RIC

ARTÍCULO DE REVISIÓN

Salt: its goodness and perversities

Michelle Lozada,* Claudia P. Sánchez-Castillo,*
Georgina del A. Cabrera,* Irma I. Mata,* Edgar Pichardo-Ontiveros,* W. Philip T. James**

* Departamento de Fisiología de la Nutrición. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán.
** International Obesity Task Force. London, United Kingdom.

ABSTRACT

Salt (sodium chloride) exists in nature in scattered form and has had social, economic and physiological influence in man's life. At present its consumption is a quotidian fact in the individual, since it exist a universal appetite of this element in the diet. Nevertheless, since relatively a short time ago it was begun to be recognized that salt is an important component for public health since an excessive intake of this element has been related to the development of hypertension. This disease affects a billion people and it is an important cause of morbidity in both industrialized and developing countries. This paper presents a review of the main historical, clinical and therapeutic aspects of salt as well as the quantitative intakes in Mexico and other societies.

Key words. Salt. Sodium. Hypertension. Salt intake.

La sal: sus bondades y nequicias

RESUMEN

La sal (cloruro de sodio) existe en la naturaleza en forma dispersa y ha tenido influencia social, económica y fisiológica en el ser humano. En la actualidad su consumo es un hecho cotidiano en el individuo, ya que existe un apetito universal por este elemento en la dieta. Sin embargo, hace relativamente poco tiempo se comenzó a reconocer que la sal es un componente importante para la salud pública, ya que su ingestión excesiva se ha relacionado con el desarrollo de hipertensión arterial. Esta enfermedad afecta a un billón de personas y es una causa importante de morbilidad tanto en países industrializados como en aquellos en vías de desarrollo. Esta comunicación presenta una revisión de los principales aspectos históricos, clínicos, terapeúticos así como consumos cuantitivos en México y otras sociedades.

Palabras clave. *Sal. Sodio. Hipertensión arterial. Ingestión de sal.*

INTRODUCTION

Throughout history, salt has been the subject of many stories, fables and is frequently mentioned in fairy tales.[1] Historically, salt has been very important and served as money at various times and places,[2] it was used as the currency when buying slaves and Roman soldiers were paid with salt, this was the origin of the word salary.[2,3] Salt also has great religious significance in many cultures.[4]

It is not surprising perhaps that given the fundamental physiological need for salt and its scarcity in the vegetables, fruit and other crops available during the evolution of homo sapiens in Africa that the man has retained the same innate biological drive for salt and the special salt taste buds which characteristically are found in all land based mammals.[5]

Uses of salt

Contrary to popular belief salt is not simply a seasoning for food but has more than 14,000 uses in the food, chemical, textile and agricultural industries and is used for example in the manufacturing of adhesives, batteries, explosives, metals, etc.[6] In the food industry, salt is used to increase and modify the flavour of food, to control microbial growth and alter the texture/consistency of food.[7] Thus traditionally bread manufacturers have emphasized the value of salt in determining the texture of the bread and only more recently have other methods been developed as substitutes for this property of salt.

Comparatively recently salt has been recognised as a major public health issue relating to the development of high blood pressure but this public health

profile is confused because salt was also identified as an ideal means for combating iodine deficiency when cretinism and goiter became to be recognised as major preventable problems in countries where, for geochemical reasons, the iodine within the rocks had been leached out by the millennia of heavy rain. The scarcity of salt meant that this sought after commodity could become a universal means of providing a whole population with extra iodine since most if not all the people would have access to only a few sources of salt. More recently the issue has been further complicated by the use of salt as a vehicle for fluoride and the prevention of dental caries.[8] The mixture of salt with fluoride is available in France, Switzerland and some countries of Latin America, where there is no policy on fluoridating the drinking water. In Mexico, there is an official standard that specifies the addition of fluoride to salt in those states where water for human consumption does not contain optimum concentrations of fluoride.[9]

Sodium and salt

Sometimes the two terms salt and sodium are used as synonyms even thought this can be very misleading if an attempt is made to specify the amounts being used and if it is not recognised that in some cultures e.g. China and Japan an appreciable part of the intake of sodium is derived from salts other than chloride e.g. monosodium glutamate (Table 1). Monosodium glutamate is the main ingredient of soy sauce. This is added to foods to enhance the appreciation of any flavours in the food. It probably works by activating N-methyl-D-aspartate (NMDA) receptors which are found in taste cells. NMDA receptors are integral receptor-ion channel complexes and when they open they allow an influx of Na+ and Ca2+ ions. This influx depolarises the taste receptor cell and acts as an excitatory influence. Then, far less of a particular taste will be required to cause the further depolarisation necessary to bring about transmitter release.[10]

Table 1. Sodium containing additives used in food processing.

Additive	Use
Sodium chloride	Flavouring, texture preservative
Sodium citrate	Flavouring, preservative
Sodium nitrate	Preservative, colour fixative
Sodium tripoliphosphate	Binder
Monosodium glutamate	Flavour enhancer
Sodium caseinate	Emulsifier
Sodium benzoate	Preservative

Source: Sánchez-Castillo CP, James WPT.[7]

Sodium is an essential element which is readily absorbed in the upper intestine in association with glucose and amino acid transport; it can also be actively transported against a substantial concentration gradient through the colonic mucosa into the blood stream where it acts as the principal cation in the extra cellular fluid.[11] Its principal systemic physiologic functions are fivefold: a) maintaining the extracellular fluid volume, which is in direct proportion to the body's total sodium content; b) determining the oncotic pressure of extracellular fluid; c) contributing to acid-base balance; d) maintaining the transmembrane potential difference and therefore the basis for the conduction of nervous impulses and control of muscle contraction as well as e) the active transport system through the cellular membrane.[12] The sodium concentration gradient across cellular membranes reflects the action of a substantial sodium pump in most cells and the induced ionic gradient is a major determinant and modifier of the membrane's facilitation of both nutrient entry and the counter-transport of cellular metabolites. Thus the cytosol of cells contains a concentration of potassium ions (K^+) as much as 20 times higher than that in the extracellular fluid. Conversely, the extracellular fluid contains a concentration of sodium ions (Na^+) as much as 10 times greater that within the cell. These concentration gradients are established by the active transport of both ions by the same transporter, Na^+/K^+ ATPase which uses the energy from the hydrolysis of ATP to actively transport 3 Na^+ ions out in exchange for 2 K^+ ions pumped into the cell. The crucial roles of the Na^+/K^+ ATPase are reflected in the fact that almost one-third of all the energy generated by the cell mitochondria is used to run this pump. The pump helps establish a net charge across the plasma membrane with the interior of the cell being negatively charged. This resting potential prepares nerve and muscle cells for the propagation of action potentials leading to nerve impulses and muscle contraction. The accumulation of sodium ions outside the cell also draws water out of the cell and thus enables it to maintain osmotic balance (without which the cell volume would be uncontrolled as water diffused inwards). The sodium gradient also provides the energy to run several types of indirect pumps: the symport action of Na^+ allows another molecule to be pumped through the membrane pump in the same direction. Thus the Na^+/glucose

transmembrane transporter protein allows sodium ions and glucose to enter the cell together with the sodium ions flowing down their concentration gradient as the glucose molecules are pumped up theirs. Then the sodium is pumped back out of the cell by the Na^+/K^+ ATPase.[13]

