for the preservation of meat, poultry, and fish, and for the preparation of leather. The hides used to make the harnesses for the thousands of mules pulling supply wagons were kept in brine until they were tanned. The South needed about 300 million pounds of salt per year for a population of 9 million, half of whom were in the Army. By 1860, people in the USA were consuming more salt than any other country, an average of 50 pounds (23 kilograms) per person per year. All meat and fish were either smoked or packed in brine. The monthly allowance for a Confederate soldier in 1864 was 1.5 pounds (680 g) of salt, which, if it were all consumed, would give a minimum of 23 g of salt per day, 4 times the current recommendation of 6 g/day. The South's sources of salt were well below these needs. Its principal salt-producing areas were in Wilmington, North Carolina, which was lost early in the war, and in Saltville, West Virginia. Most of the South's salt requirements before the war had come by sea from England. When the war started, the North blockaded the southern ports and the South's supply of salt suddenly stopped. The lack of salt severely interrupted the preservation of meat, and its absence in food lowered the morale of the population and that of

the soldiers. Some individuals cornered the available salt, which raised its price. The border dividing the Union from the Confederacy stretched for hundreds of miles and made trade between the 2 relatively easy.

Blockade running was common along the coast. A profitable run for the North was to transport contraband goods, including salt, to Cuba where they were picked up by Southern blockade-running ships. A host of illicit traders prospered on both sides. Rhett Butler in *Gone with the Wind* is a prototype of the affluent, glamorous salt-blockade runner. A successful run was an event of major local importance and was reported in the local journals. General Sherman, who purposely made war against civilians because they supplied the armies he was fighting, was the first to urge the federal authorities to decree that salt be contraband. He asserted that salt was as much a contraband of war as gunpowder. The South, to overcome its grave shortage, used desperate measures. They attempted to obtain salt by boiling seawater at various sites along the West Coast of Florida where there were forests near the beaches to supply the necessary fuel. However, these sites were regularly destroyed by the North's Navy, and, as usual, they were back in production within a few days, but the price paid by the South to keep up the production of salt was crippling. The North considered that the destruction of salt stores and the harassment of its production were equal to the winning of battles and were worth the cost of the military operations involved. It has been claimed that the lack of salt, by diverting much of the South's men and money from the first objective of war—to defeat the enemies' Army—was an important contribution to the South's defeat.

SALT AND BLOOD PRESSURE

The earliest comment relating dietary salt to blood pressure was by the Chinese in 1700 BC: ". . . if large amounts of salt are taken, the pulse will stiffen or harden." It was not until nearly 3,500 years later, when Richard Bright of Guy's Hospital in London suggested in 1836 that the blood pressure of patients with severe renal disease might be elevated. Later, another physician at Guy's Hospital noted that high blood pressure also could occur in individuals whose kidneys looked normal. Today, the latter are said to have *essential* or *primary* hypertension, i.e., of unknown cause, by far the commonest form of high blood pressure, accounting for 95% of all cases in humans and affecting 10% to 15% of the world's population of 6.2 billion people. Essential hypertension is characterized by a gradual increase in blood pressure with age so that by age 60, 50% of the population in the Western World have levels >140/90 mm Hg. In the remaining 5% of the hypertensive population, the rise in blood pressure is due to some form of renal or endocrine disease and the high pressure is then known as *secondary* hypertension. In secondary hypertension the importance of dietary salt in causing blood pressure to rise is well established. The importance of dietary salt in essential hypertension has been more difficult to discern.

The connection between salt and high blood pressure was first demonstrated in France in 1904 by Ambard and Beaujard. These investigators studied for 3 weeks 6 patients with high blood pressure from essential hypertension. They varied the intake of salt by means of 3 diets: one contained little salt but 2 liters of milk per day; the second also contained little salt but, in addition to milk, contained much protein, meat, and many eggs; the third diet consisted of the same amounts of milk as the first plus 2 liters of salty broth containing 10.5 g of salt. Salt balance was measured by estimating the amount of salt in the urine each day. They found that when the diet contained little salt, more salt was excreted in the urine than was eaten so that the patient was in negative sodium balance. The blood pressure fell even though the intake of protein was considerable. (Protein excess at the time was considered the cause of systemic hypertension.) When the diet was high in salt, less salt was excreted in the urine so that the patient was in positive sodium balance, i.e., retaining salt, and the blood pressure rose even when the protein intake was low. Ambard and Beaujard concluded that they had demonstrated a close relation between salt balance and blood pressure.

