

Diet, Hypertension and Salt Toxicity

By James J. Kenney, PhD, RD, LD, FACN Copyright 1998-2011 Food & Health Communications, Inc. This CPE course has been approved by the American Dietetic Association for 8 unit hours.

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Lesson Rationale

Hypertension is by far the most commonly diagnosed cardiovascular disease with over 75 million Americans now with hypertension and many more with pre-hypertension. Recent data show that about 60% of American adults have hypertension or prehypertension.¹ The majority of Americans now have hypertension by age 65y. At least 90% of the minority that do not have hypertension at age 65y will be developing it in the next 20 years.[24]

This is despite the fact that hypertension can largely be prevented and/or reversed simply by changing one's diet. Few will be able to adopt a DASH-style diet very low in salt (sodium) without dietary counseling. The potential to avert this growing epidemic of hypertension and its adverse effects on health should provide a huge potential growth area for Registered Dietitians and other health professionals that can provide medical nutritional therapy for the prevention and treatment of this very common disease.

Introduction

Salt is perhaps the oldest and most commonly used food additive. It improves the taste of many foods without adding calories. In larger amounts it also inhibits the growth of many microorganisms and has been used to preserve food throughout much of man's recorded history. So important was salt for preserving foods that it was often part of the pay Roman soldiers received, hence the word "salary." Today, refrigeration, modern canning and other food processing techniques have made this once essential preservative role of salt largely superfluous. However, most people who have become accustomed to the taste of salt come to prefer salted over unsalted food. This is why the consumption of salt has remained excessively high in modern societies despite the fact that it is no longer needed to prevent food spoilage. The average American consumes about 3,000 to 5,000 mg (130-217 mmol) of sodium per day or about 7.5 to 12.5 grams of salt daily. Ten grams of salt is about 2 teaspoons sodium chloride. Salt is 40% sodium and 60% chloride by weight. In modern Westernized societies only about 10% of dietary salt comes naturally from the foods consumed. Another 15-20% or so comes from the saltshaker. This leaves more than three-fourths of the salt in the typical Western diet coming from processed foods and meals consumed away from the home.²[CDC].

Handy measurement facts for salt and sodium:

Salt is sodium chloride – here are some quick equivalents:

1 gram of salt has 400mg of sodium and 600mg of chloride

1 tsp of salt weighs a little more than 5g or 5000mg

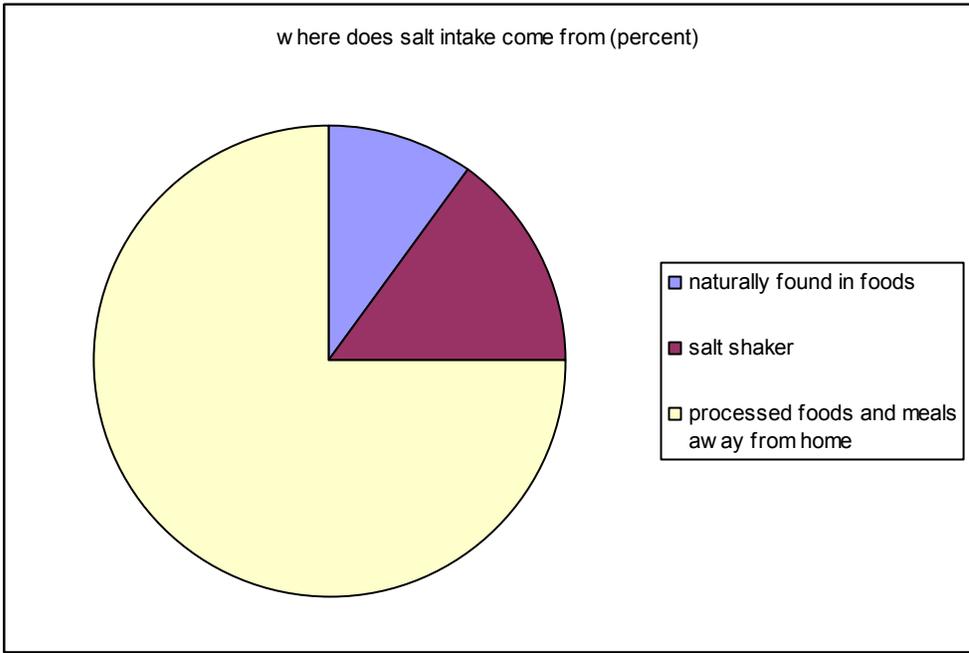
1 tsp of salt has a little more than 2000mg of Sodium and 3000mg of Chloride

The atomic weight of sodium is 23 (Chloride is 35.5).

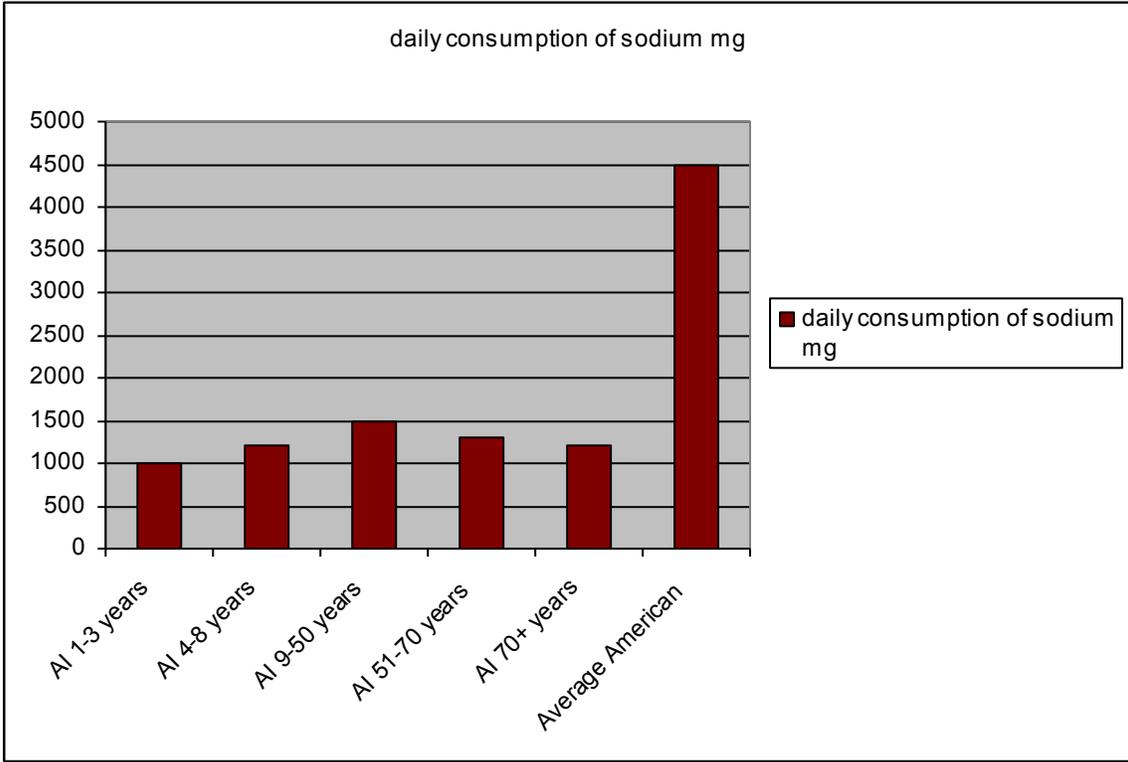
One mole of sodium is its molecular weight in grams or 23kg

A mmol or millimole (1/1000 of a mole) of sodium is 23mg.

One tsp of salt has about 94mmol of sodium



What is the New Adequate Intake (AI) for Salt?



Both sodium and chloride are essential nutrients for humans and all other animals. Nutrition researchers have known about the need for salt much longer than most other nutrients. Oddly enough there is no RDA for sodium or chloride although the 10th edition

of the Recommended Dietary Allowances states "the minimum average requirement for adults ... [is]... 115 mg of Sodium...per day. In consideration of the wide variation of patterns of physical activity and climate exposure, a safe minimum intake might be set at 500 mg/day." [p. 253]. In February of 2004 the Food and Nutrition Board of the Institute of Medicine (IOM) released the "Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate". They stated that a Recommended Dietary Allowance (RDA) for sodium and chloride "could not be derived." "Hence an Adequate Intake (AI) is provided."³ The new guidelines do make it clear that the average American is consuming far more salt (sodium chloride) than is needed for good health. Indeed, this report sets a toxic Upper Limit (UL) for sodium at 2300mg per day, which means according to the IOM's experts most Americans are consuming what they consider a toxic dose of salt nearly every day. For young adults the new guidelines state that 1500 mg of sodium is an adequate intake (AI) and discourages even young healthy Americans from consuming more this amount. Unfortunately the average young American is now consuming at least twice this amount even if they never touch a saltshaker. This is because at least $\frac{3}{4}$ of the salt in the typical American diet comes already added to restaurant meals and most convenience foods.

The report lists hypertension, cardiovascular and kidney diseases as the major adverse effects of a toxic or excessive salt (sodium) intake. However, the report also notes evidence linking the toxic amount of salt in the American diet to atrophic gastritis and stomach cancer and increased calcium loss in the urine, which contributes to kidney stone formation and osteoporosis. The IOM report also sets a lower AI for older Americans because the toxicity of salt increases in older people. For those over 50y the new AI is 1300 mg/day and for those over 70y the new AI is 1200 mg/day. The report states that "Because the relationship between sodium intake and blood pressure is progressive and continuous without an apparent threshold, it is difficult to precisely set a UL, especially because other environmental factors (weight, potassium intake, alcohol intake) and genetic factors also affect blood pressure." Simply put, the more salt a person eats the higher their blood pressure will likely go over time. Currently more 90% of Americans can expect to be diagnosed with hypertension during their lifetime. The report also recommends Americans roughly double their current potassium intake up to 4700 mg/day by eating a lot more fruits and vegetables as this helps reduce some of the toxic effects of salt. If Americans did not exceed the new AI for salt, their blood pressure would likely

rise little or not at all with age. Thus, far fewer Americans would certainly die from cardiovascular disease and other illnesses linked to salt toxicity.

Researchers have estimated that the diet of early man averaged only about 600 to 750 mg of sodium or about 25 to 33 mmol of salt.^{4 5} There is no doubt that human beings and other primates evolved consuming a diet with far less salt than is now customary in modern societies. It seems likely then that the human body is biologically designed to handle far less salt than is now the norm in modern diets. This certainly suggests that putting far more salt into the human body than it evolved to handle could pose a significant physiological stress. In the short-term the body may be able to deal with physiological stress and at least maintain a “normal” blood pressure. However, over time physiological stress may do irreparable harm to the body, the most obvious being increased blood pressure and dramatically increased risk of cardiovascular events. Is there sufficient scientific research to justify telling all Americans to cut back on salt? If so by how much should salt intake be reduced? Or do only some Americans with certain medical conditions need to cut back on salt?

The American Heart Association, the National Heart Lung and Blood Institute, the U.S. Dietary Guidelines, and most other public health organizations have recommended reducing dietary sodium intake to no more than 2000 to 2400mg/day. The latest dietary guidelines due out in 2010 are likely to reduce the US Dietary Guideline to no more than 1500mg of sodium per day. There is no doubt that virtually all Americans are consuming far more salt than their bodies actually require. How harmful all the added dietary salt is is still a matter of on-going debate but there is a growing consensus that almost all Americans would probably be better off consuming less salt. As this review will demonstrate the preponderance of scientific evidence links excessive salt intake to a wide variety of disease processes. This is why most public health and consumer organizations recommend that Americans consume much less salt than is currently the norm. However, there are many who would suffer economic harm if salt intake were curtailed. Perhaps not surprisingly many big multi-national food companies claim there is still not sufficient scientific research to be telling all Americans to reduce their salt intake and many help fund the Salt Institute to argue their case. Even so many are now hedging their bets by reformulating many of their products by reducing added salt.

Is the Salt Institute a Reliable Source of Information about Salt?

The “designated hitter” for these big food companies is the Salt Institute. The Salt Institute’s funding comes mainly from companies with an economic interest in refuting or at least downplaying scientific research linking the amount of salt in the typical American diet to disease. The Salt Institute has long maintained the position that: "Healthy persons with normal blood pressure have no problems with sodium or salt intake."⁶ The Salt Institute’s propaganda (www.saltinstitute.org) gives the impression that only about 1/3 to 1/2 of those who already have hypertension (HTN) need cut back on dietary salt because they are "salt sensitive". The Salt Institute, as well as many doctors and even several medical researchers, continue to argue that because most of the U.S. population does not have HTN they do not have to worry about their salt intake. In addition, they also argue no more than half the patients with HTN should be concerned about their salt intake, because they are not “salt sensitive.” This leaves only about 10% of all Americans who need to cut back on salt.

The Salt Institute’s spin on “salt-sensitivity” seems to be that if BP does not drop substantially within a week or two of adopting a lower salt diet then this is proof that the amount of salt in that person’s diet was not the cause of their HTN. Therefore, only about one-third to one-half of those with HTN (the “salt-sensitive” ones) should be told to reduce their salt intake. Using this same “logic” we could “prove” that excessive alcohol intake does not cause cirrhosis of the liver and smoking tobacco does not cause emphysema because neither one goes away when you quit drinking or smoking. What if the Salt Institute’s “logic” on “salt” and HTN was applied to those who have cirrhosis of the liver or emphysema. If they don’t show marked improvement in lung or liver function tests within a week or two of quitting they would be called “non-responders” and told that they are not alcohol or tobacco “sensitive”. And it would follow that since they are not alcohol or tobacco “sensitive” it would not be important for them to quit or even reduce their consumption.

The now defunct Tobacco Institute was hardly a credible source for reliable information linking cigarette smoking to health problems because they had an obvious conflict of interests. Over time even the news media (which seems wedded to the notion that there are two sides to every issue) came to dismiss the veracity of claims coming from the

Tobacco Institute and this is what eventually doomed that institution. However, the Salt Institute's frequent press releases and pronouncements about the safety of dietary salt or even the dangers of too little salt often escape much critical commentary in the news media. At best, the news media will present the Salt Institute's position as if it warrants as much credibility as that of scientific researchers. Given the economic incentives of those that support and speak on behalf of the Salt Institute it would be wise to take everything they say with a grain of salt (figuratively but not literally).

Salt Toxicity Kills More Americans than Tobacco

Dr. Bibbins-Domingo presented data, which calculated the potential impact of reducing the salt intake at the American Heart Association's 49th Annual Conference on Cardiovascular Disease Epidemiology and Prevention. The average American adult consumes 9 to 12g of salt daily. According to Dr. Bibbins-Domingo's calculations she estimated that each 1g reduction of salt intake (or 400mg in sodium) in the average American's diet would prevent about 200,000 deaths from cardiovascular disease (CVD) over the next decade. She used the Coronary Heart Disease Policy model with additional data to assess stroke risk to develop a computer model of what would happen to the health of the US population in the future based on the reduction of dietary salt. The risk calculation was based on the known adverse impact of dietary salt on blood pressure and the subsequent increased risk of heart disease and stroke. According to her computer model if the average American cut their daily salt intake by 3g/day or sodium intake by 1200mg/day this would provide morbidity and mortality benefits "on the same order of magnitude as if we could eliminate smoking in the population."^{7 8}

Dr. Bibbins-Domingo's computer model estimated that over the next 10 years more than 800,000 life years could be saved for each 1g reduction in dietary salt intake. If the average American cut their salt intake in half it would dramatically cut the risk of stroke, heart attack and heart failure. It would also dramatically reduce the risk of kidney failure. Net-net cutting the excessive amount of salt the food industry now freely adds to the diet of Americans would save far more lives than if every American smoker quit and never smoked again. In several European countries pressure on the food industry to cut the amount of salt they add to foods along with public health promotional efforts for consumers to limit dietary salt have already proven successful. Since it is now clear

added dietary salt is killing more Americans than tobacco smoke it is time the FDA to reclassify salt as some other than a “generally recognized as safe” food additive. Sadly the US Food and Drug Administration has failed completely in its job to protect Americans from the unsafe drug nicotine and its most common and deadly delivery vehicle – cigarettes. They have also failed in their duty to protect Americans from unsafe amounts of salt being added to processed foods and restaurant foods. Given that salt is the #1 killer of Americans and tobacco is #2 perhaps it is time for the FDA to stop catering to the food industry and tobacco industries?