Given the crucial nature of sodium's actions it is therefore not surprising that there is not only a capacity for salt sensing but almost total intestinal absorption.[14] This is explained by the right side of the colon transporting electrolyte very efficiently by creating a large osmotic gradient, again generated by sodium transport between the lumen and the intercellular space of the mucosal cells. This transport also depends upon the energy-dependent Na^+/K^+-ATPase pump which is located on the basolateral membrane and pumps sodium from inside the cell against a large concentration gradient into the intercellular space. Luminal sodium in turn enters the apical membrane of the cell through sodium channels, flowing down the concentration gradient created by the pump. In contrast to the small intestine, where sodium in the intercellular space can diffuse back into the lumen and become iso-osmotic, hypertonic solutions are maintained in the intercellular space because the tight junctions in the colon are much less permeable to sodium diffusion.[14] The net result is the highly efficient absorption of sodium so that of the 150 mmol of sodium that enters the colon each day, less than 5 mmol is lost in the stool and usually about 1 mmol/day.[15] The tight junctions are, however, highly permeable to potassium, in contrast to sodium, allowing potassium to move from plasma to the lumen. Nutrient cotransporters are not found in the colon.

The body also has the ability to shut down the secretion of sodium through the sweat glands as well as a remarkable capacity to almost eliminate the renal excretion of sodium once the renin-aldosterone system has adapted to its maximum effect. The balance of the body's sodium content and distribution is kept remarkably constant despite an individual's wide daily intake and the major differences between sodium intakes by different populations.[12]

SODIUM REQUIREMENTS

There has been much discussion about how much sodium or salt children and adults need, but perhaps the more difficult issue is to gauge how much people can tolerate. Much of today's thinking has muddled the two issues because policy makers instinctively change the discussion to what now seems achievable politically and industrially when the evidence is that humans in old evolutionary times lived very successfully on remarkably little sodium. This contrasts with current intakes which are remarkably high simply because we not only have responded to our instinctive drive for salt but the discovery that salt in food was an excellent preservative further encouraged its use without anybody relating high sodium intakes to hypertension until the last century.[12]

The original approach to establishing the minimum requirements was based on simple analyses. Thus Dahl[16] calculated the needs of children by noting that a mother's daily breast milk supplied less than 5 mmol sodium despite the high concentrations of sodium in mother's plasma. This, as Spitzer[17] noted, meant that these amounts are sufficient for normal growth and development because humans had survived for millennia on the basis of prolonged exclusive breast feeding. This was also in agreement with McCance and Widdowson's[18] daily estimates of the sodium requirement for sodium deposited during growth in infants which amounted to 1.3 to 1.8 mmol Na (60 mg NaCl). The average requirements for children and adults of different ages are shown in Table 2. The data are based not only on experimental studies where adult volunteers were put on very low intakes of sodium but also on observations of what some societies or isolated tribes living in their customary habitats for millennia actually eat. Thus the Indian Yanomami of Brazil[19, 20] and Chimbus of New Guinea,[21] for example, have survived and thrived on less than 10 mmol/24h (585 mg NaCl).

Some of the confusion in interpreting these low values has arisen when doctors suggest that these low levels are not optimum because if children or adults develop diarrhoea, a major cause of death in poor developing countries where clean water and sanitation is non existent, then the losses of sodium in the faeces can become life threatening since the colonic sodium absorption system fails and there is an induction of a colonic chloride secretory process[22] by a variety of enterotoxins derived from pathogenic organisms. The interiorized toxin induces a marked increase in intracellular cAMP if it is cholera whereas an *Escherichia coli* enteroxin binds to a mucosal apical receptor which induces a marked rise in cGMP. Both these energy forms then drives chloride secretion. Other pathogens e.g. *salmonella* operate with different mechanisms but they all lead to a marked chloride secretion which then also is accompanied by sodium so that in severe diarrhoea the fecal sodium content can rise to that in plasma.[22] This

condition can therefore lead to large losses of body sodium. This life threatening state has been another evolutionary pressure to develop absorptive systems which virtually extract all the sodium in the ingested food and water. In modern societies children and adults with severe diarrhoea benefit from rapid fluid replacement with suitable electrolyte replacement but the evolutionary pressure was to extract the last mmol of sodium from anything ingested.

Part of the general medical confusion about salt needs also came from industrial and military experience. It was well known that coal miners sweated heavily when working very hard below ground in very hot conditions. They would then develop severe cramps if they did not take extra salt with their water or beer on returning to the surface. Furthermore troops from the UK and other European countries during colonial times knew that they needed to take extra salt to avoid cramps if they were marching through a hot desert. Analyses showed that they were losing several litres of sweat daily with a high sodium concentration. On the basis of these observations it became accepted in military, industrial and even sporting circles that adults engaged in intensive exercise needed much higher amounts of salt and that the tribal groups on very low intakes must have adapted markedly and not had to endure such intense work under extreme conditions. These concepts were exploded, however, when it became clear that the body needs a few days to adjust to much lower intakes or sweat losses of sodium. Thus the sweat losses of British athletes who were either engaged in quiet study in air conditioned offices in Jamaica or in climbing mountains laden with heavy equipment in the hot sun was assessed.[23] When sodium intakes dropped or sweat losses rose there was a rapid adjustment in the sodium concentration of sweat and urinary sodium excretion so that within 5 days they were able to minimise the body's loss and still sustain intense physical activity. Thus when the miners and troops took salt to avoid cramps they were not allowing the compensatory systems to come into play and did not recognise that an adjustment in temperature and dietary conditions before going out on acute intense exercises was a simple way of ensuring that there would be no need to take higher salt intakes.[23] On this basis the minimum requirements set out in Table 2 seem to be universally applicable and certainly apply to Mexico.[24]

SALT INTAKES IN MEXICO

Salt consumption in Mexico has been studied by different investigators (Table 3). Connor and colleagues[25] found that the Tarahumara Indians (living in the northern state of Chihuahua) in 1978 consumed daily 5-8 g salt (85-140 mmol Na). Ten years later the standardized world-wide INTERSALT[26] study, based on the gold standard approach of 24 hour urinary sodium measurements, found the Tarahumara Indians consuming daily between 3 and 10 grams salt (50 to 170 mmol Na). This community is relatively self-contained so the issue is what normal Mexicans consume. Sánchez-Castillo and colleagues[27] assessed in 1996, also with the gold standard approach, the urinary sodium excretion in adults living in a rural area

Table 2. The lower and upper estimated requirements of sodium for children and adults.

