

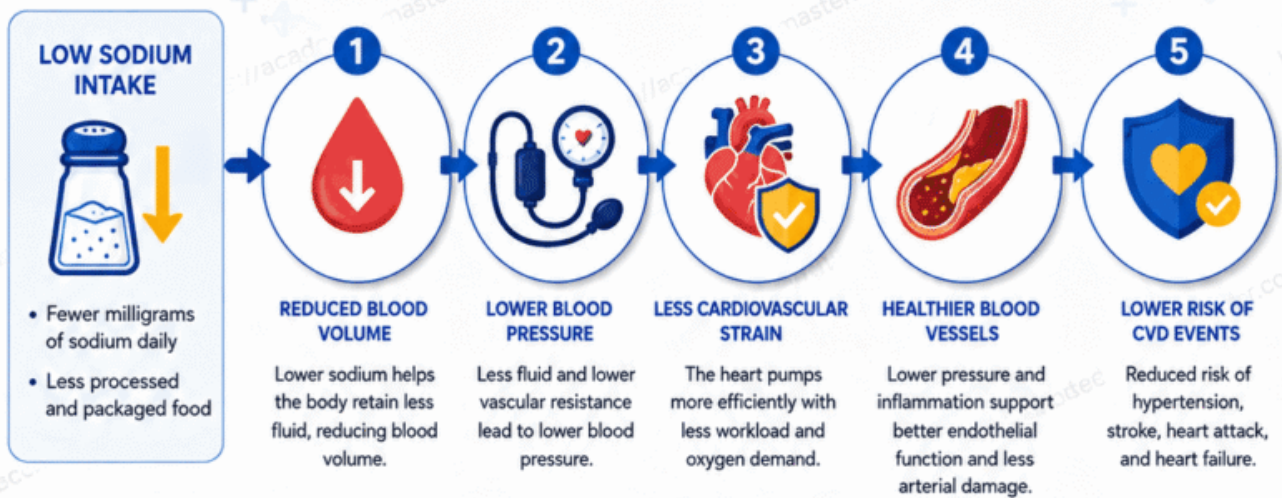
Effect Of Low Sodium Intake On The Cardiovascular System

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This paper will cover twelve different articles that cover the topic of the effect of low sodium intake on the system of cardiovascular. After the explanation of each article, a summary will be provided that serves as the finding of the article. The paper aims to identify the effect of intake of low sodium on the cardiovascular system by past researchers.

EFFECT OF LOW SODIUM INTAKE ON THE CARDIOVASCULAR SYSTEM

Sodium is essential for life, but too much can strain the heart and blood vessels.
Consistently low sodium intake supports a healthier cardiovascular system.



Consistently low sodium intake is linked to lower blood pressure and a significantly reduced risk of cardiovascular disease, especially when combined with a healthy lifestyle.

DASH-STYLE DIETARY CONTEXT

The DASH (Dietary Approaches to Stop Hypertension) eating pattern is rich in:

-  Fruits & Vegetables
-  Whole Grains
-  Legumes & Nuts
-  Low-Fat Dairy
-  Lean Proteins



✓ **DASH + low sodium = powerful protection for your heart and blood vessels.**

BALANCED GUIDANCE FOR HEART HEALTH



MONITOR YOUR INTAKE

Aim for <1,500–2,300 mg of sodium per day (as recommended by health authorities).



CHOOSE WHOLE FOODS

Cook more at home and choose fresh foods over processed and restaurant meals.



LIFESTYLE SUPPORT

Stay active, manage stress, limit alcohol, and maintain a healthy weight.



KNOW YOUR NUMBERS

Regularly monitor blood pressure and work with your healthcare provider.



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Article 1: “Long-term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of the trials of hypertension prevention (TOHP).”

According to this research, the effects of dietary sodium reduction are difficult to cope with. Many people, during their dieting and weight-loss experience, tend to reduce their sodium intake, which results in harm to the cardiovascular system. In this case, it is important to understand that balance is everything. Sodium is efficient and effective for health, and therefore this must increase its intake must not be reduced.

Therefore, from this article, it is learned that dietary reduction of sodium results in outcomes of cardiovascular diseases that must be avoided (Kalogeropoulos, 410-419).