Those interested in how the Salt Institute attempts to distort and misinterpret scientific data in an obvious attempt to conceal the demonstrated harm caused by excessive dietary salt may want to review an article written by Richard L. Hanneman (president of the Salt Institute) and the critical commentaries that follow it.⁹ The Salt Institute may be losing the scientific debate about the role of salt in promoting HTN and other health problems but it is still winning the public relations battle in the media. A recent survey found that only about 10% of Americans are concerned about their salt intake. However, as we shall see, more than 90% of Americans will develop HTN at some point in their lives and excessive dietary salt is the primary causal agent. The role of the Salt Institute in distorting the evidence linking excessive salt intake to serious health problems has largely escaped examination in the US. However, outside the US the tactics of the Salt Institute public relations efforts have not always escaped scrutiny.¹⁰ In England, Finland and several other European countries their governments have already had some success at reducing salt intake and reducing HTN and CVD mortality as a result.

Mr. Hanneman, whose academic training consists of a bachelor’s degree in history/government and a master’s degree in history has testified as an expert on salt before the FDA, HHS’s Nutrition Policy Board, and the USDA’s Human Nutrition Subcommittee. He has claimed that the “evidence now shows clearly that only a small minority of the population really benefits from restricting salt.”¹¹ Mr. Hanneman is far from being alone with the opinion that dietary salt restriction is something only a small percent of “salt sensitive” hypertensive individuals need be concerned about. In an editorial review article published in Science, investigative reporter Gary Taubes stated, “After interviews 80 researchers, clinicians and administrators around the world, it is safe to say that if ever there was a controversy over the interpretation of scientific data this is it.”¹² Mr. Taubes

concludes, “After decades of intensive research, the apparent benefits of avoiding salt have only diminished. This suggests either that the true has now been revealed and is indeed small, or that it is nonexistent and researchers believing they have detected such benefits have been deluded by the confounding of other variables.”

Another investigative reporter, John Stossel on KABC TV’s 20/20 program apparently talked to many of the same experts as Gary Taubes judging from the show he did on salt. Were both Taubes and Stossel duped by the Salt Institute well-orchestrated public relations campaign? If so why are investigative reporters so easily misled by the Salt Institute? It appears that the pro-salt advocates efforts are well coordinated. By contrast, there is no well-funded “anti-salt” group promoting research scientists whose results show harmful effects from dietary salt and/or beneficial effects from reducing salt intake. No PR firms deliver carefully chosen anti-salt spokesman to eager investigative journalists out to uncover the “truth” about this “controversy” broiling over the American dietary staple of salt. No Anti-Salt Institute or anything comparable exists. Nor is their much to be gained financially by someone taking the anti-salt side of this “debate”. As a result, these journalists end up interviewing primarily those experts referred to them directly or indirectly by the Salt Institute.

Most Americans like salt and do not want to believe the amount they consume daily is toxic. A good chunk of KABC-TV’s advertising money is generated from many of the same food companies whose funding supports the Salt Institute. Network “news shows” are driven by ratings and the need for advertising dollars. Clearly John Stossel simply followed the path of least resistance to what he may well have been led to believe was the “truth”. It is less clear why *Science* published Gary Taubes’ editorial article on the politics of salt. *Science* does not typically publish what appears to be little more than food industry propaganda that appears to have been largely written by the Salt Institute’s president Richard Hanneman. Certainly, since its publication in *Science*, Mr. Hanneman has helped publicize Mr. Taubes’ article, which won a top prize from the National Association of Science Writers. The publication of Taubes’ polemic in *Science* certainly suggests that the political/economic influence of multinational food companies is growing. This review will highlight some of the scientific evidence that perhaps Richard L. Hanneman, John Stossel, Dr. Michael Alderman, and Gary Taubes apparently missed, ignored or misinterpreted. It is clear that commercial interests are conducting a successful

public relations campaign spearheaded by Mr. Hanneman and the Salt Institute to trivialize any and all scientific data that demonstrates the harmful effects of the amount of salt consumed in a typical modern diet. At the same time these commercial interests publicize and promote questionable scientific “studies”, which were poorly designed and/or interpreted. They even promote “findings” showing that it might even be dangerous to reduce the salt content in processed foods by even a modest amount. A perfect illustration of this occurred on February 5, 2009, when the New York Times published an op-ed article written by Dr. Michael Alderman attacking the recent recommendations made by the New York City Department of Health and Mental Hygiene to reduce the salt content of foods sold in the city by about 40% over the next 10 years. While not identified as such by the New York Times Dr. Alderman is a well-paid spokesman for the Salt Institute. In this op-ed article Dr. Alderman claimed even lowering salt intake by this moderate amount would have adverse metabolic consequences, which he claimed included “...greater resistance to insulin, increased sympathetic nervous system activity and activation of the kidney-based renin-angiotensin system.” Alderman then states: “All three of these effects increase the risk of heart attack and stroke.” He then goes on to discuss some poor quality data to imply a likely unintended consequence of moderately reducing salt intake could be more deaths from CVD. The fact is an overwhelming body of far better designed and controlled studies refute his and the Salt Institute’s opinion concerning the dangers of reducing salt intake in processed foods. Let us now take a closer look at the case against adding a lot of salt to the diet.

Does Dietary Salt Cause HTN in Animals?

It was proven long ago that increasing dietary salt causes a progressive rise in blood pressure (BP) in rats.¹³ Rats fed a diet with only 0.15% salt (dry weight of food) live significantly longer than rats fed any greater amount of dietary salt. Much less than this also was harmful. The average American’s diet contains about 2.5% salt on a dry weight basis. A diet with 0.15% salt would provide an average man consuming 2500 kcal with about 300 mg of sodium/day or about 750 mg of salt per day.

Chimpanzees in the wild consume a diet not dissimilar to that of mankind’s ancient ancestors. Of all animals, chimpanzees are genetically the closest to human beings. A

study of a colony of chimpanzees found that the progressive addition of salt to their largely natural foods diet resulted in a gradual rise in their BP over 20 months. The amount of salt in the chimps diet was similar to that found in a typical American diet today. This significant rise in BP was then completely reversed within 6 months after being returned to a more natural low salt diet.¹⁴

The scientific evidence in animals conclusively shows that simply increasing dietary salt well above what occurs in their natural diets is sufficient to cause increased BP and decrease life expectancy in many different mammalian species. One important finding in animals is that a high-salt diet can cause hypertension, which is not always reversible when dietary salt is reduced to a minimal level.¹⁵ If the same is true for man then it would be wrong to claim that people whose BP does not return to normal on a low-salt diet are not “salt-sensitive”. Of course, animal studies alone cannot prove that excessive dietary salt is the primary cause of essential hypertension (HTN) in humans.

The Case Against Adding Salt to the Human Diet

The most compelling reason for encouraging people to consume less salt is its probable role in the etiology of essential HTN (a.k.a. primary HTN). The vast majority of the scientific evidence indites excess dietary salt as the single most important factor contributing to the development of HTN. For example, when Dr. Malcolm Law was interviewed about a comprehensive review article he had recently published linking excess dietary salt with the development of HTN, he stated, "the effect of universal moderate dietary salt reduction on mortality from stroke and ischaemic heart disease would be substantial - larger, indeed, than could be achieved by fully implementing recommended policy for treating high blood pressure with drugs." Dr. Law also claimed that if salt in processed foods was moderately reduced this could "prevent some 70,000 deaths a year in Britain as well as much disability."¹⁶ This estimate was based on only a 50 mmol (or about 1,150mg of sodium per day) reduction in daily salt intake.¹⁷

According to Dr. Law, "Everyone should reduce their salt intake by at least 3 grams a day." He estimated that even this fairly moderate reduction in salt intake would result in about 250,000 fewer deaths each year in the United States. Dr. Law's predictions were confirmed in a study of older people in Britain whose salt intake was cut by about 80

mmol (1830 mg Sodium). On average, the drop in their BP on the lower salt diet was similar to that achieved by the use of thiazide diuretics.¹⁸ An important question is whether or not greater reductions in salt intake than this would be safe and even more effective for preventing the development of HTN. Indeed, can the rise in BP with age that occurs in all modern industrial societies be prevented or even reversed if dietary salt intake is reduced close to the level that was customary in our ancient ancestors?

Classification and Incidence of Hypertension in America

The Salt Institute points out that only about 1 in four American adults has HTN even though nearly everyone in the US consumes a lot of added dietary salt. The Salt Institute also claims that at most perhaps one-half of those with HTN actually are salt-sensitive and so could benefit from salt restriction. Does this mean that at most 1 in 8 Americans might benefit from reducing dietary salt intake? Clearly the answer is no. The vast majority of Americans and people in other societies where salt is added to the food in large amounts will develop essential HTN in their lifetime and more than half of adult Americans currently have a BP greater than “normal” or 120/80 mmHg.¹⁹ **Table 1** below shows the current classification of BP for adults according to The Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, National High Blood Pressure Education Program, May 15, 2003.

Table 1. Current Medical Classifications of Blood Pressure in Adults:

Classification	Diastolic BP (mmHg)	Systolic BP (mmHg)
Optimal*	No more than 70	No more than 110
Normal	Less than 80	Less than 120
Prehypertension	80-89	120-139
Hypertension:		
Stage 1	90-99	140-159
Stage 2	100 or higher	160 or higher

*Optimal BP is no more than 110/70 and not up to 120/80, if we define optimal BP as the level at which people are least likely to die.

According to the new NHLBI guidelines released May 15, 2003 another 45 million Americans now have what is being termed “prehypertension” (120/80 to 139/89mmHg). This is on top of the 75 million Americans who now have HTN. The people with “prehypertension” were considered to have had “normal” blood pressure or “high normal” blood pressure under the previous guidelines. **Table 2** below shows the incidence of HTN in the U.S. for various age and gender groups. It is clear the incidence of HTN rises dramatically with age. By the age of 60y the majority of men and women in America have developed HTN and the vast majority of these have primary or essential HTN.²⁰

Table 2. The Incidence of Hypertension Among Americans in Different Age Groups

	Men	Men	Women	Men	Women
Age	%BP>140/90	%BP>140/90	%BP>140/90	%BP>150/100	%BP>150/100
18-24	13.3		2.5	2.5	0.6
25-34	17.0		6.3	5.1	2.3
35-44	28.3		25.2	13.1	16.0
45-54	37.6		34.0	20.3	19.5
55-64	52.9		57.2	30.8	36.5

More recently, data from the long running Framingham Heart Study determined the residual lifetime risk of developing HTN in men and women who were free of HTN at age 55y or 65y. As can be seen from **Table 3** below, the risk of developing HTN continues to increase with age into the 8th and 9th decades. About 70% of women age 65-75y have developed HTN, and by age 65 almost 80% of black women have HTN. It is now clear that 90% of the minority of people age 55y or 65y without HTN will likely develop it within the next 20-25 years. In my clinical experience this sad reality comes as quite a wake up call to many Americans who assume that because they have made it to age 55y or 65y without developing HTN they are not salt sensitive and so are somehow immune to the toxic effects of excess salt. This makes no more sense than a 65 year old smoker assuming that just because they don't have emphysema by now that even if they continue to smoke they are now somehow immune. This mass confusion about the risk of developing HTN with age can be laid at the feet of those who buy into the Salt Institutes siren call that only that small minority of those who are hypertensive and “salt sensitive” need be concerned about how much salt they consume.

Table 3. Residual Lifetime Risk of Developing Hypertension According to Baseline Age

Time	Percent of Those Developing HTN over Next 20-25 years			
	Women, Age		Men, Age	
	55y	65y	55y	65y
10 years	52%	64%	56%	72%
15 years	72%	81%	78%	85%
20 years	83%	89%	88%	90%
25 years	91%		93%	

Adapted from Vasani R, et al. *JAMA* 2002;287:1003

Isn't Rising Blood Pressure with Age Physiologically Normal?

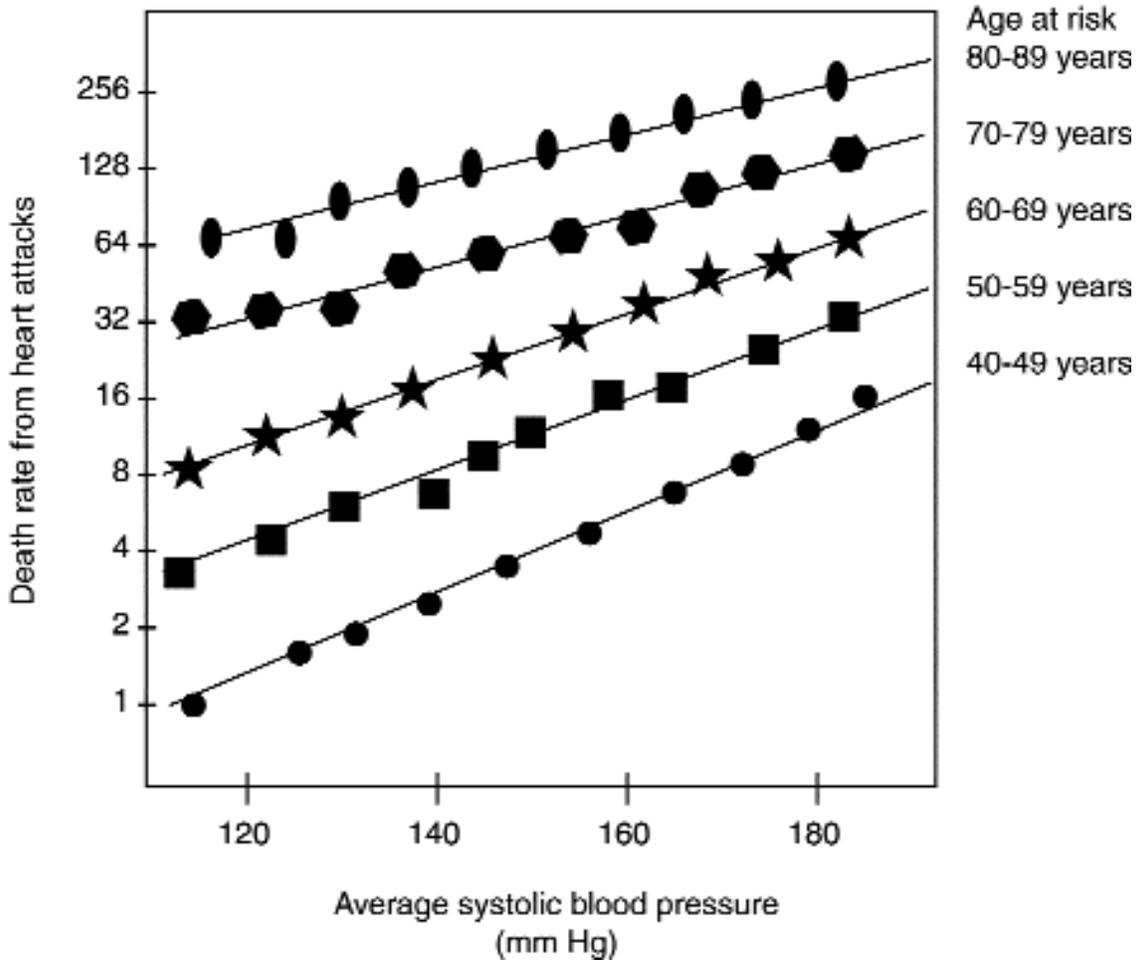
If normal were defined as average or typical in America then rising blood pressure with age would still be considered normal. However, the higher the blood pressure is the greater the risk of morbidity and mortality. It is now apparent that the rise in blood pressure in nearly all Americans as they reach middle and old age is unhealthy. Indeed, many Americans who have been told that blood pressure is “perfectly normal” or “pretty good for someone your age” were in fact being seriously misinformed about the danger that any blood pressure above 115/75mmHg posed to their health. People with blood pressure levels within what had been considered the “normal” BP range and especially those within what had been called the “high normal” BP range are now known to be at a considerably increased risk of dying from CVD compared to someone with an optimal blood pressure.