	Lower reference nutrient intake (LRNI*)			Reference nutrient intake (RNI**)		
Age	mmol Na/d	mg Na/d	NaCl g/d	mmol Na/d	mg Na/d	NaCl g/d
0-3 months	6	140	0.350	9.13	210	0.534
4-6 months	6	140	0.350	12.17	280	0.712
7-9 months	9	200	0.526	13.91	320	0.814
10-12 months	9	200	0.526	15.217	350	0.890
1-3 years	9	200	0.526	22	500	1.271
4-6 years	12	280	0.702	30	700	1.780
7-10 years	15	350	0.877	52	1200	3.051
11-14 years	20	460	1.170	70	1600	4.068
15-50 years	25	575	1.460	70	1600	4.068

1 mmol Na = 23 mg Na; 1g salt contains 17.1 mmol Na; no allowance for large losses from the skin through sweat.
*LRNI: Lower reference nutrient intake:2SD below EAR. EAR: Estimated average requirement which assumes a normal distribution of variability in interindividual requirements.
**RNI: Reference nutrient intake 2SD above EAR. Source: Appendix Dietary Reference.[24]

(Malinalco, State of Mexico) and in Mexico City (Tlalpan, D.F). A preliminary study was also made to calculate the number of 24 hour urine collections needed to characterised the sodium intake of healthy volunteers.[28] Estimated Malinalco salt intakes were 5 to 7 grams (85-120 mmol Na) whereas higher intakes of 7 to 8 grams (120 to 137 mmol Na) were found in Tlalpan. In Hermosillo, in the North, salt intakes were much higher: 15.2 ± 3.8 grams for men and 10.2 ± 3.3 grams for women.[29] Another study reported six years later in Tlalpan found lower intakes of 9.7 ± 2.5 grams of salt in men.[30]

These intakes need to be put into an international context. The full range of the salt intakes seen in the INTERSALT[26] study range from 0.2 to 242 mmol Na g/d. Law and colleagues[31] noted that the populations with low intakes of sodium (< 100 mmol/day or < 5.85 g of salt), are generally poor communities who have not gone through much economic development, while those with high intakes are generally economically advanced urban communities. Consumption in the UK, US, Canada, Belgium and other Western societies averages around 8-9 g/d.[26] Table 3 suggests that Mexican salt intakes differ between states as well as by sex and rural or urban area.[25-27,29,30] On average the women have only about 80% of the intake of men which is strikingly similar to the average difference in their energy requirements, a feature which has substantial implications once the sources of salt have been considered and shown to be generally dependent on the mass of salty food eaten rather than on what people add at table.

OPTIMUM INTAKES OF SODIUM

This has been a huge area of controversy not least because vested interests in the salt business relating to salt production and sales as well as some food companies have been anxious to recruit scientists to their cause and question the need to reduce salt intakes. Often doctors and scientists who are used to testing ideas in the most rigorous fashion cannot see that their critical comments, based on thinking in standard medical terms about discrete clinical cases, can unwittingly harm the need for a public health policy. They also tend to think that it is only a sub-group of salt -sensitive individuals with hypertension who need to consider reducing their salt intakes.

A WHO Expert Committe[32] and various other international groups[33-35] have recommended that adult daily salt consumption should be no greater than 5-6 grams (≈ 80-100 mmol Na) although we now know that a tenth of these salt intakes are what primitive societies lived on. These analyses are primarily based on the impact of sodium intakes on the development and management of hypertension although other issues such as the question of whether high salt intakes contribute to stomach cancer,[36-40] osteoporosis[41] and bronchial reactivity[7,42] have also been assessed. Turkann and Gorstein[43] have reported that behavioural stress and high dietary salt have increased blood pressure additively in non-human primates. It is clear that the evidence relating to blood pressure control is the dominant issue and that without this it is unlike-

Table 3. Salt intake in Mexico.

Year	State	Population	Age	n	Method	NaCl g/d	Na mmol/d
1978[25]	Chihuahua (northern)	Tarahumara indians	5-70	523	Dietary history	5-8	86-137
1988[26]	Chihuahua (northern)	Tarahumara indians	20-59	200	1/24h urine	3-10	51-171
1991[27]	State of Mexico (central)	Malinalco (rural)	20-64	Men = 24 Women = 54	3/ 24h urine[28]	6 5	86 120
1991[27]	Mexico City (central)	Tlalpan (urban)	20-64	77	3/ 24h urine[28]	7 8	120 137
1992[29]	Sonora (northern)	Hermosillo (urban)	21-38	Men = 12 Women = 10	24h recall + food analysis	15 10	261 174
1998[30]	Sonora (northern)	Hermosillo (urban)	30-45	Men = 38	Weighed intake	9.7	165

ly that policy makers would, on the basis of current evidence, advocate lower intakes even though they are far from the minimum requirement.

Prevalence of hypertension in Mexico

Given the concern about hypertension it needs to be understood that Mexico has an extraordinarily high prevalence of hypertension. Thus the prevalence of hypertension in adults, assessed in the National Health Survey 2000, was 30.7%, with prevalences of 34.2% in men and 26.3%, in women.[44,45] It was also shown that about a third of children are overweight[46] and this overweight/obesity is recognised as a promoter of hypertension. On a global basis hypertension affects a billion people[47] and is an important cause of morbidity in both industrialized and developing countries.[48]

Evidence that associates salt intake with the development of hypertension

Cross-sectional epidemiological, migrant and dietary intervention studies in children and adults have proven the causal relationship between a high salt intakes and the blood pressure level,[49] there being evidence that some individuals have a genetically based familial predisposition to developing hypertension on high salt intakes.[50,51] Animal studies also prove the induction by salt of higher blood pressures, these elevated pressures becoming self-perpetuating, a feature which is again modulated by genetic factors; thus the environmental-genetic interaction of salt intakes in association with polygenic variations in the genes controlling the renin - aldosterone axis can be shown in animals as well as man.