Article 2: “Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet.”

“Hypertension influences very nearly 50 million individuals in the United States and places them at higher hazard for cardiovascular sicknesses. The Dietary Approaches to Stop Hypertension trial exhibited an eating regimen that stresses organic products, vegetables, and low-fat dairy items, that incorporates entire grains, poultry, fish, and nuts, contains just small measures of red meat, desserts, and sugar-containing drinks, and that contains diminished measures of aggregate and soaked fat. And, cholesterol brings down circulatory strain generously both in individuals with hypertension and those without hypertension, as contrasted and a run of the mill count calories in the United States.”

“The two weight control plans were a control diet normal of what many individuals in the United States eat, and the DASH slim down, which underlines organic products, vegetables, and low-fat dairy nourishments; incorporates entire grains, poultry, fish, and nuts; and contains littler measures of red meat, desserts, and sugar-containing drinks than the commonplace eating regimen in the United States. The DASH abstains from food likewise contains littler measures of aggregate and soaked fat and cholesterol and bigger measures of potassium, calcium, magnesium, dietary fiber, and protein than the run-of-the-mill diet.”

“Circulatory strain was measured with irregular zero sphygmomanometers while members were situated at three screening visits, twice amid the keep running in period, week by week amid the

initial 3 weeks of each of the three 30-day intersession periods, and at five center visits amid the most recent 9 days (no less than two amid the last 4 days) of every mediation period.” (Sensei, 1253-1261)

What did I get from this article?

In this article, it is said that the reduction in sodium intake results in the decrease of both systolic as well as diastolic blood pressure. This balanced diet also reduces circulatory strain.

Article 3: “Salt intake, stroke, and cardiovascular disease: the meta-analysis of prospective studies.”

“The causal connection between regular dietary salt admission and circulatory strain has been set up through test, epidemiological, relocation, and intercession thinks. Most grown-up populaces around the globe have normal day-by-day salt admissions higher than 6 g, and for some in eastern Europe and Asia, higher than 12 g. Global proposals recommend that normal populace salt admission ought to be under 5-6 g every day. Populace-based intercession thinks and randomized controlled clinical trials have demonstrated that it is conceivable to accomplish huge decreases in circulatory strain with diminished salt admission in individuals with and without hypertension.¹ Based on the impacts of high salt admission on the pulse and on the conspicuous part of hypertension in advancing cardiovascular infections, it has been proposed that a populace-wide lessening in salt admission could significantly lessen the frequency of cardiovascular disease.² On the premise of the consequences of a meta-examination of randomized controlled trials of salt reduction, it was evaluated that a diminishment in periodic dietary salt admission of 6 g a day would be related to diminishments in systolic/diastolic circulatory strain of 7/4 mm Hg in individuals with hypertension and 4/2 mm Hg in those without hypertension. Planned companion considers performed in the previous three decades that deliberate the levels of dietary salt admission at standard and recorded the occurrence of vascular occasions have given immediate circuitous confirmation. The vast majority of these reviews discovered confirmation of such connection, albeit few had enough energy to accomplish measurable noteworthiness.” (DuPont, 530)

Result:

In this article, it is concluded that a decrease in salt intake by 6g a day will result in a reduction in the frequency of cardiovascular diseases. It means that cardiovascular diseases can be controlled by a reduction in salt intake in diet plans.

Article 4: “The effect of high-sodium and low-sodium intakes on blood pressure and other related variables in human subjects with idiopathic hypertension.”

“Nineteen patients with hypertension in whom all known reasons for pulse rise had been discounted were named “salt-delicate” or “non-salt-touchy” from the adjustments in circulatory strain with changes in sodium consumption from 9 meq to 249 meq/day. With the eating regimen containing 249-milligram sodium every day, there were no measurable huge contrasts in plasma sodium, potassium, chloride, aldosterone, cortisol or renin movement, or in urinary potassium, aldosterone or 17-hydroxycorticosteroids between the two gatherings. The “salt-delicate” patients held more sodium on the high-sodium count calories than did the patients who were not touchy to salt (“non-salt-touchy”); as needs be, sodium prompted more weight to pick up in the salt-touchy patients.” (Aburto, 1326)

Salt-sensitive patients are more exposed to diseases like obesity because salt intake increases the weight of salt-sensitive patients. Sodium consumption changes the circulatory strain level, and it decreases. Sodium intake is a delicate matter for patients with hypertension.