Any doubt that increasing BP with age is detrimental to health and longevity has been removed by a very large meta-analysis of 61 prospective studies with about a million subjects. This study showed that the risk of developing cardiovascular disease doubles every time the diastolic blood pressure (the low number) increases by 10mmHg and/or the systolic blood pressure increases by 20mmHg. This increased risk of CVD starts at or below a blood pressure of 115/75mmHg.²¹ **Figure 1** below shows that the risk of dying from an ischemic heart disease increases dramatically with increasing BP starting below

120/80mmHg. In addition, their risk of developing dementia, kidney failure or going blind is also significantly higher than those with a BP of 110/70mmHg or less.

Figure 1. Ischemic Heart Disease Mortality in each Decade of Age Versus Usual Blood Pressure.

This figure shows that as your blood pressure increases by 20 points, your risk for having a fatal heart attack doubles.



Adapted from PCS. *Lancet* 2002;360:1903-13

These new NHLBI guidelines should encourage more physicians to refer patients for medical nutritional therapy. In nearly all cases a healthier diet can lower blood pressure from the “pre-hypertensive” range to a far safer level. There is every reason to believe that most people with “prehypertension” who adopt a low-sodium DASH-style diet can return their blood pressure to the optimal range and probably keep it there for the rest of their lives. By contrast, the aggressive use of drugs to lower blood pressure down to the optimal range (<110/70mmHg) often comes with unpleasant and also potential serious

adverse side effects. Therefore using drugs to treat most people with “prehypertension” is often not clinically justifiable.

Those who had a systolic BP in the 130-139mmHg range and/or whose diastolic BP was in the 85-89 range were considered to have “high normal” BP. However, “high normal” BP is been associated with increased thickening and stiffness of the carotid arteries and adverse changes to the heart.^{22 23 24} A follow-up study compared those with a BP of less than 120/80mmHg to those with what had been called “high normal” BP (130/85 to 139/89mmHg). This study found that those with the “high-normal” BP were 2.5 times more likely to develop CVD over the next 12 years than those with a BP below 120/80mmHg.²⁵

Obviously, from the perspective of long-term cardiovascular health, mental health and longevity what had been considered “normal” and “high-normal” BP until fairly recently is now clearly associated with a greatly increased risk of CVD and overall mortality.

The data presented here make it clear that the reason the overall prevalence of HTN in the US is only about 25% is because the vast majority of younger Americans have not yet developed it. Unfortunately, nearly all of them will eventually develop either HTN or at least a “high normal” BP which at least doubles the risk of dying of CVD. This means nearly every American will eventually have a markedly increased risk of dying of a heart attack, stroke or other CVD because of higher BP and the vast majority will develop HTN. The claim that “only about 25% of Americans have HTN” and of those only one-third to one-half are “salt sensitive” lulls people into thinking that if their BP is not now high they have little reason to fear excessive salt intake. Indeed, the latest NHANES data show a significant increase in the percentage of adult Americans who have HTN. This data shows that as we enter the 21st century 29% of Americans age 18y and older have HTN. In 1988 the incidence of HTN was 25%. The increase has been greatest in Americans age 60y and older. In 1988 58% of all Americans age 60y or older had HTN. By 2000, this had increased to 66% of all older Americans (60y+) having HTN. Even more disturbing is that only 31% of all Americans with HTN had their BP under control with drugs and lifestyle changes.²⁶

An important question is whether or not this increase in BP with increasing age is simply a normal part of growing older or whether or not it is an avoidable disease process caused largely by salt toxicity? No one argues that the development of emphysema in smokers is largely the result of aging but many Americans, including many physicians seem to believe that rising BP with age is a normal part of the aging process. This is because there are many Americans who never smoke and they very rarely develop emphysema. However, nearly all Americans consume a very large amount of salt daily so only data from human populations not exposed to salted food can determine what naturally happens to BP as a consequence of the aging process alone.

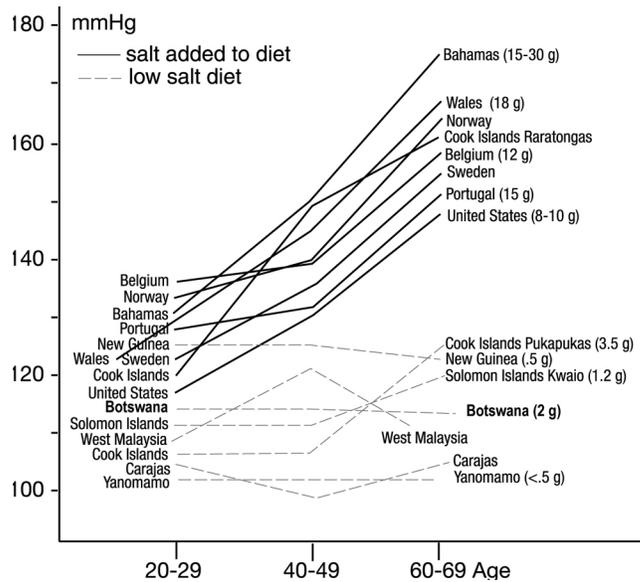
Human Population Studies Show Excessive Dietary Salt Is the Single Most Important Risk Factor for the Development of HTN with Increasing Age

Studies of more than 20 human populations, all of which add little or no salt to their food have found little or no rise in BP with age and a virtual absence of essential HTN.^{27 28} For example, Kenyan farmers who consumed a diet with little or no added salt were found to have virtually no essential HTN.²⁹ However, when young Kenyan men joined the military and began consuming a diet with added salt, their BP started to rise and by the second year it was significantly higher. Kenyan farmers who migrated to urban areas (where the diet is more Westernized) were found to have had their BP rise. The elevation grew greater the longer they consumed the higher salt diet.³⁰

Figure 2 below illustrates how average BP increases with age in every human population ever studied in which a significant amount of salt is added to their food. By contrast, in every human population in which very little or no salt is added to the diet BP changes little with age. This immunity to rising BP with age was the norm for Eskimos whose customary diets are high in fat and protein but quite low in carbohydrate and salt.³¹ The Masai of Africa have long been puzzling to researchers because their diet consists largely of whole milk and blood and yet they rarely die of heart disease. However, the Masai do not eat salt and HTN is uncommon. Research has shown that the Masai's arteries do thicken with atherosclerotic plaques as they grow older despite unusually low serum cholesterol levels. However, despite this artery wall thickening heart attacks are rare apparently because their arteries dilate sufficiently to maintain the flow of blood.³² Essential HTN causes blood vessels to stiffen with age. Could it be that the Masai avoid heart attacks largely because their blood vessels do not stiffen with age simply because

they do not add salt to their food?

Figure 2. The Relationship between Systolic BP and Age in Various Populations:



Adapted from MacGregor. *Hypertension* 1985;7:628

Immunity to HTN and rising BP with age is also common in population groups that consume high-carbohydrate diets. For example, both the New Guinea Highlanders and Tarahumara Indians of Mexico consume nearly vegetarian diets. Both have diets very high in carbohydrate but very low in salt and HTN and CVD are uncommon in both the Tarahumara and New Guinea Highlanders.^{33 34} This shows that there is a wide range of climates, genetic diversity as well as extremely varied diets among human population groups in which BP has little or no tendency to rise with age. This makes it is hard to imagine what factor other than the lack of added salt they all have in common that could be protecting them from essential HTN. The very large Intersalt study examined the relationship between sodium and potassium excretion in the urine and BP. This massive study also found that BP rose with age in every human population studied except of four groups that added very little or no salt to their food.³⁵

It is a fact that in every human population that adds a significant amount of salt to their food, BP rises inexorably with age. It is also a fact that in no human population that consumes about 40 mmol of salt day or less is there any consistent elevation of BP over time. Therefore, rising BP with age and essential HTN cannot simply be a normal or

natural consequence of aging process itself. If it were then one would expect to find it in all older people regardless of diet. This epidemiological data strongly suggest that it is primarily excessive dietary salt that plays the primary role in the development of HTN. Dietary salt, well in excess of what the human body evolved with apparently results in the very high lifetime incidence (90%+) of HTN in the US and all other societies that add appreciable amounts of salt to their food. The Salt Institute, of course, has claimed that the results of all these studies don't really mean that it is dietary salt that is causing BP to rise in all these modern societies. They claim it could be all the noise, stress, weight gain, genes, or something else that we haven't yet figured out that accounts for this connection between added dietary salt and essential HTN. The Salt Institute has claimed that even moderate reductions in dietary salt could be dangerous. The Salt Institute also maintains that without iodized salt we could have an outbreak of goiter in the US. As shall be shown these claims are specious. It has been shown that virtually all Americans easily obtain sufficient dietary iodine even if they never consume iodized salt.³⁶

A recent study of more than 11 thousand European men and women ages 45-79 examined the association between the amount of sodium in their urine and a genetic trait known to increase the risk of developing HTN. The results showed that this genetic trait, which leads to higher levels of angiotensin II in their blood, was associated with significantly higher BP in those who ate the most salt. However, in those who had the least sodium in the urine having this genetic trait did not appear to increase BP. The authors conclude: "Genotype effects in populations at low exposure to sodium are not likely to be seen." {35} This study also found a highly significant association between sodium intake and blood pressure for all genotypes.

Genetic factors no doubt make some people more susceptible to the toxic effects of excess dietary salt and so more likely to develop more serious HTN earlier in life. However, it is increasingly clear that a dramatic reduction in the amount of salt added to foods by individuals and particularly the food industry would largely eliminate a disease that is a major risk factor for heart attacks, heart and kidney failure and the #1 risk factor for stroke.³⁷

Could Stress, Noise, and Inactivity Be More Important than Dietary Salt in the Development of HTN?

There is abundant anecdotal evidence that psychological stress can raise BP in humans. No one doubts that BP goes up in response to the release of stress hormones. However, exercise also causes a marked rise in BP, but no one claims that regular exercise causes HTN. Persistent HTN has been induced in mice by exposing them to a prolonged period of very severe psychosocial stress.³⁸ However, standard laboratory diets used for experimental animals contain a generous amount of salt.³⁹ Whether stress alone with a really low-salt diet can cause a severe or sustained elevation in BP in animals is not known. However, studies of human population groups that experience on going tribal warfare but consume very-low salt diets such as the Yanomamo Indians of Brazilian rain forest or the Asaro tribes of the New Guinea Highlands have very low BP throughout life despite what must be a very stressful environment.

Claims that a stressful environment with a lot of noise, rather than added dietary salt, is primarily responsible for the rise in BP with age in modern industrial societies are not supported by solid scientific research. The Qash' qai pastoral nomads in Iran experience a marked rise in BP with age despite a fairly peaceful and quiet environment and a lifestyle that had changed little in the past 500 years.⁴⁰ However, unlike most other unacculturated populations the Qash'qai nomads do add salt to their food. It also conflicts with observational studies in the Solomon Islands. The fairly unacculturated Lau tribe, who cook their food in seawater, had a high incidence of HTN despite a fairly tranquil existence on a South Pacific island. However, on a neighboring Solomon Island there is another tribe with a very similar diet and lifestyle with the exception that they use fresh water to cook their food instead of seawater. Unlike the Lau tribe, these Solomon Islanders do not develop HTN or experience a significant rise in BP with age.⁴¹ Therefore, people who live relatively quite and peaceful lives still develop essential HTN if their diet is high in salt. The results of these studies and observations conflict with the claim that noise and stress in the modern world are the primary cause essential HTN.

This does not mean that reducing psychological stress cannot help lower BP in at least some individuals. People with HTN and suffering from a high level of social stress should be referred for counseling. If nothing else, stressed out people are unlikely to

comply with the additional time and effort required to adapt to a low-salt diet.

Nevertheless, there is virtual absence of essential HTN in every human population that adds little or no salt to their food. This observation coupled with the nearly universal development of HTN in all human populations that add salt to their food makes it hard to imagine that psychosocial stress could be nearly as important as excessive salt intake in the promotion of HTN.

Aside from salt the only environmental factor that correlates most closely to the development of HTN in salt versus no salt added cultures is activity. With only a few exceptions most of the no-salt cultures have a much higher activity level than seen in more modern societies where high-salt intake and inactivity are the norm. A twelve-year follow up study of several thousand people with normal BP found that the risk of developing HTN was inversely related to physical fitness level.⁴² A study of both normotensive and hypertensive sedentary subjects assigned them to one of 4 different activity levels. This study found that riding an exercise bike for 30 to 40 minutes at 60 to 70% of VO₂max for either 3 or 5 days a week for four weeks resulted in a average 10 mmHg fall in BP.⁴³ By contrast the subjects assigned to the two low activity groups experienced no significant change in their BP. The authors believed the drop in sympathetic nervous system activity with exercise accounted for the lower BP in the two exercise groups by reducing peripheral resistance. Increased peripheral resistance is characteristic of essential HTN.

Aerobic exercise appears to be preferable to resistance training in those with HTN. This is because there can be a very large increase in BP when performing resistance training such as weight lifting and isometric exercise. For example, BP recordings of 450/300 mmHg have been recorded in normotensive power lifters doing a maximal bench press. Some researchers have expressed concerns that it may be dangerous for people with HTN to engage in heavy resistance training.⁴⁴ So while it may be prudent to discourage patients with HTN to do heavy resistance training, the results of most studies suggest that regular aerobic exercise may help prevent HTN and also may help lower BP in HTN patients. But is aerobic exercise more effective than salt restriction for lowering BP? And could greater activity be the real reason that the no-salt human population groups avoid the rise in BP so typical in modern societies?

A study that compared the impact of moderate exercise or moderate salt restriction in 35 postmenopausal women with mild to moderate HTN suggests salt restriction is far more important. In this study the salt restriction target was 2400 mg sodium/day. It was achieved with dietary advice to refrain from using the saltshaker and avoid processed foods high in salt. This dietary advice was given to outpatient subjects who bought their own food (unlike DASH-Sodium) trial. The average drop in systolic BP over the 3-month period was 16 mmHg. By contrast, the group that walked 30 minutes every other day experienced only of 5 mmHg average drop in systolic BP.⁴⁵ The researchers "surprise" that even moderate salt restriction worked so well is an indication of how successful the Salt Institute's PR campaign has been to play down the excellent therapeutic effects of salt restriction in older Americans with HTN. This study shows that moderate salt restriction is far more effective than increased activity for lowering BP in HTN subjects. Japanese fisherman, Qash' gai nomads, Chinese farmers and other high-salt consuming but active human population groups experience a dramatic rise in BP with age also conflicts with the idea that inactivity rather than excessive salt intake is largely responsible for the very low incidence of HTN in less modern societies.

Could the Rise in Body Weight with Age in Modern Societies Be More Important than Excess Salt Intake in the Development of HTN?

In the US and most Westernized populations, body weight increases with age until at least the sixth decade of life. Many physicians believe that the rise in BP in Americans with age is due primarily to weight gain rather than excess salt intake. While there is little debate about whether or not increased body weight contributes to rising BP with age a claim that weight gain is the primary cause of rising BP with age conflict with studies of the Japanese. The Japanese diet is considerably higher in salt than that of Americans but until recently their body weight remained fairly stable throughout adult life and obesity was very uncommon in Japan. Nevertheless, the Japanese experienced an even greater rise in BP with age than did Americans back in the 1950s and 1960s despite the fact they the Japanese gained little or no weight with age.⁴⁶

By contrast, many Kuna Indians of Panama who were moderately obese but ate a diet that was low in salt have a very low incidence of HTN. In this population less than 1% of adults had HTN and BP did not rise significantly with age.⁴⁷ Today, many Kuna Indians

have adopted a more Westernized diet higher in salt and now experience a significant rise in BP with age. However, among Kuna Indians the incidence of HTN still remains lower than that seen in populations who have consumed a high-salt diet throughout life.⁴⁸

The rise in BP with increased body weight may be less harmful than that caused by excess salt intake. To understand why this may be the case it is important to understand how weight gain and salt intake impact BP. An analogy may help. There are two ways to increase the pressure of water in a garden hose pushing out against the walls of the hose. One way would be to turn up the spigot, which would increase the flow of water through the hose and also increase the pressure of the water pushing out against the inner walls of the hose. A second way to increase the pressure pushing out against the walls of the hose would be to squeeze the end of the hose. This would increase the pressure inside the hose all the way back to the spigot. Obesity raises BP in part due to an increase in cardiac output because the heart must pump a greater volume of blood to circulate in a larger body. In the long run, dietary salt creates a rise in BP primarily by causing constriction of the arterioles. This peripheral resistance is analogous to the increased pressure in the garden hose that results from placing a thumb over the end of the hose. Essential HTN results primarily from increased resistance to the flow of blood in the arterioles. This means that if an obese person and a lean person have the same BP, the lean person will generally have more of this peripheral resistance whereas the higher BP in an obese person would result more from an increased cardiac output. Since increased peripheral resistance may reduce the flow of blood at the capillary level it will restrict the flow of nutrients and oxygen to tissues and therefore, may be more damaging to the body. This may be one reason why lean HTN subjects were found to have a higher risk of dying from CVD than did obese subjects with the same BP.⁴⁹

Gaining weight, especially intra-abdominal fat leads to insulin resistance (IR) and increased serum insulin levels.⁵⁰ Higher insulin levels appear to make it more difficult for the human kidney to get rid of excess salt.⁵¹ IR is also associated with a reduced production of nitric oxide (NO) by endothelial cells that line artery walls.⁵² NO is a very powerful vasodilator. Less NO means that arteries and arterioles are less able to dilate. This would tend to exaggerate the action of any vaso-constrictive agents in the body such as angiotensin II and norepinephrine. Excessive salt intake creates electrolyte imbalances (i.e., increased intracellular calcium and sodium) that appear to contribute to both IR and

HTN.⁵³ This leads to excessive vasoconstriction, which is characteristic of the peripheral resistance typically seen in people with essential HTN. Regular aerobic exercise has been shown to reduce IR. It also lowers BP in most HTN patients.⁵⁴ This should not be surprising since aerobic exercise promotes weight loss, reduces insulin resistance and lowers serum insulin levels. In addition, with sweating some excess salt is excreted by the sweat glands, which reduces the nutritional stress on the kidney to rid the body of excessive salt.