Animal evidence

The animal evidence is interesting as it shows that with rodents one can progressively select rats which become ever more sensitive to the effects of salt on blood pressure[52,53] and if one continues the studies the life expectancy of the salt sensitive animals is markedly shortened. Chimpanzee studies have shown similar effects: one group was fed on only 10 mmol sodium (585 mg NaCl) whereas the other was given 15 to 20 times as much-equivalent to many current human intakes. After two years there had been a progressive increase in blood pressure, especially systolic blood pressure in the high salt group until the average systolic level was over 30 mmHg higher than in the group on the low salt diet.[54]

The mechanisms by which dietary salt increases blood pressure are not completely understood but the constant input of sodium to expand the extracellular volume may in addition to hormonal effects alter the vascular responsiveness of the arterioles thereby increasing the peripheral resistance.

Human epidemiological analyses

The first epidemiologic investigation showing the relation between salt intake and hypertension was Thomas' study in Greenland Eskimos in 1927,[55] this being rapidly followed by assessments in Australian natives,[56] Mountain tribes in China[57] and in Indian Cuna in Panama.[58] An historical overview of how to prevent the development of hypertension has recently been summarized by Elliott and Stamler[59] who noted that the normal rise in blood pressure with age does not happen in communities on low salt intakes. Thus of the six Solomon Island populations only one, the Lau, had high blood pressures and they were different in cooking their food in salt rich water from a Pacific inlet.[60] By contrast the Qash'quai nomads of Iran had high salt intakes and despite their body weights not increasing with age (a factor promoting hypertension) they still had a marked age related increase in blood pressure.[61]

Since then, there have been numerous studies with one of the most famous being the previously cited INTERSALT[26] study of 52 communities in 32 countries. The levels of blood pressure within the populations as well as between different groups related to their 24 h sodium excretion. After adjusting for body weight, alcohol intake, sex and age, a sodium excretion of consumption of greater than 100 mmol was associated with a 3 to 6 mmHg rise in systolic blood pressure if adults up to 40 years were considered but with an increase of 10 mmHg in those aged 40 to 70 years.[12,26]

Migrant studies also allowed ethnic differences to be eliminated as the factor determining hypertension because changes in blood pressure levels occurred within three months of African young men transferring from a rural community to the conditions in Nairobi where their diet was totally changed with increases in salt as well as fat, sugar and other mineral intake changes such as a fall in potassium intakes.[62,63] Some other cations such as potassium and calcium may also affect blood pressure[64] with clinical studies suggesting that increased potassium intakes

decrease blood pressure, particularly in hypertensive patients and in those on high sodium intakes.[12]

An age related factor has already been noted so it may not be so surprising that the effects of different sodium intakes are evident even in newborns. Thus a German study[65] showed that when one group with over 200 babies were given, for their first 6 months of life, a milk formula with a lower salt intake then this group had systolic blood pressure 2 mmHg lower than another control group. The study was then discontinued but when these children were contacted much later at the age of 15 years those who had been on a lower salt intake for their first six months showed statistically lower blood pressures, implying that there is an imprinting of salt responsiveness determining subsequent salt intakes or the blood pressure control mechanism is "set" during infancy. Recent evidence that lower birth weight babies have a greater drive for salt by virtue of changes in salt taste sensitivity[66] also implies that the higher salt intakes in the first six months of life imprinted the taste for salty foods so that this became an automatic behavioural drive of these infants. Less likely is the self-perpetuating amplification of the small differences in blood pressure during infancy. Whatever the explanation this is an alarming finding implying that we should be extremely concerned about the early exposure of babies and children to salt intakes.

INTERVENTION STUDIES

Interventions early in the twentieth century[66] had shown that those with hypertension improved when their salt intakes were lowered and the hypertensive problem could be eliminated if the salt was reduced to the minimum.

In Britain, Marmot[67] estimated that a relatively small reduction (2-3 mmHg) in mean blood pressure in the population (if the distribution remained similar), would result in a major benefit in terms of mortality. A shift of this kind in the general population would be equivalent to that achieved by hypertensive therapy in those individuals with markedly raised blood pressure. He showed that a fall in dietary salt intake from 12 to 5 grams might be expected to result in a blood pressure reduction of at least 5 mmHg diastolic and 10 mmHg systolic.

Recently a series of major studies in the US referred to as the DASH (Dietary Approaches to Stop Hypertension) trials have provided definitive proof of the value of reducing salt intake in both normotensive and hypertensive adults and distinguished between the contributions of other dietary factors. The first of the studies[68] compared three feeding schemes: a control diet similar to that typical of American diets, then a second diet with 3.6 and 1.3 more fruit and vegetables servings per day respectively (the approximate weight of a serving being 80 g). The third diet was reduced in total fat to 24% with the use of low fat dairy products which also allowed the fall in saturated fat intake to 6%. Care was taken to keep the total sodium intake to 140 mmol per day and with adjustments in total energy intake to keep body weights constant. Several centres took part in this highly controlled study involving 459 subjects with normal blood pressure or blood pressure levels of less than 160 mmHg systolic blood pressure and 80 to 95 mmHg for diastolic blood pressure. Each diet was fed for eight weeks and with a wash out period on the control diet of three weeks. The results were dramatic in demonstrating the additive effects in lowering blood pressure by the use of extra fruit and vegetables and the lowering of total and saturated fat intakes. Hypertensive subjects were also shown to have 11.4/5.5 mmHg falls in the systolic/diastolic blood pressures i.e. over three times the fall in systolic blood pressure and a two an a half times greater fall in diastolic blood pressure than observed in the normotensive volunteers whose blood pressures also fell by 3.5/2.1 mmHg.

The second study, called "DASH-Sodium",[69] assessed the effect on blood pressure of reducing sodium intake to either 106-7 mmol or to about 65 mmol on both the control and the full intervention DASH diet. Again 412 normotensive and hypertensive participants were randomly assigned to one of the two feeding plans and all of the participants consumed one of the three sodium levels: 3,300 mg, 2,400 mg and 1,500 mg a day for a month each. Whereas the full DASH diet on a high salt intake had reduced the systolic blood pressure by about 6 mmHg the lowest sodium intake on the typical normal American diet reduced it by about 7 mmHg. The two approaches were to some extent complementary because only a 3 mmHg further fall occurred if the low salt intake was used together with the full DASH diet. Nevertheless in these normotensive subjects there was about a total 7 mmHg fall in systolic pressure if all three dietary interventions were used. The hypertensive subjects had greater reductions than the healthy people.[69] Thus a moderate reduction in sodium intakes has approximately the same effect on blood pressure as a simple antihypertensive drug e.g. a diuretic or beta-blocker.[70]

The combined effects on blood pressure of the DASH diet and low salt intake were greater than either of the interventions alone and were on average for the whole volunteer group 8.9/4.5 mmHg below the high salt control diet. With this combination, the mean systolic blood pressure was 11.5 mmHg lower in participants with hypertension, and 7.1 mmHg lower in participants without hypertension. The results of the DASH Sodium trial showed that blood pressure was reduced in a stepwise fashion in response to a reduction in the salt intake on both the DASH diet and the control diet. The effects were observed in those with and without hypertension, in both sexes, and across racial groups.