Conclusion:

Patients who are salt delicate have more chances of cardiovascular diseases because a high sodium count settles patients of this disease. The increase in sodium consumption in the eating regime will increase the level of obesity. The reason is that it is due to sodium content that settles and increases in the count. (Mozaffarian, 624-634)

Article 5: “Global sodium consumption and death from cardiovascular causes.”

“Two late Cochrane meta-examinations assessed randomized trials of the impact of decreased sodium allow on blood pressure.^{1,2} One meta-examination depended on the consequences of 28 trials distributed through 2005.¹ The other depended on the aftereffects of 167 reviews and included later trials and additional trials, including low diminishments in sodium admission (<0.46 g [20 mmol] every day) or brief mediations (span of <1 week), which were barred from the principal meta-analysis.² These meta-examinations did not decide if blood pressure bringing down was direct over a scope of lessened sodium admissions and did not at the same time evaluate heterogeneity as indicated by age, race, also the nearness or nonattendance of hypertension. To figure out the impacts

of systolic circulatory strain on passing from cardiovascular causes, we consolidated comes about because of two huge worldwide undertakings (totaling 99 associates, 1.38 million members, also 65,000 cardiovascular occasions) that pooled singular-level information, reliably balanced for puzzling. We represented relapse weakening predisposition given serial circulatory strain measures over time.^{17,18} We introduced and extrapolated age-particular corresponding impacts (relative dangers) of systolic circulatory strain on cardiovascular mortality in 10-year age bunches over the pooling ventures. We utilized the same assessments of relative hazard as indicated by sex, what's more, race, on the premise of confirmation of, for the most part, comparative relative impacts of pulse on cardiovascular occasions as indicated by sex and race in trials of antihypertensive medications and observational investigations of circulatory strain and cardiovascular occasions."

Conclusion:

Sodium intake has larger effects on elder patients of cardiovascular diseases. It indicates a positive relationship between higher levels of sodium intake and [the] risk of cardiovascular disease. (Gradual, 1129-1137)

Article 6: "Effect of lower sodium intake on health: systematic review and meta-analyses."

Lower sodium intake usually causes lowering blood pressure with no effects on renal function, catecholamine levels, and blood lipids. This reduction of sodium intake also lowers blood pressure in children as well, ultimately reducing the chances of strokes. A well-scrutinized study was conducted to extract the outcomes from different data gathered. Sodium is not found in table salt; however, it is found largely in our daily edibles like milk, eggs, meats, fish, processed food, snacks, popcorn, pretzels, etc. A Meta-analysis was conducted independently to figure out the effects of lower sodium intake on adults and children. We demonstrate the configured results with 95% confidence intervals regarding the risk ratio. (Poggio, 695-704)

Article 7: "Low sodium intake—cardiovascular health benefit or risk?"

We found that a lower intake of sodium in adults causes a lowering of systolic blood pressure by 3.39 mmHg and diastolic blood pressure by 1.54 mmHg. When sodium was taken in less than 2g per day, the blood pressure lowered further, systolic by 3.47mmHg and diastolic by 1.81mmHg. As discussed at the start, it doesn't have the worst effect. There are many diseases that are triggered by increased sodium intake that are proving fatal for human health. Many casualties have been reported throughout the world in this perspective. 30% of the deaths were caused only by Cardiovascular that

is almost equivalent to the deaths caused by nutrition deficiency, infectious diseases, and to worst conditions of perinatal and maternal together. The underpinning cause of cardiovascular disease was found as the increased intake of sodium, which increases the risk of raised blood pressure and hypertension. These two risk factors contributed to 49% of coronary diseases and 62% of all types of strokes. We can find a large number of adults suffering from higher blood pressure all around the globe and about 50% of people with hypertension. Hence, hypertension and increased blood pressure are the two major problems confronted severely on a global level every day, and some modern approaches are to be considered to lower these risk factors in our daily lives. (Major, 2500-2501)

Article 8: “High dietary sodium intake impairs endothelium-dependent dilation in healthy salt-resistant humans.”