Excess body weight has long been linked to higher BP and weight loss certainly has been shown to lower BP in most obese subjects.⁵⁵ However, given the difficulty in maintaining long-term weight loss very little data is available about the impact of long-term weight control for preventing and treating HTN. The results of an ongoing clinical trial in Sweden are likely to be discouraging for those who believe that weight control is more important than reducing dietary salt in the long run. In this study, a group of 346 obese patients who underwent gastric surgery to lose weight were followed for 8 years and compared to 346 similar control subjects, who did not have the surgery. After 2y those undergoing surgery lost 23% of their initial weight while the controls maintained their initial weight. After 2 years the incidence of HTN was 2.5 times higher in the controls than in those subjects who had the stomach surgery and had lost and kept off weight. However, after 8 years of follow up, despite the fact that body weight continued to be about 20 kg less in the surgery group the incidence of HTN in the two groups was now equal. The authors concluded, “A maintained weight reduction of 16% strongly counteracted the development of diabetes over 8 years but showed no long-term effect on the incidence of hypertension.”⁵⁶ The results of this study clearly suggest that those wishing to control or prevent HTN simply by losing weight and keeping it off are likely to be disappointed.

The point here is not that obesity has no adverse effects on BP or health. Clearly it does. Obesity, particularly central adiposity, is associated with the development of HTN.^{57 58} Both salt restriction and weight loss have been shown to independently lower BP.⁵⁹ When obese native Hawaiians were placed on a very-low- fat, low-sodium diet, similar to what their ancestors consumed, they lost 17lbs and reduced both SBP and DBP by more than 10% in just 21 days.⁶⁰ Of course, they also ate less salt on their native diet and more potassium-rich fruits and vegetables. Pacific Islander peoples were shown to be immune

to the development of essential HTN as long as they avoid added salt. Therefore, the long-term success of the “native Hawaiian diet” is likely to prove far more successful than did gastric surgery for reducing the incidence of HTN in obese patients.

Too Much Salt Appears to Be The #1 Cause of Essential HTN

Even 100 years ago there was a growing suspicion that excessive dietary salt intake promotes HTN.⁶¹ Double blind controlled trials have proven that raising salt intake alone is sufficient to increase BP in people with normal BP in just a few weeks.^{62 63 64} Of course, there are many environmental factors that can influence BP at least as much as excess dietary salt in the short-term. However, no other environmental factor can explain the near universal rise in BP with age that occurs in every human population ever studied that adds a significant amount of salt to their food. Nor can any other environmental factor explain the virtual absence of essential HTN in every human population ever studied that adds very little or no salt to their food.

We have seen that excessive salt intake is the only viable explanation for the rise in BP with age that occurs in all human populations that add significant amounts of salt to their food. Obesity and excessive alcohol consumption can certainly raise BP in the short-term, but their effect on BP is usually more modest and shorter acting compared to the impact of excessive salt intake. Other factors, including stress and a noisy environment, may temporarily raise BP to be sure but there is little reason to believe that they play a major role in the rise in BP with age seen in every human population that customarily adds salt to their food. Such a theory would beg the question as to why stress does not also cause HTN in human population groups that add little or no salt to their food.

Epidemiological studies clearly indicate that a dietary salt of no more than about 30 to 40 mEq (or about 700 to 900 mg of sodium from salt) is associated with a virtual absence of essential HTN.⁶⁵ Indeed, when comparing human populations that add salt to their food to those where little or no salt is added, the average difference in systolic BP for people above age 60y is about 50 mmHg.⁶⁶ See also **Figure 2**. If excess salt is primarily responsible for the rise in BP with age in the US could reducing salt not only prevent but also even reverse the trend of increasing BP with age?

Short-term Clinical Trials Miss Long-Term Effects

The huge difference (about 50mmHg or more) in the average systolic BP between populations consuming very little salt compared to populations consuming a typical high-salt modern diet generally takes 50 to 60 years or more to appear. A difference in systolic of BP 50 mmHg is much greater than the very modest drop in systolic BP typically seen in most short-term clinical trials. Even in those few short-term trials in which achieve very large differences in dietary salt intake few patients experience a drop in systolic BP of 50 mmHg. In normotensive subjects the fall in systolic BP with short-term moderate salt restriction is typically even more modest. The authors of two highly publicized reviews using a meta-analysis of just such short-term studies concluded that the very small drops in BP observed in normotensive persons on reduced sodium diets "does not justify a general recommendation for reduced sodium intake".⁶⁷ The Salt Institute, as you may have guessed, made sure the media focused on this conclusion. But would we expect young and middle-aged smokers who reduced their smoking for a two or three weeks to show more than a very modest improvement in their lung function? Obviously not so why should we expect more than a modest improvement in BP when salt intake is only moderately reduced?

The second and similar meta-analysis of short-term clinical trials of reduced sodium intake funded by an "unrestricted grant" from the Campbell Soup Company (a major contributor to the Salt Institute) also concluded that the evidence does not support current recommendations for sodium restriction in people without HTN.⁶⁸ We would expect a few weeks on a moderately low fat and cholesterol diet to cause measurable changes in the amount of atherosclerotic plaque in people's arteries? Certainly no one would argue that the fact that the size of plaques hardly changed at all in a few weeks is proof that people should not be told to cut back on saturated fat and cholesterol. A cynic might suggest that it was not just a coincidence that these two questionable review articles both appeared in the Journal of the American Medical Association. This journal carries many ads for anti-HTN pharmaceutical agents. Pharmaceutical companies obviously benefit from treating HTN with drugs rather than an "ineffective" salt-restricted diet.

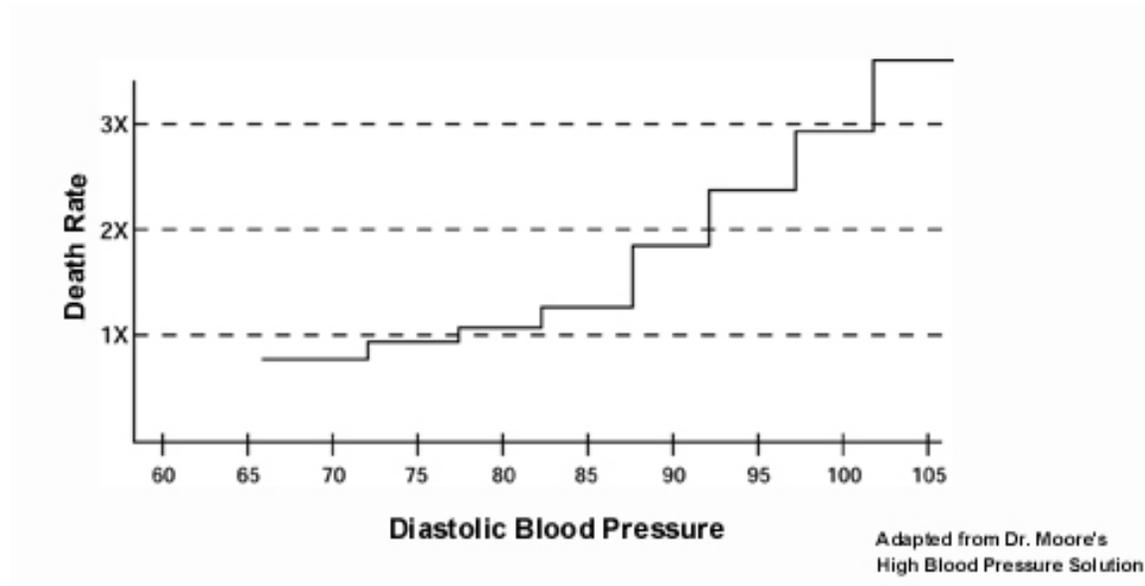
However, despite these shortsighted and limited reviews, the bulk of the scientific evidence suggests that it takes many years to see the full BP raising effects of excess

dietary salt. It is likely that it takes many years for excessive dietary salt to do its damage to the cardiovascular system. Some of these adverse effects may be only partially reversible and/or may take many years on a very low-salt diet to reverse. Of course, most clinical trials do not reduce dietary sodium to less than 30 mmol or even 70 mmol per day. If the same criteria were used to set an RDI and a UL for dietary salt as are used for other nutrients, the salt RDI would be about 30 mmol (or 690 mg of sodium) and the UL would be about 70 mmol (or 1600 mg of sodium). The fact is that neither of these highly publicized reviews looked at the big picture. In a sense, these reviewers could not see the forest through the trees. Using their clinical logic we would conclude that there is no need to tell people with normal lung function to quit smoking and no need to tell those with normal liver function to stop heavy alcohol consumption. Let us now examine some of the evidence these reviewers overlooked that strongly suggest that waiting for HTN to develop before reducing salt intake may be courting disaster.

If BP Is "Normal" Why Not Wait for Hypertension to Develop before Reducing Excessive Salt intake?

The Salt Institute would have people believe that if their doctor hasn't told them they have HTN then the amount of dietary salt they consume is of no consequence. Most people and many clinicians subscribe to this position and assume the only way too much salt can harm the body is by causing HTN. There are three problems with this "theory". First, the risk of dying from an increase in BP does not begin with the diagnosis of HTN and a BP > 140/90 mmHg. **Figure 1** shows that the risk of dying even in people who are in their 70s and 80s increases dramatically with rising systolic BP. **Figure 3** below shows the risk of dying also goes up as the diastolic BP rises above 70 mmHg. It has been estimated that about one-third of all cardiovascular disease resulting from increased BP occurs within the "normal" range of BP. ⁶⁹

Figure 3 - The risk of premature death with increasing diastolic blood pressure:



There is evidence that the relationship between BP and mortality is J-shaped, particularly in older people who take drugs to lower their BP. People with the lowest and highest BP are more likely to die than people with average BP. This is particularly true in older people. But one should not leap to the conclusion that lower BP increases the risk of dying. It depends why it is low. If you slit your wrist you will soon have very low BP and soon after that will be dead. In older people who have heart attacks BP often falls. This is because the heart is a weaker pump and cannot pump blood with the same force as before the heart attack. Chronic poorly controlled HTN can lead to congestive heart failure. Again the weaker, less efficient heart pumps blood with less force and BP is lower. Cancer or AIDS can produce a lot of weight loss. BP usually falls with the loss of body weight. Obviously having a lower BP as a result of serious cardiovascular disease or weight loss will be associated with an increased risk of death. By contrast, a low BP that results from a healthy diet and lifestyle is associated with an increased life expectancy. It should be clear that low BP can result from disease and if this is the case it is the disease not the low BP that is causing the increase in mortality.

Hypertension, Like Atherosclerosis, May Begin Early in Life

A second reason not to wait for HTN to develop is that HTN, like atherosclerosis, may begin early in life and may do serious harm before any symptoms show up. Pregnant rats fed a higher salt diet have offspring with higher BP.⁷⁰ Human infants, unlike adults, do not appear to have a taste for salt.⁷¹ Nevertheless, baby food manufacturers had for many years routinely added salt to baby foods (to please their mother's palate). Human milk, despite the high-salt intake of most mothers is surprisingly low in sodium (only 39 mg/cup) having only about 1/3 the sodium content of cow's milk (122 mg/cup).

The results from a double-blind controlled study of newborn human infants suggest that BP responds to the amount of salt in the diet. Half the infants were fed a formula diet containing the amount of sodium (and potassium) found in cow's milk, the other half were fed a similar formula with the amount of sodium and potassium reduced to the level found in human breast milk. The researchers found that after 6 months, the average BP of the infants fed the higher sodium formula was already significantly higher than that of those fed the lower sodium formula.⁷² The rise in BP in the infants fed the higher sodium formula did not occur in the first few weeks. However, the BP increased more for babies on the higher salt formula and gradually became significantly higher than that of infants on the more natural level of salt after several months. This type of evidence has led pediatricians to conclude that the requirement for sodium in human infants is less than 9 mEq/day or no more than about 200 mg daily.⁷³

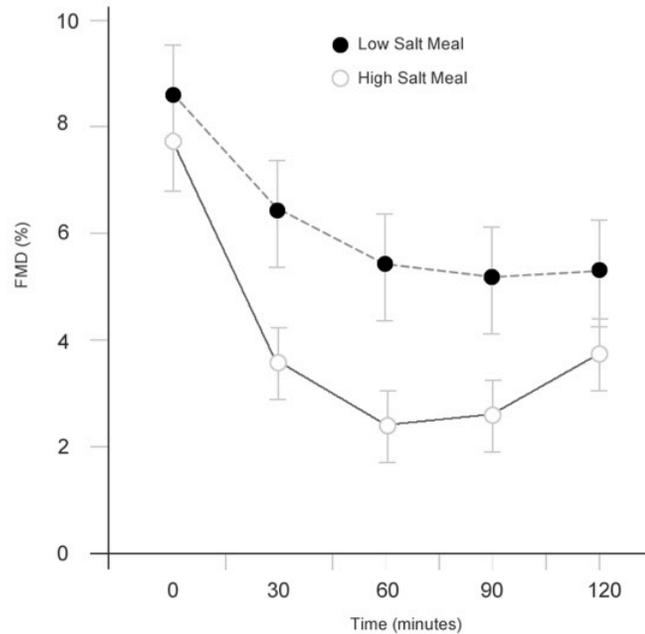
The Harm from Excess Salt May Not Be Due Entirely To Increased BP

A third reason to be concerned about excessive dietary salt, even in people with "normal" BP, is that HTN may simply be a symptom of more serious underlying adverse biochemical and physiological changes caused by salt toxicity. Rats, like people vary in their susceptibility to develop HTN in response to a high-salt diet. Indeed, rats have been bred to be especially "salt sensitive". Other strains of rats (like some people) are more resistant to the BP raising effects of a high-salt diet. A study compared the effects of a moderate (1%) or high (8%) salt diet in genetically "salt sensitive" and "normotensive" rats. On the high-salt diet, even the "normotensive" rats showed widespread fibrosis in their hearts, kidneys and arterioles compared to the "salt sensitive" rats that were fed the moderate salt diet. This was true even though the BP of the "normotensive" rats was

much lower than the “salt-sensitive” rats. The authors "suggest that excessive salt intake may be an important direct pathogenic factor for cardiovascular disease".⁷⁴ These results suggest that an excessive salt intake promotes the overgrowth of fibrous tissue in the arteries, arterioles and other tissues and this fibrosis gradually over time makes the blood vessels increasingly stiff and eventually makes them more resistant to the flow of blood. In "salt-resistant" individuals this damage may have to become more severe before BP rises to the point where HTN is diagnosed. Recent data suggests that people like rats experience significant pathological changes to their cardiovascular system even though their blood pressure remains within the normal range.