These data show that normotensive as well as hypertensive groups benefit from lower salt intakes with lower blood pressures. Of course the hypertensives benefit more but there is no clear distinctive feature between the normotensive and hypertensive groups other than the arbitrary distinction set by having a single number for the systolic blood pressure as the cut-off point for specifying hypertension. Furthermore the blood pressure distributions across the population is a continuous smooth curve so the multiple polygenic gene-environmental interactions do not allow a special group of hypertensives to be identified genetically.[71] Therefore the whole population benefits from lowering dietary salt intake and, given the large numbers with borderline hypertension in a population, particularly as they progressively raise their blood pressures with age, the effects of preventive measures in the so-called normotensives will be just as great if not greater than the effective treatment of the hypertensive group only.

Community interventions

Forte and colleagues[72] undertook a simple study in two villages in Portugal. In one they asked the people to use less salt but more importantly arranged for the local village baker to reduce the salt added to bread on a routine daily basis. The blood pressure at the beginning of the study were similar in both populations but at the end of both the first and second year the blood pressure was significantly lower in the village given such advice where the baker had changed the salt content of the bread.

APPROACHES TO THE PREVENTION OF HYPERTENSION

Lowering salt intakes-the main sources of dietary salt

In order to be able to suggest to patients or indeed to populations how they reduce their salt intake we need to know the origin of their main sources of dietary salt - is it the salt that is in drinking water, in special foods, or simply that salt that has traditionally been used at the table or in cooking? Some years ago we introduced a new technique involving the use of a lithium tracer marking of salt to evaluate the principal sources in the diet.[73-75]

In western countries there are two principal sources of salt in the diet: a) *discretionary sources*, which include the salt that is added to the foods during its preparation in the kitchen or at table and b) *the non-discretionary sources* where people simply purchase the foods or drinks without knowing what their salt contents are.[12,76]

Contrary to popular belief the non-discretionary sources in Western Communities that rely on processed foods or where an appreciable proportion of the diet is bought outside the home totally dominates salt intakes and the discretionary salt only amounts to between 9-15 percentage in men and 18-20 percentage in women.[76] Table 4 shows the salt sources in different communities in the world using the lithium technique.[7]

In England, the sources of discretionary salt represented only 15% of total intake, while the remaining 85% came from non-discretionary sources.[75] In Italy the discretionary sources represented more than one third of the total consumption in the three communities because in the Italian culture there was, at the time of the study, still a great deal of home cooking.[77] In Guatemala[78] a small study in 9 mothers of a rural community demonstrated that the use of discretionary salt is much more important because home cooking predominates whereas in another rural community of Benin[78] the non-discretionary salt already amounts to half of total intake. The lithium technique has not been applied in Mexico but using a variety of other techniques it is becoming clear that the process of industrialization and urbanization affects the composition of the many foods.[7] This is shown in Table 5 where it is clear that the more processed the food the higher the salt content and the lower the potassium. Thus corn on the cob in its natural form contains very little sodium but when it is used for tortilla production the sodium content almost triples. When maize is transformed into breakfast cereals (cornflakes) or into processed snacks the sodium concentration is increased over 200 times. Again traditional beans cooked at home have a very low concentration of sodium, but when processed their sodium content increases 14 times.[79]

Table 4. The sources of salt value through the technique of lithium marker.

Salt sources	England[75]	Italy[77]			Guatemala[78]	Benin[78]
		Cagli	Vallo	Rome		
Discretionary sources (%)	15	41	40	31	77	52
Nondiscretionary sources (%)	85	59	60	69	23	48

Sources: Modified from Sánchez-Castillo CP, James WPT.[7]

Table 5. Effects of industrialization in mexican food composition.

Food	Content in mmol per 100 g of fresh weight		
	Na	K	Ca
Maize based products			
Corn on the cob	4	284	55
Tortilla	11	192	177
Breakfast cereals	866	101	3
Processed snacks	838	197	102
Pulses			
Unprocessed, cooked	14	470	67
Processed, in cans	354	371	26

Source: Sánchez-Castillo CP, *et al.*[7,79]

Thus these industrial processes seem to explain substantially why Mexican intakes are now 15-20 times the physiological requirements of sodium.[36] The problem is affecting our children's diets too because of the high salt content of baby foods[80] and the high sodium content of children's snack foods and other fast foods.[70,81]

BENEFITS OF A LOW SALT INTAKE IN MEXICO

An apparently simple reduction in the consumption of salt it would be expected to have clinical as well as an economic and social benefit. Clinically it is now clear that doctors should be focussing on the diet and activity of their patients. In Finland patients are not reimbursed by the state for the cost of their drugs for hypertensive therapy until the patient has gone through a series of dietary changes involving an increase in fruit and vegetable consumption, a lowering of fat intake, reducing their body weight and alcohol consumption and increasing their physical activity level as well as reducing their salt intakes! The life style modifications to reduce blood pressure cannot any longer be seen to be vague non-scientific advice but a requirement of good clinical practice backed by meticulously prepared objective data where the dietary changes can reinforce the effectiveness of antihypertensive medicines and decrease cardiovascular risk.[47]

Indeed the DASH trial[68,69] showed that dietary intervention could substitute for drug intervention but our analysis of the sources of salt[73-75] (and in practice of other fat and fruit and vegetable intakes) means that the advice to any patient has to include a dietary change for the whole family and involving the minimum use of purchased processed foods, avoiding adding fats and oils in cooking, limiting or avoiding sugary drinks to limit weight gain and with a focus on more home cooked food with the use of a progressively smaller amount of salt to allow the taste buds to adapt. The taste preference for salt and salty foods is a learned preference,[82] so after a few days the taste adapts to a modest (but not to a major sudden reduction) in salt intakes. It has been shown that people become accustomed over a period of a few weeks to modest but progressive falls in salt intakes. Once this adaptation has occurred individuals will reject higher salted foods and opt for lower salt intakes. The best practice therefore demands a coherent progressive approach to dietary change backed by specific advice on how to avoid the highest salted food purchases.

