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Review Article

Lowering Effect of Potassium on Hypertension

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ABSTRACT

Hypertension is the blood pressure (BP) that is higher than the normal values in the arteries. Dietary changes and other lifestyle adjustments are essential to its management. Potassium has become known as a crucial Mineral with potential blood pressure-lowering effects among the many dietary variables. The objective of this writing is to summarize the most recent research on the connection between potassium intake and hypertension. Multiple investigations have revealed an inverse association between blood pressure and potassium intake. In addition to increasing vasodilation, lowering sodium reabsorption, and improving renal function, potassium also has antihypertensive effects. Additionally, studies have demonstrated that eating diets high in potassium increase endothelial function, lessen arterial stiffness, and lessen sympathetic nervous system activity. According to current research, increasing potassium intake, primarily from dietary sources such as fruits, vegetables, and legumes, is an effective way for managing hypertension. Individual differences, potential drug interactions, and the need for additional research to determine the best consumption levels should all be taken into account. Overall, incorporation of potassium-rich foods in the diet shows promise as a natural strategy to lower blood pressure and mitigate the symptoms of hypertension.

INTRODUCTION

Hypertension is a major global public-health concern because of its high prevalence and associated risks of cardiovascular and renal disease [1]. Because of the great frequency of chronic diseases and hypertension in most nations, it is the single most major cause of morbidity and mortality worldwide [2]. It is also known as a "silent killer" and a global chronic, noncommunicable disease due to its high death rates and lack of early signs [3]. This situation is anticipated to worsen over the next few decades as the world's population grows and ages [4]. Hypertension is defined as high blood pressure in the arteries of the body. A popular way to describe blood pressure is the ratio of systolic blood pressure (the pressure when the heart contracts) to diastolic blood pressure (the pressure when the heart relaxes). The blood pressure levels that determine hypertension change depending upon the assessment procedure. There are numerous causes of hypertension. The vast majority of patients (90-95%) have primary hypertension caused by a complex interaction between genes and the environment. A favorable family

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history is common among hypertension patients, with heritability estimated to be between 35% and 50% in most of the investigations. GWAS have identified 120 loci linked to BP regulation, accounting for 3.5% of phenotypic variance[5].

Table 1: Blood Pressure Ranges[6]

Blood Pressure Levels	Systolic (mm Hg)	Diastolic (mm Hg)
Regular	< 120	< 80
Increased	120-129	< 80
Stage 1 hypertension	130-139	80-89
Stage 2 hypertension	≥140	≥90
Hypertensive emergencies (need immediate medical attention)	> 180	> 120

Prevalence

The global significance of hypertension is becoming increasingly recognized, making hypertension a critical component of the present epidemiological change. Many developing countries' health characteristics are changing dramatically. Life expectancy in these countries is rising, and people are becoming more prone to age-related disorders such as hypertension and cardiovascular diseases (CVDs)[2]. One in every three adults in developing countries has hypertension, and at least half are ignorant of their condition. According to the most current National Health Survey results from 2012, Only 11.2 million of Mexico's 22.4 million hypertensive individuals have a health diagnosis; 8.2 million are receiving medical therapy; and only 5.7 million have their bp under control[7]. Mexico has 26 million hypertensive people, with 481,368 "new" cases reported in 2000 and a death rate of 11.8 in the same year [8]. In 2000, hypertension affected 26.4% of the world's young people, with 29.2% expected to have hypertension by 2025[1]. In 2010, the overall age-standardized incidence of hypertension in adults aged 20 was projected to be 31.1% [9]. Hypertension is a major healthcare risk in India. It is responsible for 57% of all stroke- related fatalities in India, as well as 24% of all heart disease-related deaths. A 2-mmHg drop in population blood pressure (BP) is projected to mitigate 151,000 strokes and 153,000 coronary heart disease deaths in India [10]. In accordance with the National Health Survey of Pakistan (NHSP), over 18.9% of Pakistanis around 15 and over have high blood pressure. Males had a hypertension incidence rate of 24.99%, while females had an incidence rate of 24.76%; the incidence of hypertension was higher in the urban population (26.61%) than in the rural population (21.03%).The prevalence of hypertension grew with time, from 19.55% in 1990 to 1999 to 23.95% from 2000 to 2009 and 29.95% from 2010 to 2017 [3]. Punjab had the highest rate of hypertension of 49.3%, Sindh had 46.4%, Baluchistan had 41.1% and KPK had 33.4% [11]. Disease prevention has historically been considered the best approach for promoting health and lowering disease rates. Regardless of economics, many people believe that the health rationale is compelling enough to invest in prevention [12]. The PAHO estimates that the entire cost of hypertension in some countries is in the range of 2% of the country's GDP[8]**.** In the case of Mexico, the economic cost of hypertension increased by 24% between 2010 and 2012. Using 2011 as the base year, the total cost of hypertension was $5,733,350,291[7]. In 2007, hypertension medication accounted for $68 billion in health-care expenditures in the United States, while hospitalization charges for hypertension-related admissions reached $113 billion in 2008 US dollars[13].