During the following 20 years, salt deprivation was used to lower blood pressure, mainly in patients with renal disease, but with poor results. Allen and Scherril, in 1922, described the effect of a low salt diet in 180 patients with severe essential hypertension. They were all given a normal protein intake. The blood pressure returned to normal in 19%. In 42%, the fall in blood pressure and the relief of symptoms were sufficient to be regarded as therapeutically successful. Complete failure occurred in 30%. These authors concluded that essential hypertension was a "salt nephritis." Houghton, also in 1922, discussed all the effects of salt reduction in several forms of hypertension and proposed that a rise in blood pressure is "a tertiary condition of which the immediate cause is a larger salt intake than the damaged kidneys can excrete." This is a modern view.

Despite the work of Ambard and Beaujard, Allen and Scherril, Houghton, and a few others, the connection between salt intake and hypertension continued to be denied. The position was finally clarified by Kempner in 1948. He treated hypertensive patients with a diet containing <0.5 g of salt, 20 g of protein, and little fat. It consisted of rice and fruit. Kempner was most interested in the relatively low protein content of his diet and he was reluctant to admit that it might be the low salt of the diet that lowered blood pressure. He attributed such an assertion to others who used his diet. It is ironic that Kempner is now remembered as the person who demonstrated beyond any shadow of doubt that high blood pressure can often be lowered by a low salt diet. Kempner's diet was so low in salt that the 24-hour urinary excretion of salt at the end of 2 months usually fell to <0.25 g. Kempner published

the effect of his diet on 500 patients with essential hypertension. The article illustrated by blood pressure charts showing relentless falls in blood pressure, chest radiographs showing pronounced reductions in heart sizes, electrocardiograms showing gross abnormalities reverting to normal, and photographs of damaged retinae that dramatically improved. There is no doubt that Kempner's rice diet achieved remarkable and sustained results. He made no mention, however, of how difficult it was to get patients to follow his rice diet or of the complications associated with such severe and rapidly induced reductions in salt intake. One reason he was so successful using his diet, when others failed, was that he collected all the urine excreted each day from each patient so that by the time he saw them in the ward he knew how much salt they had excreted and therefore how much salt they had eaten. Kempner's reaction when they had erred was such that the patients were unlikely to err again. His use of salt restriction at the time was the only therapeutic maneuver available to lower blood pressure. When oral diuretics were introduced in the mid-1950s, the increased urinary excretion of salt was considered a satisfactory alternative to a low salt diet and a much more convenient way of dealing with the habitual high consumption of salt.

Because diuretics have adverse consequences, moderate dietary salt restriction (to 3 to 6 g/day) to control blood pressure in essential hypertension has been advocated. Numerous trials have shown that such reductions decrease blood pressure greatest in the elderly and in those with the highest pressures.

A link between salt and blood pressure also can be demonstrated by measuring the small changes in blood pressure that are rapidly induced by an abrupt change in salt intake or salt output, for example, an intravenous infusion of saline or the administration of a diuretic. Individuals in whom these interventions cause the least change in blood pressure are termed *salt resistant,* and individuals in whom larger changes are produced are referred to as *salt sensitive* and are considered by some to be more likely to develop hypertension later in life. There is little evidence, however, that the immediate response of blood pressure to such sudden drastic changes in salt status indicates how the blood pressure of an individual responds to a lifetime's exposure to a high salt diet.

The effect of a reduction in dietary salt intake on systemic arterial pressure has been measured in normal newborn babies, school children, and adults. Nonhuman animal studies also have shown that young animals are much more sensitive to the level of dietary salt intake than adults, and that even a transient rise in intake early in life may increase their response to a high intake later in life. In a large study of 750 children, a reduction in salt intake from 9 to 7 g/day induced a significant fall in blood pressure after 6 months. In a similar study involving 32 adults (average age 40 years), reduction in salt from 9 to 4 g/day caused a significant fall in blood pressure at 12 weeks and the fall in pressure was correlated with the fall in salt excretion. In normal circumstances, salt excretion is almost the same as salt intake. In a study of adults 60 to 78 years old, a reduction in salt intake from 10 to 5 g/day for 4 weeks reduced systolic pressure by 8 mm Hg and diastolic pressure by 4 mm Hg, similar to reductions achieved by blood pressure–lowering drugs. The extent of the fall in blood pressure was the same whether the subject started with a high or a normal blood pressure.