We know that an increased amount of sodium intake can cause an increase in blood pressure and hypertension, which are the basic factors of cardiovascular diseases. So we can say that increased intake of sodium can change the function of microvascular that are closely related to the changes in arterial pressure. For example, we can demonstrate this effect through an example: if some rats are fed on high salt intakes, it will ultimately reduce the responsiveness of arteriolar to stimuli that usually elicit dilation by NO release. (endothelial nitric oxide). These abnormalities can result in abnormal states of human-like hypertension, raised blood pressure, and diabetes, generally as a pathological influence on the function of microvascular. In this way, we can analyze the underlying reasons caused due to lower or increased intake of sodium and how they affect microvascular functions. A body with a heavy sodium intake has different arterial pressure than a body with a lower intake. It was calculated to be 0.45% for normal intake and 7% for High intake, which depicts the difference in arteriolar dilation among the two feed bodies of mice/mouse. (Grimes, 189-96)

Article 9: “Associations of urinary sodium excretion with cardiovascular events in individuals with and without hypertension: a pooled analysis of data from four studies.”

The first article is based on an experiment done to examine urinary sodium excretions with cardiovascular events in individuals. The basic aim of these experiments was to devise and use such techniques by which the association between sodium intake and cardiovascular disease events can be determined. Also, it was found that Contrasted and direct sodium consumption, high sodium admission is related to an expanded danger of cardiovascular occasions and demise in hypertensive populaces (no relationship in the normotensive populace), while the relationship between low sodium

consumption with the expanded danger of cardiovascular occasions and passing is seen in those with or without hypertension.

This information proposes that bringing down sodium admission is best focused on populations with hypertension who expend high sodium diets. In this pooled investigation, we studied 133 118 people (63 559 with hypertension and 69 559 without hypertension), middle age of 55 years (IQR 45–63), from 49 nations in four vast planned reviews and evaluated 24-h urinary sodium discharge (as gathering level measure of admission). We related this to the composite result of death also, major cardiovascular illness occasions over the middle of 4•2 years (IQR 3•0–5•0) and pulse. All this experiment led to this finding that Expanded sodium admission was related to more prominent increments in systolic pulse in people with hypertension (2•08 mm Hg change for every g sodium increment) contrasted and people without hypertension (1•22 mm Hg change in every g; interaction<0•0001).

In those people with hypertension (6835 occasions), sodium discharge of 7 g/day or more (7060 [11%] of the populace with hypertension: risk proportion [HR] 1•23 [95% CI 1•11–1•37]; $p<0•0001$) Furthermore, under three g/day (7006 [11%] of the populace with hypertension: 1•34 [1•23–1•47]; $p<0•0001$) were both related with expanded hazard contrasted and sodium discharge of 4–5 g/day (reference 25% of the populace with hypertension). In those people without hypertension (3021 occasions), contrasted and 4–5 g/day (18 508 [27%] of the populace without hypertension), higher sodium discharge was not related to a danger of the essential composite result ($\geq$ seven g/day in 6271 [9%] of the populace without hypertension; HR 0•90 [95% CI 0•76–1•08]; $p=0•2547$), though a discharge of under three g/day was related with a significantly expanded hazard (7547 [11%] of the populace without hypertension; HR 1•26 [95% CI 1•10–1•45]; $p=0•0009$). (Carbone, 1414)

Summary

From all the above discussion and keeping in mind all the stats given in all of the above paragraphs, it can be concluded that high sodium is very risky. It can cause various damages to our health. Those who have the habit of high sodium intake suffer from cardiovascular events more frequently than those who don't take Sodium in a much higher amount. (Perez, 712-741)

Article 10: “Activation of the Renin-Angiotensin System Mediates the Effects of Dietary Salt Intake on Atherogenesis in the Apolipoprotein E Knockout Mouse.”

Dietary salt admission is a noteworthy determinant of the initiation condition of the renin-angiotensin-aldosterone framework. Given the vital part of the renin-angiotensin-aldosterone framework in plaque

aggregation, we examined its part in the improvement of atherogenesis related to sodium allowed in apolipoprotein E knockout mice. A month and a half of a low-salt eat less (containing 0.03% sodium) brought about a 4-overlap increment in plaque gathering in apolipoprotein E knockout mice at the point when contrasted and mice getting typical chow (containing 0.30% sodium). This was related to the initiation of the renin-angiotensin-aldosterone framework, expanded vascular articulation of attachment particles and fiery cytokines, and expanded attachment of marked leukocytes over the entire aorta on a dynamic stream examination.