Non-pharmacological therapy

Adequate hypertension therapy can significantly lower the risks of stroke, myocardial infarction, chronic renal disease, and heart failure[2]. The World Heart Federation and the Lancet Commission on Hypertension both emphasized the significance of raising awareness of high blood pressure as a critical step towards addressing the related health burden [4]. Effective hypertension population control necessitates an increase in knowledge (among both health professionals and the general public), an assessment of total absolute CVD risk, and an increase in the efficacy of non-pharmacological and pharmacological therapies [2]. Lowering blood pressure with commonly used medications that are diuretics, angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, and calcium channel blockers has been demonstrated to lower the likelihood of CVD and overall death rate. According to a large meta-analysis of 123 research studies including 613,815 patients, the relative risk reduction for CVD and fatalities from all causes was proportional to the degree of blood pressure drop obtained [14]. Given the fundamentally comparable correlations of blood pressure to the risks of CHD and stroke, it is expected that SBP and DBP will be strong independent predictors of the risk of all cardiovascular illnesses [15]. Non-pharmacological therapy or primary prevention through lifestyle modifications such as increased exercise, a healthy weight maintenance, restricted alcohol use, limited smoking, reduced sodium consumption, and increased dietary potassium [9]. And the DASH diet resulted with better blood pressure reduction [16]. Excess salt consumption raises blood pressure (BP) that is an important predictor for cardiovascular disease, as well as stomach cancer, the leading cause of death globally [17]. Positive patterns regarding blood pressure, sodium consumption, and hypertension therapy and management have been observed in North Karelia, and subsequently in other regions of Finland, since the 1970s. Salt intake in Finland was rather high in the latter part of the 1970s, although it fell significantly in both genders until 2007.

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Between 1992 and 2012, the proportion of people with hypertension decreased somewhat in men but remained constant in women [18]. The magnitude of the risk associated with hypercholesterolemia is comparable to that associated with hypertension. Despite the fact that both of these disorders are recognized as significant risk factors for CHD [19]. The Framingham study found that 78% of men and 82% of women with hypertension had a minimum of one extra risk indicator [20]. Egan examined three National Health and Nutrition Examination Surveys (NHANES) reports (1988-1994, 1999-2004, and 2005-2010) and discovered that 60.7-64.3% of hypertension patients also had hypercholesterolemia. From 1988 to 2010, concomitant hypertension and hypercholesterolemia developed gradually; hypertension and elevated LDL climbed from 5.0 to 30.7%, and combined hypertension, LDL, and HDL increased from 1.8 to 26.9%. The data imply that treating both hypertension and hypercholesterolemia effectively will cut CHD by 50% and lower residual risk reported when just hypertension is treated [21]. Regular exercise is commonly recommended as a vital behavioral adaptation that can aid in the prevention of hypertension [22]. According to animal research, aerobic activity may lower blood pressure via enhancing insulin tolerance and autonomic nervous system function [23] although resistance exercise may lower blood pressure by increasing vasoconstriction control [22]. As a result, there is a greater interest in lifestyle changes, such as aerobic exercise, for the treatment and prevention of hypertension. Several meta-analyses and systematic reviews have been conducted to look into the relationship of exercise with blood pressure [24].