Severe increases in salt intake for a few days have little effect on normal blood pressure. In young adults, 28 g of salt per day was required to cause a rise in blood pressure. In subjects aged $\geq$50 years, however, only 20 g/day for a few days was necessary to cause a rise in blood pressure. It appears, therefore, that the effect of a sudden rise in dietary salt intake on normal blood pressure during a person's life varies and is most pronounced in the very young and after age 50.

A well-documented connection between systemic hypertension and dietary salt intake has been demonstrated in normal dogs, rabbits, rats, baboons, and chimpanzees. The work on rats and chimpanzees is the most relevant to essential hypertension. A study performed in 1951 found that the substitution of a 1% salt solution for drinking water produced hypertension in the chicken, rat, and rabbit. Whatever experimental method was used to induce secondary hypertension (e.g., by partially obstructing a renal artery), it was facilitated by increasing the salt intake and prevented by salt restriction.

Experiments in chimpanzees strongly reinforced the proposal that essential hypertension is due to the prevailing high intake of salt. Chimpanzees normally consume a diet low in salt, but when their salt intake increases to that of present-day humans they, like humans, develop hypertension. Again like humans, a number of chimpanzees do not develop high blood pressure on the high salt diet. The rise in pressure was gradual and it was still rising 18 months after they started eating the high salt diet. Upon returning to a diet that contained $<$0.5 g of salt per day, blood pressure fell to its original level.

There have been nearly 40 accounts of certain primitive populations in which blood pressure did not rise with age—in other words, they did not have essential hypertension. Their dietary intake of salt was $<$3 g/day, in a few it was $<$1 g/day, and in 1 it was about 0.5 g/day. Studies of populations with a high prevalence of hypertension (some Japanese and Portuguese communities) have shown high intakes of salt. In 1 study, salt intake averaged 26 g/day. In between are the bulk of westernized societies that consume 7 to 12 g/day (average 10). The connection between salt intake and hypertension in these intermediate populations is evident but more difficult to discern mainly because of the narrow range of salt intake.

The Yanomamo Indians are probably the most primitive native tribe in the world. They live in about 100,000 square miles along the border between Venezuela and Brazil. There are approximately 18,000 individuals scattered through the Amazon rain forest

in about 200 villages with 40 to 200 persons in each. They have been described as seminomadic "slash and burn" agriculturists living on a diet of locally produced crop and game supported by wild fruits and insects. Their staple foods are cooked bananas and manioc. In most villages there is little access to salt, refined sugar, alcohol, or dairy products. In one group of 206 persons aged 20 to 50 years, which comprised all the adults from 3 villages, the mean 24-hour urinary excretion of salt was 0.5 g/day, with a potassium excretion of 3 g/day. The mean weight of the men (50 kg) was about the same as that of chimpanzees. Their blood pressure was much lower than that found in western populations and there was no rise in blood pressure with increasing age. Their blood pressure, just like that of the Western world, is approximately 90/60 mm Hg at birth and that is their blood pressure their entire lives. The Yanomamos probably represent the ultimate human example of the importance of salt on blood pressure.

Although numerous studies have confirmed that there is a significant relation between salt intake (measured as 24-hour salt excretion) and blood pressure, 1 obstacle has bothered a great many researchers and continues to do so. It is that within a single community there is no relation between blood pressure and the salt intake of its individuals. There are many reasons for this apparent lack of correlation between blood pressure and the intake of salt within individuals of a single community. Blood pressure varies from day to day in an individual. The day-to-day fluctuations in salt ingested and excreted by each person in any one 24-hour period varies enormously (3 to 18 g/day). Such variations depend on the type of food eaten the previous day and its salt content. To obtain an accurate estimate of an individual's average salt excretion, it is necessary to measure the salt excretion on >5 occasions, which is not practical when studying large numbers of people. Such large methodologic difficulties mask the detection of differences between subjects.