A new public health policy on salt involving the food industry in Mexico

The main observations in INTERSALT[26] are that: 1) for individuals, a difference of 100 mmol (equivalent to 5.85 g NaCl) per day in sodium intake is associated on average with a difference of 3 to 6 mmHg in systolic blood pressure; and 2) for populations, a 100 mmol per day lower sodium intake is associated with attenuation of the rise in systolic blood pressure by 10 mmHg in persons aged 25 to 55 years. As with observations in several other isolated, preliterate populations, the four remote INTERSALT population samples had both a low sodium excretion and a low blood pressure with little or no upward slope of blood pressure with age and little or no hypertension.[26,83] A universal reduction of dietary salt of 50 mmol Na, equivalent to 3 g salt would

reduce Mexico's population blood pressure by an average of 5 mmHg. On a population basis it has been estimated that a reduction of 2 mmHg in diastolic blood pressure would result in 15% reduction in risk of stroke and transient ischemic attacks, and a 6% reduction in risk of coronary heart disease.[84] McPherson and colleagues[85] have estimated that 6% of deaths from coronary heart disease could be avoided if the numbers of people with high blood pressure were reduced by 50%.

These assessments then explain why current public health recommendations propose that salt intake should be reduced from 9-12 to 5-6 grams per day for adults. In Mexico, the government has endorsed this recommendation in the year 2000.[86] Draft guidelines from the Foods Standards Agency in Great Britain, indicate that infants (0 to 12 months) should be limited to intakes of 1 g salt per day, children from 1 to 6 years to 2 g, and children from 7 to 14 years to less than 4 g.[35] A conservative estimate indicates that a reduction of 3 g per day would reduce strokes by 13% and ischemic heart disease by 10%. The effects would be almost doubled with a 6-gram reduction and tripled with a 9-gram one.[67] A reduction of salt intake by 5.85 g (100 mmol) is feasible if the food industry co-operated and another estimate suggests that this by itself could reduce the mortality by stroke and ischemic heart disease by 39% and 30%, respectively in a long term.[87]

The food industry could also help if it labelled its products with simple and comprehensible information about the sodium content of the food but at present the US and other legally valid systems are incomprehensible to the ordinary person.

The recommendation of how to reduce the salt intake in Mexican rural areas might well need a public health campaign to limit salt use in cooking and at the table. Avoiding salty foods and not adding salt to food when cooking or at the table, reduces the salt intake by about 50 mmol Na or 3 g salt, the amount depending on the culinary traditions. In order to reduce the salt intake by twice this amount means also avoiding processed food consumption.[87]

It is therefore of great importance to create awareness of the dangers that an excess consumption of salt does to health, moreover in a population such as the Mexican population where the prevalence of essential hypertension is extraordinarily high, and the consumption of non-discretionary sources such as processed and fast foods are increasing out of proportion.

REFERENCES

1. Salt Institute. History of Salt. Available at: http://www.saltinstitute.org Accessed date: April 11, 2006.
2. Sánchez-Castillo CP. La sal a través de la historia. Su papel en el desarrollo de los pueblos. *Cuadernos de Nutrición* 1988; 11(4): 33-9.
3. Multhauf RP. Neptune's gift: a history of common salt. Baltimore: John Hopkins Press; 1978.
4. MacGregor GA, De Wardener HE. Salt, diet and health. U.K.: Cambridge University Press; 1998.
5. Encyclopaedia Britannica. Chemoreception in the vertebrates. Available at: http://www.britannica.com/ebc/article-9380308?query=hemoreception%20mammals&ct= Accessed date: December 9, 2006
6. Salt Institute. Consumer Tips for Salt Use. Available at: http://www.saltinstitute.org/29.html Accessed date: April 10, 2006.
7. Sánchez-Castillo CP, James WPT. Salt epidemiology. Encyclopedia of Human Nutrition. 2th Ed. 2005.
8. Marthaler TM, Petersen PE. Salt fluoridation: an alternative in automatic prevention of dental caries. *Int Dent J* 2005; 55(6): 351-8.
9. Norma Oficial Mexicana NOM-040-SSA1-1993, Bienes y Servicios. Sal yodada y sal yodada fluorurada. Especificaciones sanitarias. Fecha de publicación: 13 de marzo de 1995.
10. Chaudhari N, Yang H, Lamp C, Delay E, Cartford C, Than T, Roper S. The taste of monosodium glutamate: membrane receptors in taste buds. *J Neurosci* 1996; 16: 3817-26.
11. Turnbull GK, Vanner SJ, Burnstein M. The colon. 3rd Ed. Chapter 11. In: Thomson ABR, Shaffer EA (eds.). First pinciples of gastroenterology: the basis of disease and an approach to management. Janssen-Ortho, editorial. Canadian Association of Gastroenterology. Astra Zeneca; 2005, p. 350.
12. Sánchez-Castillo CP, López P, Pichardo-Ontiveros E. Sodio, cloro y potasio. En: Bourges H, Casanueva E, Rosado JL (eds.). Recomendaciones de Ingestión de Nutrimentos para la población mexicana. Bases fisiológicas. Tomo I. México: Editorial Médica Panamericana; 2005, p. 195-207.
13. Banks MR, Farthing MJ. Fluid and electrolyte transport in the small intestine. *Curr Opin Gastroenterol* 2002; 18 (2):176-81.
14. Sandle GI. Salt and water absorption in the human colon: a modern appraisal. *GUT* 1998; 43: 294-9.
15. Sánchez-Castillo CP. The sources of salt in the British Diet. PHD Thesis. United Kingdom: The University of Cambridge; 1985.
16. Dahl LK. Salt in processed baby foods. *Am J Clin Nutr* 1968; 21: 787-92.
17. Spitzer A. The role of the kidney in sodium homeostasis during maturation. *Kidney International* 1982; 21: 539-45.
18. McCance RA, Widdowson EM. Mineral metabolism of the fetus and new-born. *Brit Med Bull* 1961; 17: 132-36.
19. Oliver WJ, Cohen EL, Neel JV. Blood pressure, sodium intake and sodium related hormones in the Yanomamo Indians, a "no-salt" culture. *Circulation* 1975; 52: 146-51.
20. Mancilha-Carvalho JJ and Silva Nelson AS. The Yanomami Indians in the INTERSALT study. *Arq Bras Cardiol* 2003; 80(3): 295-300.
21. Maddocks I, Rovin L. A New Guinea population in which blood pressure appears to fall as age advances. *Papua New Guinea Med J* 1965; 8: 17-21.
22. Barret KE. New insights into the pathogenesis of intestinal dysfunction: secretory diarrhea and cystic fibrosis. *World J Gastroentero* 2000; 6(4): 470-4.
23. Ashworth A, Harrower ADB. Protein requirements in tropical countries: nitrogen losses in sweat and their relation to nitrogen balance. *Br J Nut* 1967; 21: 833-43.