A potassium-rich diet to prevent and treat hypertension

The impact of nutrition on blood pressure is a public health problem. According to the Global Burden of Disease Study, in 2010, the three most important hazards for worldwide medical load were hypertension (7.0% of all disability-adjusted lifespans), cigarette use (6.3%), and alcohol misuse (5.5%). In 2013, the two risk variables were nutritional hazards and high systolic blood pressure, resulting in more than 7.5% of disability-adjusted life years [18]. Low blood pressure (BP) is hypothesized to be connected with increased dietary potassium intake. Whether potassium supplementation is useful as an antihypertensive agent [25]. It is crucial to emphasize that a high-potassium diet should not only be administered after hypertension has developed, but it can also be advantageous in the normotensive condition and should be mandatory within the high-normal range of blood pressure. Furthermore, as with any other blood pressure control technique, increased potassium consumption should be sustained over time, assuming normal renal function. The use of certain antihypertensive drugs does not prevent the consumption of a high-potassium diet [26].

Nutritional physiology of potassium

K+ is the most common component in the human body. Its content in cells fluctuates at 150 mmol/l, that's roughly 30 times greater than the concentration in plasma (3.5-5 mmol/l) and accounts for 99% of the total body's potassium. The small extracellular potassium pool is influenced by external consumption, endogenous distribution (the preservation in the liver and muscles), and elimination. In healthy people, roughly 90% of dietary potassium is taken up by the body, with the remainder eliminated through the kidneys. The proximal tubule reabsorbed most of the potassium. The cortical collecting duct, on the other hand, regulates potassium excretion by the kidney. Figure 2 shows the mechanism. Distal salt delivery (and thus dietary sodium consumption) and blood aldosterone concentration are two factors that influence potassium secretion. Other factors that influence potassium secretion include urinary flow rate, acid-base balance, and adaptability to a potassium-rich meals [27]. Figure 1 shows that the proximal tubule absorbs a substantial amount of filtered K+, mostly via the paracellular pathway driven by solvent drag. The change in lumen potential from -ve to +ve in the proximal tubule is another driving factor for K+ reabsorption. The Na+-K+-ATPase transports K+ into the intracellular compartment and exits via a conductive route on the basolateral surface. As Na+-coupled glucose and amino acid reabsorption depolarizes the cell, a K+ channel on the proximal tubule's terminal side aids in cell voltage stability [28].

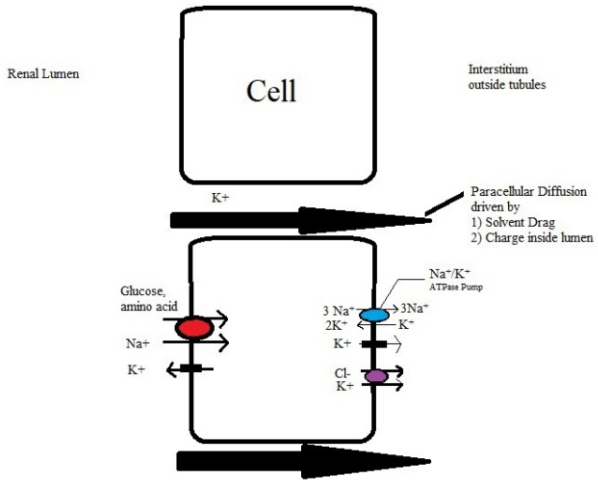

Figure1: Factors affecting K^+ movement inside and outside the proximal tubules of nephrons

The primary roles of potassium are to maintain: 1) cell membrane potential at rest and 2) intracellular osmolarity. Deviations in serum potassium levels, such as hypo- or hyperkalemia, are linked to plasma membrane hyper- or

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depolarizations, which may give rise to a disruption in muscle- and nerve- cell stimulation, with the myocardium being particularly susceptible [29]. It can also be caused by bad dietary habits, which can result in hypokalemia or hyperkalemia, both of which are harmful to the human body or health. The numerous components that are involved in the regulation of potassium secretion and reabsorption are illustrated in the Table 2.