The relation of blood pressure to salt intake was studied extensively by Dahl. Over a number of years, he and his associates measured the blood pressure and the 24-hour urinary excretion of salt in Alaskan Eskimos, Marshall Islanders in the Pacific, and employees at the Brookhaven Laboratory in the USA where Dahl worked. Correlation between the average daily salt intake and the prevalence of hypertension in these different centers was excellent. The Eskimos, whose salt intake was about 4 g/day, had no hypertension. The Japanese had the highest salt intake and the highest prevalence of hypertension, and the other 3 were in between. Although the relation between salt intake and blood pressure was not evident within the individuals in most populations, salt intake controlled blood pressure. He studied Brookhaven's employee use of salt at the table. He classified the subjects into 3 groups: (1) those who did not add and had never added salt to food; (2) those who added salt to food only if, after first tasting it, found it insufficiently salty for their palate; and (3) those who added salt to food without first tasting it. Although the mean blood pressures of the 3 groups were similar, fewer persons had high blood pressure among the group with low salt intake (group 1).

SALT AND HYPERTENSION IN AFRICAN-AMERICANS

African-Americans have the highest prevalence of high blood pressure in the world. The prevalence of high blood pressure is nearly twice as high among African-Americans than in Caucasian Americans (38% vs 20%) and 2 to 4 times higher than in West Africans. The degree of hypertension appears to collate with the darkness of skin color. The blood pressure of African-Americans also is more sensitive to increases in salt intake than that of American whites, and they retain an intravenous load of salt far longer than whites. Conversely, it is easier to lower the blood pressure of African-Americans with a diuretic. Thus, African-Americans have an enhanced ability to retain salt or they have a diminished ability to get rid of a high salt intake.

A hypothesis to explain the high prevalence of hypertension in African-Americans proposes that the process of enslavement decimated those who were least able to conserve salt so that the survivors were individuals who were best able to conserve salt. For 300 years, between 1500 and 1800, over 12,000,000 black people were transported against their will from the West Coast of Africa to the Western Hemisphere. Most went to South America, but 6% of the total, estimated to be about 430,000, ended up in North America. Many came from vary low salt areas in West Africa, such as the sub-Sahara Savanna. Those who already had the best ability to conserve salt would have had a better chance of surviving the voyage, but this would also have made them and their descendants more likely to develop a higher blood pressure subsequently when exposed to a high salt intake.

The slaves were conquered by African neighbors to the north and east. After capture they were force-marched 100 to 200 miles to the coast by African slave handlers. There they were confined to crowded huts known as barracoons to wait several weeks or months for the ships to take them away. The death rate from the point of capture to the coast was about 10%, and another 10% died in the barracoons. Conditions on board ship were terrible and, on average, 15% died during the 2-month crossing. Another 5% died while waiting in the USA to be sold, and a further 10% died in the first 2 years when they were being "seasoned" to their new environment. Thus, on average, only 60% of those captured survived >2 years. The causes of death were most often due to an illness associated with loss of salt and water. There was the heat and excessive sweating during the forced marches to the coast and the incarceration in the unventilated barracoons and

ships' holds. During the sea voyage, vomiting due to seasickness was common. Diarrhea was always rife and it was the predominant cause of death. This hypothesis suggests that the possession of kidneys with ability to hold on to salt would have increased the chances of surviving. It is this selective survival among the descendants of the surviving slaves of genes responsible for an increased ability to hold on to salt that is now responsible for the exceptionally high prevalence of hypertension in African-Americans.

MECHANISM FOR SALT'S EFFECT ON BLOOD PRESSURE

The lack of an obvious mechanism whereby salt intake controls blood pressure has been 1 factor that has delayed acceptance of the relation between the 2. In essential hypertension in humans, in secondary hypertension in humans associated with overt renal disease, and in hereditary hypertension in rats, the kidney has difficulty excreting salt, and this sets in motion a train of events that causes the blood pressure to rise. The observations upon which this evidence is based can be divided into those which show that the rise in blood pressure is due to an abnormal kidney and those which demonstrate that the kidney has a diminished ability to excrete salt. When blood pressure rises, it causes widespread changes, particularly in the kidney, which it sometimes even destroys. Therefore, the evidence that is pertinent to the search for the initial cause of the rise in pressure has to be distinguished from the changes produced by high blood pressure itself. This distinction is most easily made by studying the human or nonhuman animals who are going to develop hypertension but before the actual development of hypertension.