These progressions were obstructed by the angiotensin-changing over catalyst inhibitor perindopril (2 mg/kg every day). A high-salt eating routine (containing 3% sodium) lessened vascular aggravation and atherogenesis related to concealment of the renin-angiotensin-aldosterone framework, albeit systolic circulatory strain levels were humbly expanded (5! 1 mmHg). Constitutive initiation of the renin-angiotensin-aldosterone framework in angiotensin-changing over chemical two apolipoprotein E knockout mice was likewise connected with expanded atherosclerosis and vascular bond, and this was constructed by a high-salt eating routine related to concealment of the renin-angiotensin-aldosterone framework. By differentiation, a low-salt eating routine neglected to enact the renin-“angiotensin aldosterone” framework additionally or to build atherosclerosis in angiotensin-changing over catalyst two protein E knockout mice. Together, this information approves a connection between salt-interceded renin-angiotensin-aldosterone framework, initiation, and “atherogenesis,” which may incompletely clarify the uncertain or dumbfounding discoveries of late observational considers, regardless of clear consequences for the circulatory strain. (de Borst, 344)

The result of these experiments showed that coursing lipids levels were raised in the greater part of the gatherings of E-KO mice. Changes in the admission of dietary sodium had no impact on lipid or glucose levels, weight picks, or bolstering conduct. The systolic circulatory strain was unobtrusively raised by high sodium eating fewer carbs (Table). Every day urinary sodium discharge was connected with the dietary sodium content in E KO mice, with the most minimal sodium discharge seen in mice on a low-salt eating routine containing 0.03% sodium and the most elevated discharge seen in mice on a high-salt eating regimen containing 3.00% sodium. The admission of dietary sodium additionally corresponded with coursing aldosterone levels, a marker of systemic RAAS enactment, with the end goal that coursing aldosterone levels were expanded in mice on a low-salt eating regimen and imperceptible (“20 g/mL) in mice on a high-salt eating routine when contrasted and those getting ordinary chow.

Summary

This experiment was based on the urinary sodium discharge of some of the mice. These were taken as samples so that the damage could be prevented. The salt-eating routine of these animals was judged by their attitude and the percentage of the particles present in their urine. It was concluded that those who have the habit of taking a high amount of salt suffer from various sort of health hazards. (Raz, 228-235)

Article 11: “Fatal and Nonfatal Outcomes, Incidence of Hypertension, and Blood Pressure Change about Urinary Sodium Excretion.”

The main purpose of this experiment was to survey whether 24-hour urinary sodium discharge predicts circulatory strain (BP) and well-being results. It was a planned populace study, including 3681 members without cardiovascular sickness (CVD) who were individuals from families that were haphazardly enlisted in the Flemish Study on Genes, Environment, and Health Outcomes (1985-2004) or in the European Project on Genes in Hypertension (1999-2001). Of 3681. It was an experiment designed in which about 3681 members followed up for a middle 7.9 years, CVD results diminished crosswise over expanding “tertiles” of 24-hour sodium discharge, from 50 events in the low (mean, 107), 24 in the medium (mean, 168), and 10 in the high discharge gathering (mean, 260; $P=0.001$), bringing about individual demise rates of 4.1% (95% certainty interim [CI], 3.5%-4.7%), 1.9% (95% CI, 1.5%-2.3%), and 0.8% (95% CI, 0.5%-1.1%).

In multivariable-balanced investigations, this opposite affiliation held noteworthiness ($P=0.02$): the HR in the lower ones was 1.56 (95% CI, 1.02-2.36; $P=0.04$). Standard sodium discharge anticipated neither aggregate mortality ($P=0.10$) nor lethal joined with nonfatal CVD occasions ($P=0.55$). Among 2096 members followed up for a long time, the hazard of hypertension did not increment crosswise over expanding “tertiles” ($P=0.93$). Occurrence hypertension was 187 (27.0%; HR, 1.00; 95% CI, 0.87-1.16) in the low, 190 (26.6%; HR, 1.02; 95% CI, 0.89-1.16) in the medium, and 175 (25.4%; HR, 0.98; 95% CI, 0.86-1.12) in the high sodium discharge gathering. In 1499, members followed up for a long time, and systolic blood weight expanded by 0.37 mm Hg for each year ($P=0.001$), though sodium discharge did not change (-0.45 every year, $P=0.15$).