A study by Australian researchers examined the impact of feeding a group of 29 overweight and obese subjects a typical high-salt Western diet or a diet with less salt but the same amount of saturated fat, potassium, and other dietary variables for two weeks. Urinary sodium was measured to determine compliance with the two diets. On the normal high-salt diet 24-hour sodium excretion was about 3,600mg/day compared to only about 1475mg/day on the low-salt diet. The subjects had normal blood pressure and all followed both diets for two weeks in a crossover design. The researchers assessed flow-mediated dilation (FMD) of the brachial artery on the high salt and low salt diets. Impaired FMD is generally due to dysfunction of the endothelial cells that line the arteries and is believed to be involved in the pathology of cardiovascular disease. The results of this study showed FMD was reduced by 45% on the high-salt diet compared to the low-salt diet.⁷⁵ More recently this same group of research fed a group of normotensive subjects who had fasted overnight either tomato soup with about 1500mg of sodium or the same soup with only 115mg of sodium. They found that FMD was reduced significantly more at 30 minutes and 60 minutes after the high-salt soup meal than with the no salt added soup meal.⁷⁶ Importantly, both of these studies found that the impaired FMD caused by increased salt intake was independent of its effects on blood pressure. The authors conclude, “These findings suggest additional cardio-protective effects of salt reduction beyond blood pressure reduction.”

A Single High-Salt Meal Impairs Flow-Mediated Dilatation



The American Journal of Clinical Nutrition

Source: Dickinson KM, Clifton PM, Keogh JB. Endothelial function is impaired after a high-salt meal in healthy subjects. *Am J Clin Nutr* 2011;93:500-5.

Excessive salt intake appears to interfere with normal artery function and impair blood flow long before it leads to the development of hypertension. Clearly if one wants to avoid damage to their cardiovascular system waiting for the development of hypertension before restricting salt intake is ill advised.

Why Do Low-Salt Diets Often Fail to Sufficiently Lower Blood Pressure?

Some doctors are led to believe salt is not a major cause of HTN based on their clinical experience. When doctors encourage their patients to reduce dietary salt they often find that most of their patients experience little or no drop in BP. Of course, most patients who are told by their doctors to reduce salt intake do not actually reduce their salt intake very much because many high-salt foods do not taste particularly salty and they are not given sufficient dietary counseling to achieve a significant drop in salt intake. For example, bread has about double, and corn flakes about triple the salt content of potato chips (on a Na/kcal basis) but most people would guess that the chips are higher in salt than the bread

or cereal. In addition, since no more 15 to 20% of the salt in a typical patient's diet is added at home, even patients who try hard to comply often are not very successful in cutting salt intake by even 30%. This type of modest reduction in salt intake alone is generally not enough to have much effect on BP over a period of just a few weeks.

However, some primary HTN patients will not see their BP return to normal even if a low-salt diet is achieved and in a few BP does not fall at all. Nevertheless, it would be a mistake to assume that just because salt reduction doesn't always reverse HTN that it didn't cause it in the first place. After all, do doctors assume smoking didn't cause emphysema because the emphysema doesn't go away when patients stop smoking? It would also be a mistake to assume that if BP does not fall that a low sodium (high potassium) diet is of no clinical value. Studies in rats have shown that some, which develop high BP while consuming a high-salt diet, will continue to have high BP even after their salt intake is markedly reduced. Their littermates, which were only fed the low-salt diet for their entire lives experienced no significant change in their BP with age.⁷⁷ At least in animals it is clear that salt toxicity can result in vascular changes that are not always completely reversible when the excessive salt intake is stopped. If the same were true in man then waiting for people to be diagnosed with HTN before recommending they cut back on salt is irresponsible public health policy.

Even though HTN often takes decades to develop, many clinicians expect to see complete reversal of HTN in a week or two on a low-salt diet. In fact, in patients with severe or malignant HTN, restricting salt to very low levels has been shown to cause a substantial fall in BP in most patients.^{78 79} At the Pritikin Longevity Center, it was shown that even more modest restriction of sodium intake to less than 1600 mg per day lowers BP in most HTN people within four weeks if other diet and lifestyle changes are also made.⁸⁰ There are other reasons the Pritikin Program lowers BP besides the low salt content of the diet and these will be discussed later on. Back in the 1940s Walter Kempner was the first to show that a low-fat, very-low-salt diet was the only successful treatment to reverse very severe malignant HTN.⁸¹ The Kempner rice diet had less than 15 mmol of salt (or 350 mg of sodium). While both the low-salt, very-low-fat, near vegetarian approaches used by Kempner and later Pritikin and others were extremely effective at lowering high BP both are rarely utilized today in part because compliance is so difficult because dietary salt is so ubiquitous in commonly consumed American foods. It should be noted that the BP-

lowering impact of these diets depends primarily on their low-salt content but other dietary factors and exercise probably help as well. It should be noted that the success of the Kempner rice diet does not depend on either its low fat or its low animal protein content.⁸²

A study of two rural communities in Portugal examined the impact of a community intervention plan of reducing salt intake on blood pressure. The average fall in sodium/salt intake in the “low-salt” community was about 2000mg of sodium in the first year and this fell to only about 800mg less sodium daily in the second year compared to the control community that continued their normal (very high) salt intake. Despite this rather modest reduction in average salt intake in the intervention community the average systolic BP was 13.3mmHg lower and the average diastolic BP 6.1mmHg lower than in the control community after two years. Blood pressure fell significantly on average in men and women and young and old people. Blood pressure fell much more on average in those who were initially hypertensive but also dropped significantly in those who had “normal” BP.⁸³

Unfortunately, the long-term effects of a low-salt diet have received little attention. After an initial drop in BP, which can be fairly large in some individuals, the decline in BP often continues for many months or years, provided the salt restriction is maintained. For example, one group of researchers found that BP was still trending downwards after two years on a diet with about 70 mEq (1600 mg of sodium).⁸⁴ Given that the BP raising effects of excessive dietary salt take many years to develop it is surprising that most clinicians expect to see the full impact of a low-salt diet within just a few weeks. In my clinical experience it may take as long as 5 to 7 years to see the full BP lowering impact of a low-salt diet.

Excess Salt May Be the Real Culprit: Hypertension May Just Be a Symptom

There is growing evidence that HTN is merely a symptom of an underlying nutritional imbalance caused largely by an excessive intake of salt. Salt intake was found to correlate more closely with left ventricular hypertrophy (LVH), cerebrovascular disease, microproteinuria (an early sign of kidney disease), and reduced arterial compliance than BP. A recent review found that "there is clinical and experimental evidence... that salt intake directly affects hypertensive renal disease, cerebrovascular disease, and

compliance of the large arteries. The close and partially independent correlation between salt intake and hypertensive target organ disease suggests dietary sodium to be a direct perpetrator of cardiovascular disease."⁸⁵ Simply put HTN is just one of many symptoms that can result from the damaging effects of chronic salt toxicity.

It was long believed that the only way salt could cause disease was by raising BP. So as long as BP remained in the normal range one's salt intake was not excessive and was doing no real harm to the body. Now it appears that chronic salt toxicity damages the heart, brain, kidneys and arteries directly and HTN is a very common symptom of that damage, which physicians can easily measure is elevated BP. However, the absence of one symptom of a disease does not mean that the disease process is absent. It may only mean that it is not being diagnosed simply because physicians mistake the symptom (high BP) for the underlying disease (salt toxicity) and assume incorrectly that as long as this symptom is not present there is no disease. This would be like assuming a patient has no atherosclerosis if they have not had a heart attack, stroke or developed angina. This also means that drugs designed primarily to treat a symptom (in this case HTN) may be far less effective at correcting the real cause of this disease than a healthier diet and lifestyle. Essential HTN increasingly appears to be due primarily to a nutritional imbalance of essential electrolytes. Eliminating the cause of the disease is always preferable to symptom relief but when physicians fail to recognize what is causing the disease process they end up treating the symptoms.

The greater the dietary consumption of salt is, the greater the cardiac preload to the left ventricle. This was shown to be the case even in normotensive subjects.⁸⁶ Over time this increased workload on the heart may lead to left ventricular hypertrophy (LVH). Other researchers examining people with both HTN and LVH and were able to "identify dietary salt as a strong determinant of cardiac structural adaptation to a persistent increase in arterial pressure." These authors went on to say that "a high salt intake might aggravate and, conversely, dietary salt restriction might prevent (or at least mitigate) the development of left ventricular hypertrophy in patients with essential hypertension."⁸⁷ Researchers put 91 men and women with HTN and LVH on either a moderately low-sodium diet (the goal was 70 mmol Na/d or less) or a "normal"-sodium diet for one year. They found a significant reduction in LVH on the low-sodium diet and concluded, "that long-term non-pharmacological treatment with moderate sodium restriction decreases

LVH".⁸⁸ Since the sodium restriction achieved was closer to 2200 to 2500 mg sodium/day, it is likely that an even more dramatic reduction in LVH would have occurred if their therapeutic goal for salt intake had actually been achieved.

The difference between the systolic and diastolic BP is known as the pulse pressure (PP). In all human populations that add significant amount of salt to their foods the PP tends to increase gradually with age. This process often accelerates after reaching age 50y. Over 2500 years ago a Chinese doctor described a hardening of the pulses as a symptom of too much salt in the diet. Today, an increased PP is an established risk factor for congestive heart failure (CHF).⁸⁹ CHF is the only serious cardiovascular disease that has been increasing in America and is currently the leading hospital diagnosis in those over the age of 65y.⁹⁰ It is characterized by salt and water retention that can lead to swollen ankles and fluid in the lungs, which causes congestion. Essential HTN gets its name from the fact that the human kidney's capacity to excrete salt and water increases with increasing BP. In order to excrete the high-salt intake of the typical modern diet and prevent chronic salt and water retention it is "essential" that BP be high enough for the kidneys to remove it in a timely manner. A failing heart cannot pump blood with sufficient force through the vascular system to allow the kidney to excrete all the salt and fluid it needs to. Not surprisingly then, one of the most important dietary precautions for those with failing hearts is to keep salt intake very low.

The rise in PP with age does not occur in any human population that adds little or no salt to their food. Therefore, it seems likely that increasing arterial stiffness with age is not a natural part of the aging process. Indeed, when normotensive people were maintained on a low-salt diet (average of 44 mmol sodium/day or about 1000 mg of sodium/day) for an average 24.8 months they experienced a 22% reduction in arterial stiffness. In contrast to this experimental group, there was no change in arterial distensibility in age-matched control group, which maintained their normal diet. The authors of this study conclude, "This is prima facie evidence that reduced salt intake has a beneficial effect in improving distensibility of the central aorta and large peripheral arteries, which is independent of its antihypertensive action".⁹¹ As was noted earlier, this observation is consistent with what happens in the Masai in Africa. They consume a diet high in saturated fat and cholesterol but low in salt. They rarely suffer heart attacks in part because their large blood vessels continue to dilate in response to the narrowing caused by increased artery wall thickness.

HTN has long been recognized as a major risk factor for CHD but dyslipidemia and atherosclerosis may pose far less danger if dietary salt intake remains low throughout life. More research is needed to determine if excessive salt intake is perhaps a necessary component in restricting blood flow on a diet high in saturated fat and cholesterol.

The scientific evidence continues to mount implicating a diet high in salt with adverse changes in the cardiovascular system that lead LVH and CHF, kidney damage, and arterial and arteriole damage. As seen with salt induced damage to major arteries, the damage to the arterioles results in a reduced capacity for them to dilate and this can lead to essential HTN. But even in the absence of HTN, a high-salt and low potassium diet appears to increase risk of CHD and both hemorrhagic and ischemic strokes. Increasingly, HTN appears to be more a symptom of salt toxicity than the direct cause of these associated serious medical problems.

Salt Toxicity Creates an Imbalance of Electrolytes in the Body

Other electrolytes besides sodium affect BP and vascular disease. Not only did the human Paleolithic diet have only about 1/5 to 1/10 as much salt as the typical American diet today, but it was also about 2-3 times higher in potassium, calcium and magnesium.⁹² The ratio of potassium to sodium was probably at least 10 to 1 in our ancient ancestor's diet but is only about 0.6 to 1 in the typical American diet today. This means the ratio of these two cations was at least 15 times higher as humans evolved as is the norm for Americans today. In rats, it has been shown that increasing potassium in the diet reduces deaths, cerebrovascular disease and increases life expectancy independently of its effects on BP.⁹³ The DASH diet works to lower blood in part because it is higher in calcium, potassium and magnesium.

A study of 111 vegetarians age 55y and older in Hong Kong found that those who ate the least calcium and the most sodium were much more likely to have HTN. The amount of sodium, potassium and calcium in their diets was assessed by 24 hour recall method and fasting urinary sodium and potassium/creatinine ratios. Seventy-one subjects (64%) had HTN. Compared with normotensive subjects, the hypertensive subjects consumed less calcium and had a greater urinary sodium/creatinine ratio and a greater sodium/potassium ratio. Among 88 subjects not taking diuretics or other antihypertensive drugs, systolic BP was correlated inversely to calcium intake ($r=-0.40$), and positively with urinary

sodium/creatinine ratio ($r=0.39$), urinary sodium/potassium ratio ($r=0.30$) and age ($r=0.23$). Twenty-three subjects with high urinary sodium/potassium and low calcium intake and 16 subjects with low urinary sodium/potassium ratio and high calcium intake differed markedly in systolic blood pressure (159 ± 26 vs 130 ± 15 mmHg) and the prevalence of hypertension (78% vs 25%). The authors concluded “Older Chinese vegetarians are predisposed to hypertension because of their sodium-rich but calcium-deficient diets.”⁹⁴

Even though the ratio of salt to potassium in the diet is very important, one should not conclude that the absolute amount of salt is irrelevant. In animals, it was shown that increasing the amount of both sodium and chloride 3-fold resulted in a substantial 15 to 20 mmHg rise in systolic BP.⁹⁵ Recall also the 6 month study discussed earlier of human infants. In this study BP rose significantly over 6 months even though both sodium and potassium were both increased together to maintain the same ratio found in human milk. In patients with essential HTN a lower intake of potassium resulted in sodium retention and increased calcium excretion and this further exasperated their HTN.⁹⁶

Yi farmers from southeast China have a low average BP and experience only a modest rise in BP with age despite a moderate salt intake. Like the Kenyan farmers discussed earlier, the Yi farmers also experience a rise in BP when they migrate to urban centers for work. But unlike the Kenyan farmers, the Yi farmers do add a modest amount of salt to their food in their rural setting. However, the Yi farmer’s diet is exceptionally high in potassium (even for a relatively unacculturated population) and part of the reason their BP starts to rise when they migrate to the cities appears to due as much to the decrease in potassium, as it is the increased salt. Based on the amounts and ratio of potassium to sodium in their urine the researchers concluded that the rise in BP was due as much to decreased potassium (and perhaps also magnesium) as it was to increased salt.⁹⁷

Epidemiological evidence has found a decreased risk of stroke in US men who consume diets higher in potassium, magnesium and fiber.⁹⁸ A 12-week long randomized, controlled, parallel-group study found that 73% of those on antihypertensive medications could eliminate their medications and still maintain a BP within acceptable limits.⁹⁹ Because Americans now spend more than \$8 billion on antihypertensive medication per year the cost savings of such a simple dietary intervention are potentially enormous.

Studies of vegetarians have often found that they have lower BP than non-vegetarians. This has been attributed in part to the higher potassium content of the vegetarian diet compared with a more typical Western diet.¹⁰⁰ Other epidemiological studies have found that men who eat more fruits and vegetable have fewer strokes.¹⁰¹ Simply substituting a mixture of salt plus salts of potassium and magnesium for regular table salt for 24 week was found to significantly lower BP in a group of older HTN patients by an average of 7.6/3.3 mmHg.¹⁰²

Data from the DASH trials have led some to believe that a Mediterranean-style diet may be helpful for preventing of hypertension even if salt intake is not limited. However, it is well known that blood pressure rises with age in all Mediterranean countries just as it does in the United States and Asian countries with over 90% of these populations developing hypertension sooner or later. Indeed, a recent study in Spain showed that adherence to a more Mediterranean-style diet was not associated with a significant reduction in the risk of developing hypertension.¹⁰³

The lower saturated fat and higher potassium, calcium and magnesium content of the DASH diet and a more Mediterranean-style diet may mitigate some of the damage to the cardiovascular system over time and possibly slow the development of hypertension and cardiovascular disease and reduce the risk of cardiovascular events. However, as the DASH Sodium Trial showed reducing salt intake from about 3400 to 1500mg sodium/day in addition to reducing saturated fat and increasing whole grains, beans, fruits, vegetables, and low-fat dairy products lowered blood pressure more than doubled the reduction in blood pressure over 8 weeks. Clearly people who are led to believe that following a “Mediterranean-style” diet or the DASH diet without also restricting salt intake are likely to be disappointed.