Confirmation that the initiating trigger that causes blood pressure to rise in hypertensive strains of rats and in essential hypertension is in the kidney has been obtained by kidney cross transplant experiments. The animal experiments consist of cross-transplanting 1 kidney from 1 animal (the donor) to another (the recipient) in which the kidneys have been removed. The kidney may come from either a prehypertensive strain rat or from a normotensive control animal. When a kidney from a prehypertensive hypertensive strain rat is transplanted into a control normotensive rat, the blood pressure rises. When a kidney from a control rat is transplanted into a prehypertensive hypertensive strain rat, the blood pressure does not rise. If a kidney from a normotensive rat is placed into a hypertensive strain when it has already developed high blood pressure, the blood pressure comes down. These experiments demonstrate that blood pressure follows the kidney.

Similar results have been obtained in humans with essential hypertension. In 6 black patients with terminal renal failure due to prolonged essential hypertension, the blood pressure fell to normal and remained normal for many years after receiving a kidney from a young normotensive donor. In another investigation, the blood pressure of the parents of the donors of kidneys and the recipients were measured. The patients who received a kidney from a donor from a family with high blood pressure needed significantly more blood pressure–lowering therapy than those who received a kidney from a family with normal blood pressure.

There is some evidence that normotensive children of parents with essential hypertension have difficulty excreting salt. Compared with control subjects, intravenous administration of a salt solution at a certain rate to normotensive first-degree relatives of patients with essential hypertension leads to a rise in blood pressure and reduced salt excretion. An increase in salt intake to 16 g/day for 7 days causes an increase in blood pressure in normotensive offspring of hypertensive patients, but does not raise the blood pressure of normotensive offspring of normotensive parents. These observations suggest that although the kidney in essential hypertension looks normal, it has an inherited impaired ability to excrete salt. It is now evident that the difficulty in excreting sodium by normal-looking kidneys of young patients with essential hypertension and of prehypertensive hypertensive strain rats is due to the presence of many intrinsic renal functional abnormalities. There are disturbances of kidney blood flow and of several locally produced kidney hormones and other substances that control salt excretion. Thus, the rise in pressure in essential hypertension depends on the magnitude of the excess salt intake, the type of severity and combination of intrinsic renal abnormalities that impair the kidney's ability to excrete salt, and the number of years the individual has had this conflict. The exact mechanism whereby the kidney's difficulty to excrete salt raises the blood pressure is not known.

COMMERCIAL REASONS FOR HIGH SALT FOODS

The above evidence indicates a strong connection between salt intake and blood pressure. Why then is so much salt continually added to foods? The first is taste. Tomato juice without salt is virtually intolerable for example. The food industry is more than happy to agree in public that taste is the major reason why they add salt to food. The other 2 reasons, however, are entirely commercial and for most foods are the real reason the food industry wants the intake of salt to remain high. The salt content of food is an important determinant of the amount of water the food contains, and it increases the weight of food at very little cost. If the salt content of sausages is increased from 0.5% to 2.5%, which is the usual concentration of salt in sausages, the water content is increased by approximately 20%. Far less salt could be added if other flavors were substituted, but because this would reduce the weight of the sausage, consumers would expect the price to fall. They would resist paying the same amount for a lighter sausage.

The second commercial reason is that salt increases thirst. In most temperate climates the body needs

about a liter of fluid a day. If the consumption of salt is increased, the salt concentration of the body will tend to rise, which stimulates thirst and therefore the amount one drinks. There is a direct relation between salt intake and fluid intake. It is not surprising that in pubs there are often free supplies of salted peanuts and potato chips, and that many of the soft drink manufacturers, some of whom also make alcoholic drinks, own companies that specialize in the manufacture of highly salted snacks. If salt intake were to be reduced, those companies would lose hundreds of millions of dollars in sales of soft drinks!

The salt extractors and the salt manufacturers in the USA finance a public relations body known as the Salt Institute, which provides a one-sided story supporting the high salt content of processed food. The Institute propagates the view that there is a considerable debate within the medical and scientific community as to whether any relation exists between hypertension and sodium intake in the general population and that a decrease in sodium intake may result in a rise or a decrease in blood pressure for some and an increase in blood pressure in others and no significant fall in blood pressure for most. This view is very reminiscent of that taken by the tobacco industry for many decades regarding the danger or the lack thereof of cigarettes. The Salt Institute, which seemed to know about the Intersalt study (the worldwide investigation on the relation of salt excretion to blood pressure) in advance of its publication, turned the study on its head and interpreted it in a way exactly opposite to that of the authors, that salt intake had no relation to blood pressure. The Institute's attempts to discredit the Intersalt study have continued unrelentingly.