24. Appendix Dietary Reference Values (terminology: UK Department of Health). In: Human Nutrition. 11th Ed. Geissler C, Powers H (eds.). Elsevier Churchill Livingstone; 1991.
25. Connor WE, Cerqueira MT, Connor RW, et al. The plasma lipids, lipoproteins and diet of the Tarahumara Indians of Mexico. *Am J Clin Nutr* 1978; 31: 1131-42.
26. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. Intersalt Cooperative Research Group. *BMJ* 1988; 297: 319-28.
27. Sánchez-Castillo CP, Solano ML, Flores J, et al. Salt intake and blood pressure in rural and metropolitan Mexico. *Arch Med Res* 1996; 27: 556-9.
28. Sánchez-Castillo CP, Escamilla-Cejudo JA, Velázquez C, et al. Muestras de orina de 24 horas necesarias para caracterizar la ingestión de sodio en voluntarios mexicanos sanos. *Acta Bioquím Clín Latinoam* 1993; 27(3): 313-23.
29. Grijalva M, Valencia M, Wyatt J. Sodium, potassium and calcium intake in adults consuming normal diets in Northern Mexico determined by analytical and calculated methods. *J Food Compos Anal* 1992; 5: 127-33.
30. Ballesteros-Vásquez MN, Cabrera-Pacheco RM, Saucedo-Tamayo RM, Grijalva-Haro MI. Consumo de fibra dietética, sodio, potasio y calcio y su relación con la presión arterial en hombres adultos normotensos. *Salud Pública Méx* 1998; 40(3): 241-7.
31. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? *BMJ* 1991; 302: 811-5.
32. WHO Expert Committee on Prevention in Childhood and Youth of Adult Cardiovascular Disease. Prevention in childhood and youth of adult Cardiovascular disease-time for action: report of a WHO Expert Committee. World Health Organ Tech Rep Ser 1990; 792.
33. Dietary Guidelines Advisory Committee on the dietary guidelines for Americans. Washington, DC: US Government Printing Office; 1995.
34. National Research Council, Committee on Diet and Health, Food and Nutrition Board, Commission on Life Sciences: Diet and Health: implications for reducing chronic disease risk. Washington, DC: National Academy Press; 1989.
35. Scientific Advisory Committee on Nutrition, 2003. Salt and Health. London: The Stationary Office. Publisher for the Food Standards Agency and The Department of Health.
36. Sánchez-Castillo CP. La sal y la salud. *Cuadernos de Nutrición* 1988; 11(5): 3-9.
37. Buiatti E, Palli D, Decarli A, Amadori D, Avellini C, Bianchi S, et al. A case-control study of gastric cancer and diet in Italy: II. Association with nutrients. *Int J Cancer* 1990; 45(5): 896-901.
38. Tsugane S, Sasazuki S, Kobayashi M, Sasaki S. Salt and salted food intake and subsequent risk of gastric cancer among middle-aged Japanese men and women. *Br J Cancer* 2004; 90: 128-34.
39. MacGregor GA. Salt: Blood pressure, the kidney and other harmful effects. *Nephrol Dial Transplant* 1998; 13(10): 2471-9.
40. Joossens JV, Hill MJ, Elliot P, et al. Dietary salt, nitrate and stomach cancer mortality in 24 countries. European Cancer (ECP) and the Intersalt Cooperative Research Group. *Int J Epidemiol* 1996; 25(3): 494-504.
41. Antonios TFT, MacGregor GA . Deleterious effects of salt intake other than effects on blood pressure. *Clin Exp Pharmacol Physiol* 1995; 22: 180-4.
42. Burney PGJ. A diet rich in sodium may potentiate asthma: epidemiological evidence for a new hypothesis. *Chest* 1987; 91 (Suppl.): 143S-148S.
43. Turkann JS, Goldstein DS. Stress and sodium hypertension in baboons: neuroendocrine and pharmacotherapeutic assessments. *J Hypertens* 1991; 9(10): 969-75.
44. Sánchez-Castillo CP, Velázquez-Monroy O, Berber A, et al. Anthropometric cut-off points for predicting chronic diseases in the Mexican National Health Survey 2000. *Obes Res* 2003; 11(3): 442-51.
45. Velázquez MO, Rosas PM, Lara EA, Pastelín HG. Hipertensión arterial en México: Resultados de la Encuesta Nacional de Salud (ENSA) 2000. *Arch Cardiol Mex* 2002; 72: 71-84.
46. Del Río-Navarro BE, Velázquez-Monroy O, Sánchez-Castillo CP, et al. The high prevalence of overweight and obesity in Mexican Children. *Obes Res* 2004; 12(2): 215-23.
47. Chobanian A, Bakris G, Black H, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The JNC 7 Report. *JAMA* 2003; 289(19): 2560-72.
48. Sánchez-Castillo CP, Velásquez-Monroy O, Lara-Esqueda A, et al. Diabetes and hypertension increases in a society with abdominal obesity: results of the Mexican National Health Survey 2000. *Public Health Nutr* 2005; 8: 53-60.
49. Meneton P, Jeunemaitre X, de Wardener HE, Macgregor GA. Links between dietary salt intake, renal salt handling, blood pressure and cardiovascular diseases. *Physiol Rev* 2005; 85(2): 679-715.
50. Weinser R. Salt and the development of essential hypertension. *Prev Med* 1976; 5:7-14. In: Grijalva M, Valencia M, Wyatt J (eds.). Sodium, potassium and calcium intake in adults consuming normal diets in Northern Mexico determined by analytical and calculated methods. *J Food Compos Anal* 1992; 5: 127-33.
51. Altschul AM, Grommet JK. Sodium intake and sodium sensitivity. *Nutr Rev* 1980; 38: 393-401.
52. Dahl LK. Possible role of chronic excess salt consumption in the pathogenesis of essential hypertension. *Am J Cardiol* 1961; 8: 571-5.
53. Meneely GR. Electrolytes in hypertension: the effects of sodium chloride. *Med Clin N Am* 1961; 45: 271-83.
54. Denton D, Weisinger R, Munday NI, et al. The effect of increased salt intake on blood pressure of chimpanzees. *Nat Med* 1995; 1: 1009-16.
55. Thomas WA. Health of a carnivorous race: a study of the Eskimo. *JAMA* 1927; 88: 1159-60.
56. Hicks CS, Matters RF. Standard metabolism of the Australian aborigenes. *Aust J Exp Bio-Med Sci* 1933; 11: 177-83.
57. Morse WR, Beh YT. Blood pressure amongst aboriginal ethnic groups of Szechwan province, West China. *Lancet* 1937; i: 966-7.
58. Kean BH. The blood pressure of the Cuna Indians. *Am J Trop Med* 1944; 24: 341-3.
59. Elliot P, Stamler J. Primary prevention of high blood pressure. In: Marmot M, Elliot P (eds.). Coronary heart disease epidemiology. From aetiology to public health. 2nd Ed. Chapter 43. Oxford University Press; 2005, p. 751-68.
60. Page LB, Damon A, Moellering RC. Antecedents of cardiovascular disease in six Solomon Islands societies. *Circulation* 1974; 49: 1132-46.
61. Page LB, Vandevert DE, Nader K, et al. Blood pressure of Qash'qai pastoral nomads in Iran in relation to culture, diet, and body form. *Am J Clin Nut* 1981; 34: 527-38.
62. Shaper AG, Leonard PJ, Jones KW, Jones M. Enviromental effects on body built, blood pressure and blood chemistry of nomadic warriors serving in the army in Kenya. *East Afr Med J* 1969; 48: 262-89.
63. Poulter NK, Khaw KT, Hopwood BEC, et al. The Kenyan Luo migration study: observations on the initiation of a rise in blood pressure. *BMJ* 1990; 300: 967-72.
64. Durán E, Soto D, Asenjo G, et al. Ingesta dietaria de sodio, potasio y calcio en embarazadas normotensas. *Rev Chil Nutr* 2002; 29: 40-6.