Table 2: Factors Influencing Potassium Secretion and Reabsorption

Change	Potassium secretion		Potassium reabsorption	
	Homeostatic	Contra-homeostatic	Homeostatic	Contra-homeostatic
Increases effect	Aldosterone potassium loading in the presence of hyperkalemia	Enhanced luminal flow rate Enhanced delivery of luminal sodium Reduced luminal chloride Fludrocortisone, diuretics, and exogenous mineralocorticoid agonists Alkalosis of the Metabolism	Potassium restriction and depletion Progesterone	Acidosis Exogenous mineral-ocorticoid agonists (e.g., fludro-cortisone)
Decreases effect	Restriction and depletion of potassium	Reduced luminal flow rate Lower luminal sodium delivery Amiloride, triamterene, trimethoprim, pentamidine, and digitalis are examples of drugs that limit sodium absorption. RAAS inhibitors* Potassium channel inhibitors and additional causes, such as metabolic acidosis, cyclooxygenase inhibitors (NSAIDs), and calcineurin inhibitors	Potassium loading Tissue kallikrein	Inhibitors of RAAS

RAAS inhibitors include aldosterone synthesis inhibitors (e.g., heparin), renin secretion inhibitors (e.g., beta-blockers, cyclooxygenase inhibitors), direct renin inhibitors (e.g., aliskiren), angiotensin-converting-enzyme inhibitors (e.g., captopril), and angiotensin II receptor blockers [30].

Potassium intake

The Food and Nutrition Board of the Institute of Medicine suggests that adolescents and adults consume 4700 mg of potassium per day (National Academies of Sciences, Engineering, and [31]. NHANES a representative cohort 2003-2006, only 3% of Americans met the sufficient consumption [32]. The World Health Organization (WHO) advises that individuals consume at least 90 mmol/day (3.5 g/day) of dietary potassium in order to lower blood pressure and the incidence of CVDs, cerebrovascular events, and coronary heart disease. There is no substantial difference in flavor and taste between potassium-enriched salt and ordinary salt, according to current studies [33]. Some potassium rich sources are mentioned in Table 3.

Table 3: Potassium rich food sources

Food items	mg Potassium/100g	Resources
Banana	499	[34]
Zucchini	177	[35]
Tomato	244	[36]
Cauliflower, raw	328	[37]
Potato, cooked in skin	443	[38]
Broccoli, raw	370	[39]
Cow's milk, 3.5 % fat	382	[40]
Avocado	340	[41]
Watermelon juice	112	[42]
Coconut water	247	[43]
Black beans, cooked	306	[44]
Pumpkin	304	[29]
Apricot	132	[45]
Sweet potato, baked in skin	450	[46]
Spinach, baby boiled	950	[47]
Brussel sprouts	389	[48]
Yogurt, plain, whole milk	352	[49]
Chickpea	1150	[50]

Potassium and blood pressure

According to the American Heart Association, increasing potassium intake would reduce hypertension by 17% and enhance longevity by 5.1 years. Furthermore, in a 10-day study of hypertension patients, a low-potassium diet (16 mmol/day) improved average blood pressure by 6 mmHg compared to a high-potassium diet (96 mmol/day) [51]. Several research have been published on the effects of dietary potassium deficiency or supplemental intake on blood pressure effects in hypertensive and healthy individuals. These trials were summarized in three meta-analyses. In 1991 researchers included 19 trials with oral potassium supplementation in their meta-analysis in 1991, totaling 586 individuals (412 hypertensive along with 174 healthy subjects). Blood pressure was reduced by 5.9/3.4 mm Hg with an average potassium supplementation of 86 mmol/day over a 39-day period [27]. Whelton and colleagues conducted the largest meta-analysis, which comprised 33 trials and 2,609 participants ranging in age from 18-79 years. 12 studies were undertaken in healthy persons and 21 trials in hypertension patients, with patients receiving antihypertensive medication simultaneously in four trials. The overall pooled estimates of potassium supplementation's effects were 4.4/2.4 mm Hg [52]. A meta-analysis of 27 potassium studies was conducted by researchers that lasted at least two weeks. Potassium intake increased by 44 mmol/day on average, with a significant blood pressure change of 2.42/1.57 mm Hg [27]. Dickinson and his colleagues conducted a meta-analysis on five potassium trials over the course of eight

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weeks in 2006. Over 100 mmol/d of potassium chloride, 48-120 mmol/d of potassium citrate and bicarbonate, and 120 mmol/d of potassium citrate and bicarbonate were added to the average diet. The blood pressure was reduced by 11.2/5.0 mm Hg [53] (Table 4).