The Salt Institute, a large snack company, and the Dairy Council in the USA have been very much involved in putting forward another concept, that what really raises blood pressure is not a high salt intake but a low calcium intake and that eating more calcium (e.g., milk has a high content of calcium) would solve the blood pressure problem. Giving calcium to patients with high blood pressure, however, has not lowered it and indeed there is little or no relation between calcium intake and blood pressure in different populations. The Salt Institute then argued that a very high calcium intake lowered blood pressure in individuals who were already on a high salt intake. This has not proven to be the case. A high salt intake is an important aggravating factor in bone demineralization, and reducing salt intake is likely to have a greater beneficial effect on bone density than increasing calcium intake. The food industry's next rather rash maneuver was to assert that a moderate reduction in salt intake may be dangerous. Close analyses of the study cited in support failed to back up this claim.

DECREASING SALT INTAKE

Since there is little doubt that increased salt intake increases blood pressure, what steps can we take to decrease the intake of salt? There are at least 3:

1. *Do not add salt at the table.* It is not impolite not to pass the salt shaker! Sauces that are also added to food or added at the table, such as tomato ketchup, are also usually high in salt. Adding salt at the table is essentially a habit. This was clearly demonstrated in a study in an Australian canteen where the hole in the saltcellar was reduced. As a result, the habitual unthinking number of shakes delivered only half the quantity of salt. No one noticed any difference.
2. *Stop adding salt when cooking.* This is more difficult because it requires the agreement of the person who does the cooking in the household. At first the food will taste bland. Two to 4 weeks later, however, as the sensitivity of the salt taste receptors in the mouth become more sensitive to the taste of salt in the usual concentrations, it is more pleasant. (It is the same as giving up sugar in tea or coffee—initially it is difficult, but later the taste of sugar in tea or coffee is unpleasant.) Salt is often added inadvertently; all stock cubes, gravy browning, soy sauce, and ready-prepared mustard all contain large amounts of salt and should be avoided.
3. *Avoid manufactured foods or processed foods that have had salt added.* This is by far the most difficult step because many processed foods are not labeled with their salt content and if they are labeled, the labels tend to be confusing. Instead of being labeled as the amount of salt or equivalent of salt, they are labeled as sodium and in most countries as grams of sodium per 100 g of food. Buying as much fresh food as possible or foods that are not processed with salt or if processed have <0.1 g of sodium per 100 g of food is useful, but few processed foods achieve such low concentrations.

The labeling system in the USA is cumbersome. The packet is labeled with the percent of the daily dietary intake that eating 1 portion of that food contributes to the recommended dietary intake (6 g of salt per day). An average packet of salted crisps contains 2 g of salt, which would account for 33% of the recommended daily salt intake. The idea behind this food-labeling system is that one can then add up all of these percentages and work out whether one's salt intake for the day is above or below the recommended intake.

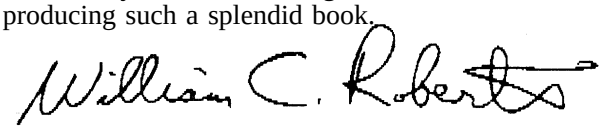
Foods with a low salt content are fresh and frozen vegetables. Vegetables in tin cans generally have salt added. Fresh meat is low in salt. All uncooked pasta, rice, olive oil, rapeseed oil, unsalted nuts, fruit juices, tea, coffee, and most alcoholic drinks are low in salt. In contrast, foods that have a high salt content include: meat products (e.g., bacon, ham, cured meat, canned meat, sausages, paté), smoked fish and fish in tin cans, instant noodles and soups, tinned or packet soups, stock cubes, gravy brownings, yeast extracts, meat extracts, vegetable juices, soy sauce, and salted fish.

Most fast foods contain large amounts of salt as well as saturated fat. A hamburger with french fries generally contain approximately 5 g of salt.

CONCLUSION

Salt and blood pressure go together. The more salt we take in, the higher our blood pressure. Systemic hypertension is the major cause of stroke, of aortic dissection, a major contributor to aortic aneurysm, and an accelerator of atherosclerosis. We all must decrease our salt intake! As Freis stated in 1976: ". . . the evidence is very good . . . that reduction of salt in the diet to below 2 g/day would result in the prevention of essential hypertension and its disappearance as a major public health problem."[2] Sacks and colleagues[3] 25 years later appear to support this earlier recommendation.

Thank you Drs. MacGregor and de Wardener for producing such a splendid book.



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