65. Geleijnse JM, Hofman A, Witteman JCM, et al. Long-term effects of neonatal sodium restriction on blood pressure. *Hypertension* 1997; 29: 913-17.
66. Stein LJ, Cowart BJ, Beauchamp GK. Salty taste acceptance by infants and young children is related to birth weight: longitudinal analysis of infants within the normal birth weight range *Eur J Clin Nutr* 2006; 60(2): 272-9.
67. Marmot MG. Diet, hypertension and stroke. In: Turner MR (ed.). Nutrition and health. British Nutrition Foundation, MTP Press Ltd; 1982.
68. U.S. Department of Health and Human Services, National Institutes of Health, National Heart, Lung and Blood Institute. National High Blood Pressure Program. The DASH Eating Plan. NIH Publication No. 03-4082. Originally Printed 1998. Reprinted February 1999. Revised May 2003.
69. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med* 2001; 344: 3-10.
70. MacGregor GA. Dietary sodium and potassium intake and blood pressure. *Lancet* 1983; 2: 750-3.
71. Corvol P, Persu A, Gimenez-Roqueplo A-P, Jeunemaitre X. Seven lessons from two candidate genes in human essential hypertension. *Hypertension* 1999; 33: 1324-31.
72. Forte JG, Pereira JM, Pereira MJ, et al. Salt and blood pressure: a community trial. *J Human Hypertens* 1989; 3: 179-84.
73. Sánchez-Castillo CP, Seidell J, James WPT. The potential use of lithium as a marker for the assessment of the sources of dietary salt: cooking studies and physiological experiments in man. *Clin Sci* 1987; 72: 81-6.
74. Sánchez-Castillo CP, Branch WJ, James WPT. A test of validity of the lithium-marker technique for monitoring dietary sources of salt in man. *Clin Sci* 1987; 72: 87-94.
75. Sánchez-Castillo CP, Warrender S, Whitehead TP, James WPT. An assessment of sources of dietary salt in a British population. *Clin Sci* 1987; 72: 95-102.
76. James WPT, Ralph Ann, Sánchez-Castillo CP. The dominance of salt in manufacturated food in the sodium intake of affluent societies. *Lancet* 1987; 21: 426-8.
77. Leclercq C, Ferro-Luzzi A. Total and domestic consumption of salt and their determinants in three regions of Italy. *Eur J Clin Nutr* 1991; 45: 151-9.
78. Melse-Boonstra A, Rozendaal M, Rexwinkel H, et al. Determination of discretionary salt intake in rural Guatemala and Benin to determine the iodine fortification of salt required to control iodine deficiency disorders: studies using lithium-labeled salt. *Am J Clin Nut* 1998; 68: 636-41.
79. Sánchez-Castillo CP, Dewey PJS, Reid MD, et al. The mineral and trace element content of mexican cereals, cereal products, pulses, and snacks: preliminary data. *J Food Compos Anal* 1997; 10: 312-33.
80. Heird WC, Ziegler P, Reidy K, Briefel R. Current electrolyte intakes of infants and toddlers. *J Am Diet Assoc* 2006; 106: S43-S51.
81. Gregory J, Lowe S. "National Diet and Nutrition Survey: Young People aged 4 to 18 years". Volume 1: a survey carried out in Great Britain on behalf of the Ministry of Agriculture, Fisheries and Food and the Departments of Health by the Social Survey Division of the Office for National Statistics and Medical Research Council Human Nutrition Research. Office for National Statistics, The Stationery Office, London. 2000.
82. Denton D. The hunger for salt. Springer-Verlag Berlin Heidelberg New York: 1982.
83. Kotchen TA, McCarron DA. Dietary electrolytes and blood pressure. *Circulation* 1998; 98: 613-17.
84. Cook NR, Cohen J, Hebert PR, et al. Implications of small reductions in diastolic blood pressure in primary prevention. *Arch Intern Med* 1995; 155: 701-9.
85. McPherson K, Brittan A, Causer L. Coronary heart disease: estimating the impact of changes in risk factors. London 2002. The Stationery Office.
86. Norma Oficial Mexicana NOM-030-SSA-1999, Para la prevención, tratamiento y control de la hipertensión arterial. Publicada en el Diario Oficial de la Federación el 5 de abril de 2000.
87. Law MR, Frost MR, Wald NJ. By how much does dietary salt restriction lower blood pressure? III. Analysis of observational data within populations. *BMJ* 1991; 302: 819-24.

Correspondence and reprint request:

Dra. Claudia P. Sánchez-Castillo
Instituto Nacional de Ciencias Médicas y Nutrición
Salvador Zubirán.
Departamento de Fisiología de la Nutrición
Vasco de Quiroga No.15
Col. Sección XVI, Tlalpan
14080 México, D.F.
Tel. and fax: (55) 5513-7235
E-mail: kailas@prodigy.net.mx

Recibido el 12 de febrero de 2007.
Aceptado el 22 de junio de 2007.