A recent observational study looked at the risk of stroke and heart disease in over two thousand participants (30-52y) who were followed 1.5 to 3 years. The amount of sodium and potassium in their urine was measured several times during this period and researchers found a significant increased risk of stroke and heart attacks developing in those with a higher ration of sodium to potassium ratio in their urine.¹⁰⁴

Certainly there is reason to believe that eating a diet with more potassium, magnesium and calcium may mitigate some of the adverse effects of excessive salt intake. And eating a diet lower in fat, saturated fat, trans fat, and cholesterol will certainly cut the risk of cardiovascular disease but people should be led to believe that if they are eating an otherwise healthy diet that added dietary salt will not adversely effect their health.

Sodium without Chloride Appears Less Toxic

The human kidney plays a central role in the human body's ability in sodium and chloride balance. It turns out the human kidney is extremely efficient at retaining salt but has difficulty excreting large excesses quickly.¹⁰⁵ Research has shown that the same amount of sodium as MSG, baking soda, sodium phosphate or sodium citrate has far less impact on BP as it does when consumed with chloride.^{106 107} This difficulty excreting excess sodium chloride efficiently results in fluid retention. Salt and fluid retention begins somewhere between 20 and 50 mmol of salt daily.¹⁰⁸

Most salt substitutes contain potassium chloride, which has little or no impact on BP. However, the human kidney's ability to excrete potassium is more limited because the level in blood is so much lower than that of sodium and chloride. This requires metabolic energy to "push" the excess potassium into the kidney distillate. People with impaired kidney function or dialysis must avoid salt substitutes containing potassium chloride. In addition, the use of potassium supplements with ACE inhibitors, angiotensin antagonists and potassium-sparing diuretics can lead to a dangerous elevation of serum potassium levels. All of these drugs are frequently used to treat essential HTN. For this reason, potassium chloride salt substitutes should not be used without medical/dietary guidance. Nevertheless, potassium chloride, which is often sold as a salt substitute is available in most supermarkets in the US. Perhaps it is fortunate that potassium chloride has a relatively unpleasant metallic/bitter aftertaste. In rats, HTN is made worse when the diet is high in chloride and only has a moderate amount of salt.¹⁰⁹

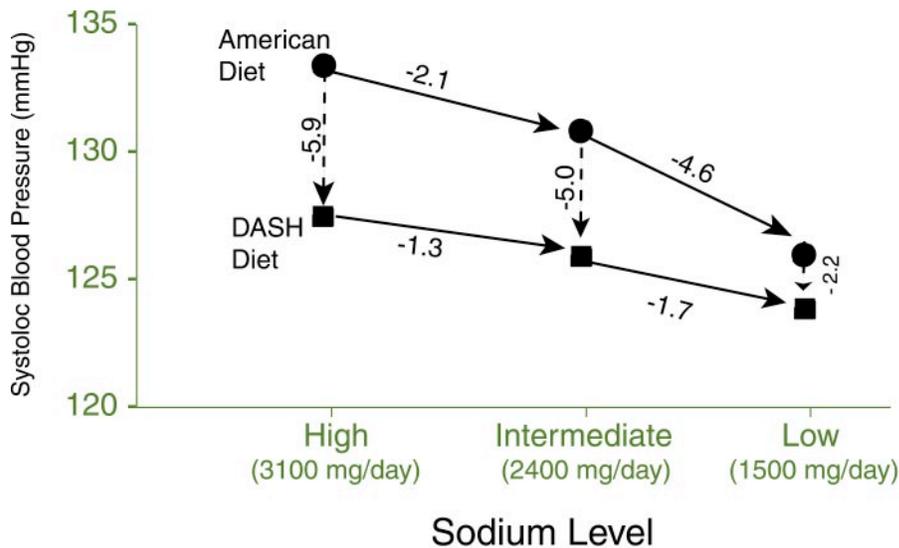
Sodium without chloride has been shown to have far less tendency to raise BP in people than salt.¹¹⁰ This means that other sodium salts such as sodium citrate or MSG would be far less likely to cause HTN than salt and so would be far safer for most Americans to add to their food than table salt.¹¹¹

Second DASH Study Proves Reducing Salt Lowers Blood Pressure

The now famous DASH study found that a low-fat diet with more fruits and vegetables, whole grains and low-fat or nonfat dairy products can "substantially lower blood pressure" even though dietary salt intake was still a fairly high 7.5 g/day (3000mg of sodium or 128 mmol). These authors conclude, "This diet offers an additional nutritional approach to preventing and treating hypertension."¹¹² Note that they did not conclude that the DASH diet offered an *alternative* to a low-salt diet for treating HTN. The Salt Institute was fairly successful at spinning the results of the first DASH study. Their spin led many health professionals to believe the DASH study showed eating more fruits, vegetables, whole grains and low-fat dairy products was far more important to reducing BP than cutting back on dietary salt intake. Of course, dietary salt was held constant in this clinical trial so technically it was not even studied.

To answer once and for all whether or not dietary salt influenced BP the DASH researchers conducted a second trial. In this study, both dietary salt as well as other dietary components were varied. This second DASH trial (a.k.a., DASH-II or the DASH-Sodium trial) showed that the drop in BP in response to reducing sodium from about 3300 mg/day to about 1500 mg/day lowered BP even more than simply following the DASH diet without reducing salt intake. The authors of DASH-Sodium trial conclude, "The reduction of sodium intake to levels below the current recommendation of 100 mmol per day and the DASH diet together both lower blood pressure substantially, with greater effects in combination than singly."¹¹³ Both the DASH diet and sodium restriction alone lowered BP compared to a typical American diet. However, the reduction in BP in both those with prehypertension and hypertension was greater when salt intake was cut from 3300 to 1500mg/d than when the DASH diet was consumed in place of the American-style diet and the salt intake was held constant. The DASH-Sodium trial clearly refuted the Salt Institute's spin on the first DASH study. This DASH-Sodium Trial clearly showed that reducing dietary salt to only 1500 mg of sodium is effective in lowering BP in people with "high-normal" (now called prehypertension) BP as well as those with "mild" HTN – see **figure 4** below.

Figure 4. Results of the DASH-Sodium Trials



Sacks FM, et al. *N Engl J Med* 2001;344:3-10

Of course, at even 1500mg of sodium daily this far exceeds the human requirement for sodium and chloride. Would even greater reductions in salt even be more effective for reversing established HTN? To be fair with Gary Taubes, it should be noted that the results of the DASH-Sodium trial were not available back in 1998. In 1998 *Science* published Taubes “editorial piece” in which he claimed a growing scientific consensus now believe that lowering dietary salt intake has little or no impact on BP except perhaps that of those unlucky hypertensive individuals who are “salt-sensitive”. Apparently neither *Science* nor Mr. Taubes feel the results of DASH-Sodium trial warrant a retraction of his irresponsible polemic.

The impact of far greater reductions in dietary salt intake than achieved in the DASH-sodium trial can cause an even more dramatic fall in BP, particularly in those with moderate to severe HTN. As discussed earlier, both the Pritikin¹¹⁴ and Kempner rice diets^{115 116} can dramatically lower very high BP in most patients. More recently, a study examined the impact of diet 174 HTN patients. They were placed on a low-fat, no-added-salt vegan diet for the first 3 days. Then they fasted on distilled water only for an average of 11 days. They were then placed back on the same very-low-salt vegan diet for about another week. Average weight loss over an average of 20 days was nearly 15 lbs. No doubt some of this weight loss was due to diuresis. Despite their HTN only 11 of the 174 were initially on anti-hypertensive drugs and by the end of the study all subjects were off all BP lowering drugs. The results are shown in **Table 4** below. The average drop in BP

was 37/13 mmHg. However, for those with the most severe HTN, the average drop in BP was a remarkable 60/17 mmHg. Nearly 90 % of all patients were able to attain a BP below 140/90 mmHg within 20 days despite all of them having Stage 2 HTN initially.¹¹⁷

Table 4. Effect of fasting followed by a no-salt-added, vegan diet on blood pressure

HTN Category	Subjects	Initial BP	BP after Fasting	BP after refeeding
Stage 1	92	146/86	122/76	116/74
Stage 2	57	166/92	131/79	126/77
Stage 3	25	194/96	140/82	134/76

Source: Goldhamer et al. *J Manipulativ Physiol Ther* 2001;24:335-9

Of course, compliance with any very low-salt, low-fat, near vegetarian or vegan diet is difficult at best in most patients. However, patients should be told what the most efficacious dietary approach is for treating their disease. They should be told that any dietary change from the typical American diet towards such diets should be beneficial as they reduce not only BP but also other dietary factors associated with CVD. Indeed, the TONE study demonstrated that even moderately reducing salt intake and modest weight loss are clearly a safe and effective treatment for HTN in older people.¹¹⁸ It now appears that a low-salt diet coupled with plenty of fruits, vegetables, whole grains, and nonfat dairy products and perhaps a little fish is best for treating and preventing essential HTN. This is partly because it promotes weight loss without hunger and more importantly long term it increases the ratio of potassium, calcium and magnesium to salt in the diet. Such a diet is more consistent with the nutritional needs of human beings and no doubt removes the nutritional stress that leads to the development of primary HTN in the first place and also accelerates the disease process once HTN has become established.

Does Dietary Fat Influence Blood Pressure?

There is some evidence that an increase in omega 3 fatty acids may have small hypotensive effect.^{119 120} There is also some evidence that very large changes in the ratio of polyunsaturated to saturated fat may also have a modest tendency to lower BP.¹²¹ But suggestions that the amount of dietary fat or the type of dietary fat play a greater role than dietary salt and/or the ratio of salt to potassium in the diet in promoting HTN has not been supported by any credible scientific research. As discussed earlier, the Kempner rice

diet, which is very low in salt and fat, was shown many years ago to lower BP just as effectively if more fat was added to this diet.¹²²

Epidemiological studies have linked increased total fat and saturated fat intake with an increased incidence of HTN. These studies are usually confounded by the tendency for dietary fat and saturated fat intake to rise in conjunction with increased dietary salt and body weight as populations come to rely more on manufactured foods and animal products. One exception to this may prove instructive. In the Samoan Islands, saturated fat intake has actually dropped precipitously (from 30% to 16% of calories) as their diet has become more Westernized, due largely to a reduction in coconuts. However, their sodium intake increased by about 1/3 (from 622 mg to 884 mg/1000 kcal) and the ratio of K/Na was cut nearly in half (from 2.5 to 1.3) as the Samoan diet Westernized and these are more typical changes associated with a more modern diet. In Western Samoa the diet is higher in total fat (46% versus 36%) and saturated fat than in American Samoa (the latter is more Westernized). However, the incidence of HTN (defined as SBP>160mmHg or DBP>95mmHg or on anti-HTN medication) was 44% and 32% for men and women, respectively in American Samoa but only 17% and 15% for men and women in the less acculturated Western Samoa.¹²³

Data from epidemiological studies of human populations that vary dramatically in their customary salt intake and other dietary components strongly suggests that the impact of dietary salt and potassium are far greater in the development of essential HTN than the impact of dietary fat (including both the percent fat and type of fat). However, there may be a modest benefit to including one or two servings of a high omega-3 fatty acid fish each week in a low-salt, DASH-style diet for treating patients with HTN. More research is needed to establish the ideal ratio of potassium, calcium, and magnesium, as well ratio of different types of fatty acids for treating and preventing HTN. A reasonable target for the ratio of potassium to sodium would be at least 4 or 5 to 1. Ideally sodium intake (from salt) would be no more than 1200 mg daily and even less is likely better. The current RDIs for potassium, calcium and magnesium are reasonable targets but should be easily achieved primarily by consuming a low-fat DASH-style diet. A low-fat diet may assist weight loss, which reduces the risk of many diseases. Reducing saturated and trans fatty acids and increasing omega-3 fatty acids is warranted whether or not such changes impact BP because such modifications do reduce dyslipidemia and help prevent CVD.

What Effect do Alcohol, Caffeine, Licorice and Smoking have on BP?

One or two drinks a day have little effect or no impact on BP. Alcohol in excess of two drinks a day tends to increase cardiac output, which mainly increases systolic BP.¹²⁴ When alcohol consumption is discontinued, the drop in systolic BP can take at most several weeks to be complete. Unlike excessive dietary salt, alcohol does not increase peripheral resistance (the hallmark of essential HTN) and its effects are more easily reversed so it is unlikely to play a significant causal role in the development of essential HTN. Alcohol's effects on BP are generally greater in older people whose arterioles and arteries are less pliant due (at least in part) to chronic over consumption of excess salt and/or too little dietary potassium. The Tarahumara Indians of Northern Mexico have a fairly high consumption of alcohol and yet HTN is very uncommon - no doubt due largely to the low salt content of their diet.^{125 126}

Smoking also increases heart rate and cardiac output with elevations of SBP of about 5-10 mmHg occurring shortly after lighting up a cigarette. However, this very transient rise in BP does not appear to lead to the development of essential HTN. Quitting smoking for 6 weeks was not found to have any significant effect on BP.¹²⁷ Of course, even if quitting smoking does not produce any significant reduction in BP in the long-term, there are still many other proven dangers to smoking so this is no excuse not to quit.

Licorice contains a plant steroid that causes fluid retention, hypokalemia and raises BP. The body can cope with small amounts of licorice but homeostasis is limited on a high-salt diet when plasma renin levels are low (as is often the case in older people). The availability of licorice candy for children and teenagers with its rather potent steroid activity seems questionable.¹²⁸ This is particularly true since a very acceptable artificial licorice flavor is widely available and perfectly acceptable. In this case natural is not better than synthetic.

Caffeine has long been known to elevate BP acutely. A double blind, randomized, cross over design study examined the acute impact of 250 mg of caffeine in subjects who consumed no coffee in the previous 3 weeks. The researchers found that plasma rennin activity jumped 57% and that of plasma epinephrine and norepinephrine increased by

75% and 207%, respectively.¹²⁹ Mean BP increased by 14/10 mmHg one hour after the caffeine was consumed. Coffee contains two diterpenes that can increase LDL-cholesterol levels.¹³⁰ Increased renin promotes the conversion of angiotensin I to the vasoconstrictive angiotensin II. This means caffeine increases 3 of the most powerful vasoconstrictive agents in the human body. Coffee, including decaffeinated has been shown to raise homocysteine (Hcy) levels in the blood.¹³¹ Both increased Hcy and LDL-cholesterol are known to damage endothelial cells and reduce their NO activity. NO is the major vasodilating agent in arteries and arterioles. Caffeine has also been shown to reduce insulin sensitivity by about 15% in human subjects, possibly due to the increase in epinephrine levels.¹³² This means coffee can raise BP by both increasing vasoconstriction and reducing vasodilation. A clinical trial of patients with HTN who stopped drinking coffee did find a significant drop in BP, at least in the short-term.¹³³

There is no doubt that caffeine can elevate BP acutely but the more important consideration is whether or not coffee or caffeine consumption contributes to the gradual rise in BP with seen with age in the United States and other modern societies. A study of 1017 former medical students who graduated between 1948 and 1964 were followed for an average of 33 years. BP and the incidence of HTN were determined each annually by self-report. Researchers did find a modest increase in the incidence of HTN over time in the coffee drinkers. However, even in those who never drank coffee BP still rose markedly with age. The authors of this study conclude, “Over many years of follow-up, coffee drinking is associated with small increases in blood pressure, but this appears to play a small role in the development of hypertension.”¹³⁴

The impact of caffeine and coffee is largely short-lived and there is no compelling evidence to suggest it plays a major role in the inexorable rise in BP with age seen in all human populations that add appreciable amounts of salt to their food. Nevertheless, HTN patients should be advised to cut back or even stop drinking coffee. At least in the short-term this will help lower BP and may reduce other CVD risk factors as well. However, coffee consumption is at best only a very minor factor in the very high prevalence of HTN in the US and other societies that consume a high-salt diet.