Conclusion:

Be that as it may, in multivariable-balanced investigations, a 100-mmol increment in sodium discharge was related to a 1.71 mm Hg increment in systolic pulse ($P=0.001$), yet no change in diastolic BP. In this populace-based associate, systolic circulatory strain, however not diastolic weight, changes after some time lined up with the change in sodium discharge, yet this affiliation did not convert into a higher danger of hypertension or CVD entanglements. Bringing down sodium discharge was related to higher CVD mortality. 5.06 mm Hg for systolic and 2.70 mm Hg for diastolic among patients with hypertension, and 2.03 mm Hg also 0.99 mm Hg, separately, among members without hypertension. Three late reports,⁵⁻⁷ utilizing measurable displaying in light of the previously mentioned proof and an arrangement of complex presumptions, inferred that unassuming decreases in dietary salt admission, in the request of 3 g/d, could considerably decrease cardiovascular occasions and medicinal expenses. However, these projections ⁵⁻⁷ ignored the irregularity of the relationship between blood pressure and various indexes of salt allowed in observational studies,^{8,9} the sweeping

extrapolations from short-term medication trials in normotensive or hypertensive volunteers to the general population, 10 the possible adverse impacts of an aimless decrease of salt intake,¹¹ and the farfetched plausibility of a summed up confinement of salt consumption.^{12,13} The assumption that lower salt admission would be over the long haul lower circulatory strain, as far as anyone is concerned, has not yet been affirmed in longitudinal populace-based reviews. We tended to these issues by arbitrarily choosing European population samples. We studied the incidence of mortality and morbidity and the rate of hypertension in connection to 24-hour urinary sodium discharge at the gauge. We analyzed in cross sections and longitudinally the relationship between circulatory strain and 24-hour urinary sodium. (Mente, 465-475)

Article 12: “Sodium Intake and All-Cause Mortality Over 20 Years in the Trials of Hypertension Prevention.”

This review analyzed the connection between very much portrayed measures of sodium admission evaluated from urinary sodium discharge and long haul mortality. Two trials, stage I (1987 to 1990), more than a year and a half, and stage II (1990 to 1995), more than 36 months, were embraced in TOHP (Trials of Hypertension Prevention), which executed sodium decrease mediations. The reviews incorporated various 24-hour pee tests gathered from pre-hypertensive grown-ups 30 to 54 years old during the trials. Post-trial events were found out over the middle of 24 years, utilizing the National Death Index. The relationship between mortality, what's more, randomized mediations, and also with normal sodium admission was analyzed. Comes about Among 744 stage I and 2,382 stage II members randomized to sodium decrease or control, 251 events happened, speaking to a non-significant 15% lower chance in the dynamic mediation (danger proportion [HR]: 0.85; 95% certainty interim [CI]: 0.66 to 1.09; $p = 0.19$). Among 2,974 members not allocated to a dynamic sodium mediation, 272 events happened. There was a direct straight relationship between normal sodium admission and mortality, with an HR of 0.75, 0.95, and 1.00 (references) and 1.07 ($p = 0.30$) for <2,300, 2,300 to <3,600, 3,600 to <4,800, and $\geq 4,800$ mg/24 h, separately; and with an HR of 1.12 for each 1,000 mg/24 h (95% CI: 1.00 to 1.26; $p = 0.05$). There was no confirmation of a J-molded or nonlinear relationship. The HR per unit increment in sodium/potassium proportion was 1.13 (95% CI: 1.01 to 1.27; $p = 0.04$).

Conclusion

We found an expanded danger of mortality for high-sodium admission and an immediate association with adding to mortality, even at the lowest levels of sodium admission. These outcomes are reliable with the advantage of lessened sodium, and sodium/potassium allows for aggregate mortality over a 20-year time frame. (O'Connell, 612-623)

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