Other Factors That May Increase Blood Pressure

Sleep apnea is often associated with HTN. Heavy snoring, even without sleep apnea may increase BP. Effective treatment of sleep apnea does lower BP in most afflicted patients.¹³⁵ Heavy metals such as lead, mercury and cadmium are all quite toxic to the kidney. Anything that damages the kidney may reduce its capacity to excrete excess salt and fluid and so contribute to the development of HTN. Indeed, a recent review suggests that it is a subtle development of kidney damage that leads to the development of salt-sensitive HTN.¹³⁶

Drugs such as oral contraceptives, estrogens, NSAIDs, antidepressants MAO inhibitors, cyclosporin A, decongestants, Meridia, inhaled pressor amines and herbal products containing ephedra (a.k.a., Ma Huang) can all raise BP and alternatives should be sought in patients with HTN. Patients with HTN should be instructed to check with both their physician and pharmacist as to whether any prescribed and/or nonprescription drug they are taking or thinking of taking could further elevate their BP.

What About Studies that Found More Deaths in People with Lower BP or with a Lower Salt Intake?

There are many known risks of serious health problems associated with higher BP. Therefore, it is somewhat surprising that in several studies of older people those with the lowest levels of BP, cholesterol and body fat are actually most likely to die compared to those with more moderate levels of BP, cholesterol and weight. Increased mortality at high and low levels results in what epidemiologists call the J-shaped curve with the lowest risk of dying in the moderately low range but with risk of dying rising at the very low range. This has led some to speculate that we should not aggressively lower these known risk factors for cardiovascular disease, at least in older people. However, these studies are always confounded because of the poor health at the start of the study of many of those with the lowest BP, cholesterol levels and body weights. People in poor health often have poor appetites and eat less. They gradually lose weight, their cholesterol level and BP fall and then they die. For example, people who suffer a heart attack often have lower BP afterwards because their heart is weaker and can't pump as hard. People with long standing poorly controlled HTN often develop congestive heart failure and as they do their BP falls. People with cirrhosis often have very low cholesterol levels and poor

appetites. People with many types of cancer and those with long-standing diabetes often lose weight and experience a fall in BP before they die. So there are a lot of ways an older person can end up with a lower BP, body weight and/or cholesterol level and yet still be more likely to die. However, to claim the low BP, serum cholesterol level or body weight caused them to die is confusing cause with effect.

In the long-term, lower BP in old age is associated with better survival. Short-term studies may come to differing conclusions because co-morbidity and frailty can result in a lower BP near the time of death. Overall, most research suggests that lowering BP in the aged is still efficacious.¹³⁷ A recent publication¹³⁸ used a meta-analysis of 61 prospective studies that included data from one million adults to examine the relationship between one's usual BP and their risk of dying from CVD and also from all other causes in adults in different age groups. They looked at the impact of BP at different ages to determine the BP level at which the risk of dying of a stroke, heart attack or other CVD event was the lowest. They also looked at the relationship between dying of non-CVD related illnesses and BP for people in the same age groups. **Figure 1** below shows the relative risk of dying from a heart attack for people with different usual systolic BP levels at different ages. This risk was lowest for those whose usual systolic BP was 115 mmHg regardless of age. Regardless of age, the death rate from heart attacks increased dramatically with increasing systolic BP. It is clear that the risk of dying from a heart attack and other CVD is lowest for those with the lowest systolic and diastolic BP and the risk of dying increases with increasing BP regardless of age. In fact, the risk of dying from a heart attack for people 40s, 50s, 60s, 70 and even 80s about doubled for every 20mmHg increase in systolic BP. This study also found that every 10mmHg increase in diastolic BP starting 75mmHg doubled the risk of dying from CVD.

There is no longer any doubt that the risk of dying of a CVD increases above systolic BP of 115mmHg and/or a diastolic BP of 75mmHg and this was true for all middle-aged and older adults. The authors of this massive meta-analysis conclude, "Throughout middle and old age, usual blood pressure is strongly and directly related to vascular (and overall) mortality, without any evidence of a threshold down at least to 115/75 mmHg." Other data presented in this study made it clear that the risk of dying of a stroke increases somewhat more rapidly with rising BP than the risk of dying of a heart. For strokes an increase of 20mmHg in systolic BP or 10mmHg in diastolic BP was associated with more

than double the risk of dying from a stroke. The lower a person's usual BP the lower his risk of dying from any form of CVD at least down to a usual BP of 115/75mmHg. In addition to a much lower risk of dying from CVD this study also showed a higher BP was also associated with a modest but statistically significant increased risk of death from all other causes combined.

There is no plausible theoretical basis to explain how a lower BP, body weight and/or cholesterol level can promote disease and increase the risk of dying in the elderly and yet be protective in younger people. A 6 year follow-up study of 18,022 Norwegians (age 65y+) found that the J-shaped curve for mortality and BP was indeed "indirect, possibly caused by serious underlying disease".¹³⁹ Claims that higher BP ceases to be an important risk factor for CVD in older people have now been completely discredited. A claim that a lower BP at any age might cause an increased risk of dying from CVD or from other causes is simply inconsistent with the scientific evidence.

Could Reducing Dietary Salt Intake by One-Third Be Dangerous?

On Jan. 21,1999, Salt Institute president Dick Hanneman wrote to NHLBI's director Dr. Claude Lenfant and suggested that the NHLBI's Workshop on Sodium and Blood Pressure focus on the "safety" of reducing dietary salt intake by 1/3. Mr. Hanneman wanted the assembled experts (on very short notice as the conference was to be held on Jan. 29th+30th) to alter their agenda. He claimed the focus should shift so as to "identify published data demonstrating that (reducing dietary salt by 1/3) would not impair mental function, disturb sleep, produce deficiency-level intakes of other electrolytes and he stressed, would health outcomes be improved (sic)".¹⁴⁰ Of course, most scientists know you cannot prove a negative assertion (a.k.a. the "null hypothesis.") To suggest that moderately reducing the already very high-salt intake of most Americans could result in a "deficiency-level" or somehow be harmful is simply not supported by any credible scientific research. Such fear mongering by the Salt Institute is an example of food terrorism. However, the Salt Institute's spin on salt has sadly become widely accepted by physicians, other health professionals and the general public.

Even wackier than the questionable interpretation of the association between very low levels of BP and increased mortality are "studies" which purport to show an increased risk of dying in people who eat less salt. For example, Michael Alderman (a former

consultant to the Salt Institute) published a study that purported to show that people who ate less salt were more likely to have high BP and more likely to die.¹⁴¹ Their salt and calorie intake was estimated based on a single 24-hour diet recall back in the early 1970's. In Dr. Alderman's study, the people consuming the "lower sodium" diets were actually consuming more sodium per calorie than those consuming the "higher sodium" diets. The only reason they were on a "low sodium" diet was that they had reported eating less than 1000 kcal during the past 24 hrs. When asked to comment on this study Dr. Lawrence Appel of John Hopkins University stated: "No good researcher would even publish the unadjusted data" and "I don't know how this study got published. It's outrageous."¹⁴² Letters to the editor exposed the flaws in Alderman's "studies". The numerous flaws in these studies were pointed out in two letters to the editor.¹⁴³

The timing of Dr. Alderman's study is also suspect. It was released at about the same time as Dr. Whelton's TONE study, which clearly established the efficacy of a low-salt diet (<1800 mg/d) in treating older HTN patients. One can only wonder how Dr. Alderman's bogus study ever garnered so much favorable media attention. Could the massive press releases by the Salt Institute have been a factor? Mr. Hanneman admitted to being "distressed" by the withdrawal of Dr. Alderman from the NHLBI Workshop (Jan. 29&30, 1999). Perhaps Dr. Alderman simply hoped to avoid further personal embarrassment regarding his two bogus "studies" which will probably only be cited as an example of poor science like the "cold fusion" fiasco back in the 1990s.

Doesn't A Low-Sodium Diet Lead to Hyponatremia in Athletes?

Sweat typically contains about 1 gram of salt or 400 mg of sodium per liter. There have been cases of sodium depletion or hyponatremia in endurance athletes even those consuming a fairly high salt intake (10-15 gm/day). This has led some to speculate that a low-salt diet could be dangerous for athletes or anyone who is sweating profusely in a hot, humid environment. Although it is not well recognized, the human sweat glands are quite capable of conserving sodium and chloride when the diet is low in salt. A study at the University of Michigan many years ago found that young men could lose an average of 7 liters of sweat daily by doing several hours of intense exercise in a hot, humid environment. However, they were able to maintain normal sodium levels consuming just 0.75 gm of salt (300 mg of sodium) per day.¹⁴⁴ The historian Michael Hanneman (Salt

Institute president) apparently overlooked this historic study when he suggested that a low-salt diet could be dangerous for athletes.

The human body adapts to a low-salt intake by reducing salt in sweat and in the urine. In Dr. Conn's study the men acclimatized to a very low-salt diet had sweat that contained only 40 mg of Na/L and their kidneys excreted just 20 mg Na/day. The Yanomamo Indians of the tropical rain forest of South America do not develop hyponatremia despite a sodium intake of less than 200 mg/day, an active lifestyle, and an average daily temperature of 100+ degrees Fahrenheit.¹⁴⁵ This suggests that hyponatremia in athletes could be due in part to a high-salt intake. This is because it takes a week or more for the body to fully adapt to a very-low-salt intake. A study of 488 runners in the Boston Marathon found that 13% developed hyponatremia (serum sodium <135mmol/L) and 0.6% developed severe hyponatremia (serum sodium <120mmol/L).¹⁴⁶ The single biggest predictor of developing hyponatremia was weight gain. Weight gain while running a marathon is certainly the result of excessive fluid consumption. Indeed, some of the athletes who developed hyponatremia were drinking sports drinks containing salt. Hyponatremia is more the result of water intoxication than it is a lack of salt in the diet. Indeed, a high-salt diet reduces aldosterone levels and greatly increases salt loss in the sweat. If athletes consume large quantities of fluid without salt or even sports drinks containing salt and gain weight they may be risking hyponatremia. The risk is greatest for those who are adapted to a high salt intake because their sweat will still contain a lot of salt and after several hours of profuse sweating they can become salt depleted to the point where hyponatremia can develop. The irritation of dried salt on the skin of athletes could be significantly reduced if they consumed a very-low-salt diet.

But Aren't Drugs the Most Effective Treatment for HTN?

Natural allies of the Salt Institute are the big pharmaceutical companies who rake in billions of dollars every year from the sale of their numerous antihypertensive drugs. About 75 million Americans have HTN and most of them are being treated with BP-medications. HTN is the #1 reason Americans visit a physician's office and BP-drugs account are either the #1 or #2 most prescribed class of drugs in America. In the words of Dr. Laragh (a long-time critic of the salt causes HTN hypothesis) and a well-paid consultant for the pharmaceutical industry, "existing drugs are potent enough to render

the misery of effective life long salt restriction unnecessary."¹⁴⁷ Mr. Hanneman found it "distressing" that Dr. Laragh withdrew from a NHLBI Workshop on the role of. One might suspect that the CEO's at some big pharmaceutical companies were also distressed. Americans spend billions of dollars each year on drugs, doctor visits, hospitalization, dialysis treatment and nursing care for those disabled by strokes, heart failure, and other cardiovascular diseases due to HTN. Some would argue that treating HTN with drugs is still cost effective because it is the safest and most effective way to treat HTN. Does the research bear this out?

To the surprise of many, the MRFIT trial found that men judged to be at high-risk for CHD who were treated more aggressively with drugs to lower their BP did not fair any better than those in the usual care group unless their initial DBP was greater than 100 mmHg. In those with an initial DBP of 90-94 mmHg those treated more aggressively with drugs in the "Stepped Care" model were actually more likely to die.¹⁴⁸ These findings are all the more disturbing if it is true that a low-salt DASH-style diet could have prevented HTN from having developed in the first place. We know the difference in life expectancy between middle-aged people with "stage 1" or higher HTN compared to those with an optimal BP (<110/70) is probably at least 10 to 20 years. With drug therapy even successful treatment of "moderate" HTN adds on average no more than a year or two to life expectancy. If ever it were true that an ounce of prevention is worth a pound of cure this has got to be it.

An even bigger challenge to the notion that drug therapy is the best way to treat "stage 1" to "stage 2" HTN was the even larger study conducted by the British Medical Research Council. In this study over 17,000 HTN patients with initial diastolic BP between 90 and 109 mmHg were either given a placebo or an anti-HTN drug (either a thiazide diuretic or a beta-blocker) or a placebo. In those receiving the drugs the dosage was gradually increased and additional drugs added (if need be) to get the diastolic BP below 90 mmHg. The results showed a decreased risk of strokes, but the overall mortality of women in the drug treatment was increased by 25%, while in men the overall mortality rate in those taking anti-HTN drugs did drop but only by 13%. The authors of this study conclude, "treatment did not appear to save lives or substantially alter the overall risk of coronary heart disease." The results of this study suggest that no treatment is nearly as effective as drug treatment for those with "mild" to "moderate" HTN! And the one proven benefit of

drug therapy of reduced strokes came at a very high cost. In addition to the adverse side effects of the drugs used in this study, analysis of the results showed that for every 850 HTN patients treated with these drugs there would only be about one stroke prevented each year. With the political climate turning warmer for adding a drug benefit to Medicare society should be clear as to what the overall cost (both adverse side effects and monetary) versus benefits are. It seems likely that if this same amount of money went into medical nutritional therapy and public health measures that society as a whole would benefit (albeit not the large pharmaceutical companies, hospitals or physicians).

The good news is that the use of anti-HTN medication is of some value in reducing strokes in those with "mild" to "moderate" HTN and in reducing overall mortality in those with very high BP (i.e. 180/110 mmHg or higher). However, the 1993 Report of the Working Group of the NHLBI pointed out that "Even in those who derive optimal benefit from their antihypertensive treatment, they are likely to have a higher risk of morbidity and mortality than their untreated "normotensive" counterparts with a similar level of blood pressure."¹⁴⁹

It is known that some of the drugs used to treat HTN such as thiazide diuretics and beta-blockers can elevate blood sugar and promote the development of type 2 diabetes. Because type 2 diabetes increases the risk of CVD the long-term efficacy of such drugs has been questioned. A 14-year follow-up study examined the impact of treating isolated systolic HTN in 4,732 elderly subjects with either a thiazide diuretic or a placebo. The results showed those taking the diuretic were 40% more likely to end up with type 2 diabetes than those taking the placebo. Nevertheless, CVD mortality dropped nearly 15% (from 22% down to 19%) over 14 years in those taking the diuretic compared to the placebo.¹⁵⁰ Of course, while a 15% reduction in CVD mortality is important this has to be compared to the 300-500% increased risk of CVD mortality that we now know accompanies even the mildest forms of HTN.¹⁵¹ High blood pressure is prevented in most people with a diet low in salt so it appears dietary prevention is far more likely to prevent CVD events than waiting for HTN to develop and then treating it with drugs. Indeed, a low-salt diet often eliminates the need for drugs to treat HTN.

The HOT study found no evidence that decreasing systolic BP much below 140 mmHg with drugs reduced overall mortality even though epidemiological evidence clearly shows

mortality rising with any systolic BP above 110 mmHg.¹⁵² Until recently only about 5 million of 50 million HTN people in the US have their BP reduced below 140/90 mmHg. The good news from the HOT study is that physicians can now use higher dosages and/or more drugs to lower their hypertensive patient's BP to even lower levels without worrying as much about more aggressive drug treatment killing them more quickly. This may make the CEOs at the big pharmaceutical companies happy. The not so hot interpretation of the HOT study is that spending more money on drugs to more aggressively lower BP in HTN patients is not likely to reduce their overall mortality rate much either. They'll have to hope that HMOs and the government (Medicare and Medicaid) never do a cost/benefit analysis on outcome of drug therapy versus dietary therapy for treating patients with HTN. Of course, no drug is now approved for the prevention of HTN and yet dietary therapy alone has the largely untapped potential to make essential HTN an avoidable disease in nearly everyone.

Intensive Drug Treatment of Hypertension Fails

One of the ACCORD trials examined the efficacy of using more BP drugs to more aggressively lower elevated BP in patients with type 2 DM. A total of 4733 people with type 2 DM were randomly assigned to either lowering systolic BP to <120 or to the conventional BP target of <140. In theory pushing BP even lower than the older BP target of <140/90 makes sense because research has clearly shown the risk of CVD events increases dramatically with increasing BP starting at levels below 115/75mmHg. Indeed, the risk of death from CVD roughly doubles for each 20mmHg rise in systolic BP and this is true whether or not one also has diabetes. In this study the average BP for those in the more intensively treated group averaged only 119 compared to 134 in the usual care control group throughout the average 4.7y study period. The greater BP reduction achieved in the intensive BP control group used the same FDA approved drugs used in the control group subjects except that subjects in the more intensive drug treatment group received more of those drugs and/or took the drugs at higher doses than those in the control group. If the higher doses of BP drugs used in the intensive care group completely eliminated the increased CVD risk attributable to higher BP then the greater reductions in BP (Systolic BP reduced 15mmHg on average) seen in this study should have reduced CVD events by about 70%. Unfortunately, despite a significant

reduction in nonfatal strokes seen in the more intensively treated group the lower BP achieved with more drugs in the intensively treated group did not reduce total CVD events or CVD deaths significantly. There were 60 deaths in those treated more aggressively with BP-meds compared to 58 deaths from CVD in the control group despite the fact that the BP drugs maintained BP significantly lower throughout the study. Worse still total mortality after 4.7y of follow-up was 1.19% (or 144 deaths) in the standard therapy group and 1.28% (or 150 deaths) in the intensive drug treatment group. While neither of these small increases in CVD or total mortality in the more intensively BP-drug treated group were statistically significant they clearly were in the wrong direction for those who would like to argue that lowering BP more aggressively with more drugs will help save lives. In addition the study authors found 3.3% compared with only 1.3% of those in the more intensive drug treatment group experienced significantly more serious adverse events attributed to the antihypertensive treatment in the standard treatment group.¹⁵³

Another observational study examined the impact of achieving tighter BP control using FDA approved pharmaceutical agents in type 2 DM subjects with coronary artery disease. The results showed no reduction in total mortality and/or CVD events in patients whose BP was reduced below 130 compared to those who achieved a more modest reduction of systolic BP into the 130-139 range. However, in patients whose BP was reduced close to the "optimal" BP range of 110-115 there was actually a strong trend (adjusted hazard ratio of 1.63, $p=.06$) towards increased all-cause mortality. And in those who reduced their BP to the presumably optimal level with drugs (to systolic BP <110mmHg) there was more than a doubling (adjusted HR =2.18 $p<.02$) of the risk of dying.¹⁵⁴ The findings of this observational study are disturbing because current guidelines from the American Heart Association, American Diabetes Association, and the Seventh Report from the Joint National Committee all recommend more aggressive BP targets in line with what was accomplished in many of the subjects in this study.

The results of the ACCORD studies clearly show that prescribing yet more drugs to more aggressively lower BP and other CVD risk factors in type 2 DM patients is usually not medically justified. Indeed the authors of the ACCORD study examining the impact of more aggressively lower blood glucose levels with drugs for several years concluded:

"As compared with standard therapy, the use of intensive therapy to target normal glycated hemoglobin levels for 3.5 years increased mortality and did not significantly reduce major cardiovascular events. These findings identify a previously unrecognized harm of intensive glucose lowering in high-risk patients with type 2 diabetes."¹⁵⁵ Simply put this study showed that using drugs to achieve more physiologically normal and presumably healthier levels of blood sugar with drugs actually result in more deaths than less aggressive drug therapy. Patients who wish to reverse insulin resistance, hypertension, and dyslipidemia should be told that more aggressive dietary approaches such as a low-sodium, very low-fat, more vegetarian diet composed largely of whole grains, fruits and vegetables and weight loss is not only a far safer but also a far more efficacious treatment strategy than the increasingly knee-jerk response of using more aggressive pharmacotherapy to "improve" blood glucose levels, blood lipids, and CVD risk factors.¹⁵⁶ It is a sad commentary on America's drug-oriented approach to healthcare that diseases known to be largely caused by the modern Western diet and other lifestyle factors are still largely treated with only marginally effective drugs that often cause adverse side effects. Is this not why CVD remains the killer of almost half of all Americans who die each year while the prevalence of HTN, obesity, and type 2 DM continue to rise?

Reducing Salt Effective for Lowering BP in Resistant Hypertension

Resistant Hypertension (HTN) is diagnosed when the patient's blood pressure (BP) remains at 140/90 or higher despite being on 3 or more anti-HTN drugs. Clinical trials have shown that between 20 and 30% of the people in America with HTN now have resistant HTN.¹⁵⁷ While most patients initially diagnosed with HTN can have their BP "controlled" by BP drugs the longer the patient follows the typical medical treatment model the more likely it is they will require more and more drugs to get their BP below 140/90. Eventually taking even 3 or more BP drugs cannot "control" their BP. This failure of the modern medicine to prevent and treat HTN begins with its failure to recognize and successfully deal with salt toxicity, which is the known primary causal factor of primary HTN and is the main factor driving BP higher over time.

For the first time ever researchers evaluated the effects of consuming a low-salt diet on the BP of patients with resistant HTN. They also examined dietary salt-related changes in the RAAS system, blood volume and arterial stiffness that occurred in just one week on either a low-salt or high-salt diet. The study participants included 12 mostly overweight and obese people with resistant HTN ages 34-66y. Their average BP at entry to the study was 145.8/83.9mmHg despite taking an average of 3.4 BP drugs. The subjects were then fed for one week either a low salt (<50mmol or 1150mg of Sodium/day) or high salt (>250mmol or 5750mg of Sodium/day) diet. The low-salt diet was provided by the researchers and was designed to be nutritional similar to their usual diet except that it was much lower in salt. In just one week the average BP of the subjects on the low-salt diet dropped to 122.8/74.9. By contrast, after one week on the high-salt diet their BP was 145.6/84.0. The researcher also assessed 24hr ambulatory BP on the 2 diets and found it averaged 150.3/82.1 on the high-salt diet and 130.0/72.8 on the low-salt diet. The authors of this study said there results “...suggest that patients with resistant hypertension are particularly salt-sensitive and emphasizes the importance of low dietary salt intake in the clinical management of resistant hypertension.”¹⁵⁸ They also found evidence that this BP reduction was due in part to a reduction in blood volume and a decrease in arterial stiffness. The authors also stated that: “All of the subjects enrolled into the current study reported having been previously advised to lower their dietary salt intake, and all reported having done so.” However, they also noted not one of the subjects had received expert dietary counseling to reduce salt intake. An initial 24-hour urine test showed that the average sodium intake for these subjects was 194.7mmol, which would correspond to a daily salt intake 11.6g.

The results of this study on resistant HTN and the ACCORD hypertensive study discussed in the previous section clearly demonstrate that drug therapy is far from an optimal treatment of HTN. It shows that modern medicine’s failure to focus on the main cause of primary HTN, which is salt toxicity, leads most MDs to inadequately treat what is driving the disease process and instead focus on elevated BP, which is merely a symptom of salt toxicity. It is because most physicians focus treatment primarily on pharmacological therapy rather than salt toxicity, that resistant HTN develops. Current medical guidelines for drug therapy target a BP below the hypertensive range (<140/90) but rarely return BP to its normal physiological range of around 110/70. Indeed, as the

ACCORD study showed even if more FDA approved drugs are used to push BP lower the result is not fewer CVD deaths or total mortality.

Since it is now known CVD events begin to increase exponentially as BP rises above 110/70mmHg BP-drugs are far too dangerous physiologically to high BP much below 140/90mmHg in most people. It should be clear that anyone who is told their BP is being “controlled” by drugs it is not coming close to eliminating pathological changes to the cardiovascular system from salt toxicity. Indeed, as the ACCODR data indicate the toxic effects of more drugs appear to outweigh all or nearly all the health benefit of achieving a lower BP with drugs. In addition to their shortcomings as a treatment for HTN most BP meds do little or nothing to prevent many of the other toxic effects of excess salt such as the promotion of stomach or kidney cancers, kidney stones, headaches, edema, heartburn, and osteoporosis. Indeed, in some cases BP-drugs may make some of these other salt toxicity effects even worse. As a result even when it is successful drug treatment does not come close to eliminating all the harm caused by excessive salt intake nor does it correct and may worsen electrolyte imbalances often coupled with excessive salt intake such as an inadequate intake of potassium, magnesium, and other minerals. This study on resistant HTN also clearly demonstrates that most people diagnosed with a disease caused largely by excess salt should be referred to RDs for expert dietary counseling as the limited dietary advice provided by physician typically produces little or no reduction in salt/sodium intake.

With 75 million Americans now diagnosed with HTN and over 15 million with resistant HTN it is clear that the FDA should reclassify salt as something other than “GRAS” and start taking steps to limit the amounts added to foods.

Does the scientific evidence support Dr. Laragh’s claim that anti-HTN drugs are now so “effective” that “the misery of effective life long salt restriction” is “unnecessary”? Or does it appear that anti-HTN drugs are far from an optimal treatment for HTN? Drugs are also far too dangerous to be used life-long to prevent HTN from developing in the first place. Even when restricted to those with established HTN their overall effect on mortality is questionable unless the HTN is severe. Aside from their cost, drugs have many adverse side effects that make a lot of people miserable such as impotence, dry mouth, dizziness, chronic cough, and reduced eyesight capacity. At least some beta-

blockers promote weight gain and increase the risk of developing type 2 diabetes. In the long run given the adverse side effects of drugs and reduced desire for salty foods with compliance with a low-salt diet one must ask if most people diagnosed with HTN would really be even more miserable eating a low-salt diet than they end up when their physician convinces them to go the drug rather than medical nutrition therapy route?

Comment on HTN drugs and Alzheimer's disease: Actually high blood pressure itself can cause senility and the higher it is the more rapidly mental function deteriorates. Many people with HTN (even on meds) suffer numerous little strokes that don't cause obvious stroke symptoms but do permanently damage the brain over time and lead to senility. HBP meds would not directly cause senility or Alzheimer's Disease (AD) - more likely they would slow the process down as we know they cut the risk of big strokes and lower BP. Now some BP meds we know speed up the development of diabetes and adversely affect blood lipid values and both diabetes and dyslipidemia do increase the risk of AD so indirectly they may play a role.

Must a Low-Salt Diet be Unpalatable?

Nearly all Americans grow up eating a high salt diet. However, the taste for salt appears to be largely learned. Anthropologists who have studied human populations that add no salt to their food report that people in these populations do not like the taste of salt when they are first exposed to it. Indeed, people in such populations often find salted foods unpalatable. Traditional Eskimos do not add salt to their foods. The word for salt in their language is "mamaitok", which is synonymous with "evil-tasting". The claim that foods without added salt are "tasteless" is ethnocentric. If the taste for salt is largely learned then it should be possible to alter one's preference for salt in foods.

When test subjects were fed a diet with only 70 mmol (about 1600 mg Na) per day and given free access to a saltshaker, researchers found that they compensated by adding only an additional 300 mg of sodium as salt. This resulted in a greater than 50 mmol reduction in salt intake, even though the test subjects were free to add as much additional salt to their food as they desired. The authors conclude, "A substantial reduction in dietary sodium is possible if lowered sodium foods are consumed in conjunction with ad libitum table salt."¹⁵⁹ Recall that 75% of the average Americans salt intake comes from manufactured foods. If the food industry would cut their use of salt by 50% it is unlikely

that many people would be miserable. However, according to Dr. Law's extensive review of the scientific literature even a 50 mmol reduction in salt intake would save about 250,000 lives in America each year as well as prevent many nonfatal strokes, heart attacks and other health problems that lead to real misery.

In addition to this, researchers have shown that moderate reductions in dietary salt for 2 months reduces the concentration of salt in food judged most pleasant.¹⁶⁰ A controlled study of young adults on a self-maintained low sodium diet for 5 months found that they came to enjoy lower sodium content foods.¹⁶¹ Another study found that after 24 weeks of a low-sodium diet the subjects most preferred level of salt in food declined from .72% to .33% (wet weight basis).¹⁶² It seems clear that one's salt preference can be reduced by the regular consumption of a diet much lower in salt.

It has also been shown that one can mask a reduction of salt in bread and water by adding acetic or lactic acid.¹⁶³ Sodium compounds other than salt including MSG and sodium citrate can enhance the taste of food. Both MSG and sodium citrate can improve the taste of food without also causing a significant rise in BP as does salt.¹⁶⁴ It seems likely that with a little creativity food scientists and chefs could develop food products that are as palatable or nearly as palatable as current high salt products but which will not lead the development of essential HTN.

One should not use salt substitutes containing chloride and should avoid significant amounts of other chloride-containing compounds if one elects to use baking powder (sodium bicarbonate), monosodium glutamate, sodium citrate and other sodium compounds in foods. It appears to be too much of both sodium and chloride together in the body that leads to the release of endogenous digitalis-like substance (EDLS). This ouabain-like hormone or EDLS is released from the brain and helps the kidney get rid of excess salt by blocking the re-uptake of sodium from the collecting tubules of the kidney. Unfortunately, it also blocks the sodium/potassium pump in the smooth muscle and endothelial cells of the arterioles. This leads to increased intracellular sodium and calcium. Whether or not it is EDLS or some other substance released in response to excessive salt intake has been a matter of scientific debate. More recently, a substance called marinobufagenin (MFG) may be more directly responsible than EDLS for the electrolyte disturbances that lead to chronic BP elevation.¹⁶⁵ By disrupting normal cell

chemistry MFG and/or EDLS (released in response to volume expansion caused by excess salt intake) may be behind much of the physiological damage to the vascular system seen in patients with essential HTN.^{166 167 168}

The Bottom Line

This review has presented evidence that many dietary and other factors can influence BP, at least in the short-term. However, the preponderance of evidence continues to favor excessive dietary salt as the single most important modifiable causal agent in the development of essential or primary HTN in the US and other modern societies. The food industry is largely responsible for most of the excessive salt consumed by Americans because most of the salt Americans eat was added to their food before they purchased it. One might argue that the food industry is simply supplying Americans with the amount of salt they want in their foods. But then how do we explain why Americans given foods prepared with far less salt do not add it all back when they have free access to a saltshaker? Also, much of the food industry, through its sponsorship of the Salt Intake and in many other ways, is playing a leading role in distorting the scientific evidence linking excessive salt intake to HTN, CVD and several other serious health problems. Clearly consumers are not making a free choice when they are being misled as to the likely consequences of their choice.

There is also reason to question why most medical doctors and the pharmaceutical industry are not fully informing patients with HTN about minimal proven benefits of drugs for the treatment "mild" to "moderate" HTN patients, at least in terms in terms of reducing overall mortality. Many doctors are also negligent in failing to fully inform their patients about the likely long-term benefits of reducing salt intake for reducing not only CVD risk but also the risk of osteoporosis, kidney stones and stomach cancer. Nor do most doctors routinely encourage their patients to consume a more natural foods diet high in fiber, potassium, calcium, magnesium and other potentially useful chemicals that may help reverse the biochemical and physiological damage caused by the typical American diet.

Salt appears to be a serial killer which has escaped indictment and prosecution thanks in large part to a well orchestrated public relations campaign conducted by the Salt Institute

and others whose financial interests conflict with those of people who'd like to be accurately informed about how what they eat impacts their risk of developing disease. Both the pharmaceutical industry and medical profession have financial interests that conflict with those of dietitians. The business of prescribing and selling pharmaceutical agents to treat HTN is highly profitable. Both medical office visits and increasingly prescription drugs to treat HTN are paid for private health insurers and Medicare/Medicaid. Dietitians and nutritionists services for expert dietary counseling are rarely covered by private and insurers and not covered by Medicare/Medicaid. By contrast, clinical dietitians make a living encouraging and assisting people to adopt a healthier diet, which if followed can more often eliminate HTN and the need for prescription drugs and medical office visits. Of course, this would not be the case for dietitians being paid by the pharmaceutical or food industry. As the clinical nutrition experts, registered dietitians have both a duty, as well as a financial incentive to fully inform their patients about the dangers of too much dietary salt and the impact of other dietary variables on BP regulation and disease.

For an in-depth review of Salt versus MSG (in favor of MSG) see <http://www.foodandhealth.com> and click on CPE courses at the top of the site. You will find the papers in the "heart disease and diet" section. <http://www.foodandhealth.com/continuinged.php> is the direct link.

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