Table 4: Summary of Meta-Analysis of Potassium Trials

Meta-Analysis	Number of Trials	Interventions	Average Duration	Average BP Lowering (SBP/DBP) mm Hg
Cappucio, 1991	19	100 mmol/d diet, 48–120 mmol/d potassium chloride, 66 mmol/d potassium Gluconate + citrate	39 Days	5.9/3.4
Whelton, 1997	33	potassium chloride, 120 mmol/d potassium citrate + Bicarbonate	5 weeks	4.4/2.4
Geleijnse, 2003	27	44 mmol/d supplement (form not given)	2 weeks	2.42/1.57
Dickinson, 2006	5	>100 mmol/d diet, 48–120 mmol/d potassium chloride, 120 mmol/d potassium citrate + Bicarbonate	8 weeks	11.2/5.0

BP stands for blood pressure; CI is for confidence interval; DBP stands for diastolic blood pressure; and SBP stands for systolic blood pressure. 39.09 mg are equal to one Meq or mmol of K+, Potassium gluconate, potassium citrate, potassium bicarbonates are all the different forms of potassium [54]. Observational studies and clinical investigations have linked elevated potassium levels to lower blood pressure. Each of the above meta-analyses reveal an association between potassium consumption and blood pressure lowering. Potassium intake must be improved since it manages the nervous system, cardiovascular system, and reduces the number of platelets. According to the findings of the INTERSALT study, consuming potassium (independent of salt) is a significant predictor of population blood pressure. A further investigation found that elevating potassium consumption from 750 to 1000 mg/day may decrease blood pressure around 2 to 3 mmHg. Consuming 2.35-3.9 g of potassium per day has been shown to lower blood pressure by 4.4/2.5 mmHg [55]. It should be noted that several studies were brief in duration and had just a few of subjects.

Potassium homeostasis mechanisms

Earlier research demonstrated that maintaining potassium homeostasis in the face of excess dietary intake/ potassium-rich diet was assisted by a blocking impact of potassium on sodium resorption in the thick ascending limb and proximal tubule (part of renal nephron), allowing for raised supply of sodium and flow of urine to mineralocorticoid-responsive areas of the distal nephron. According to new research, this process is standardized, with the distal convoluted tubule (DCT) acting as a renal potassium sensor. Electroneutral sodium and chloride transport in the proximal DCT (DCT1) is reduced by high potassium consumption. Increased salt supply and flow of urine, together with elevated aldosterone, stimulate electrogenic potassium release through the ROMK (renal outer medullary potassium) channel. Aldosterone and urine flow via the Maxi-K channel both increase potassium secretion. A rise in secretion can be initiated by the introduction of potassium into the gastrointestinal tract, which suppresses Na/Cl-cotransporter (NCC) function irrespective of an alteration in plasma levels [54].

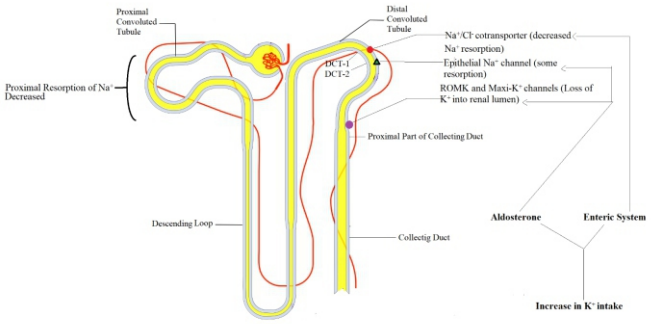

Figure 2: How homeostasis of potassium is done by ingesting potassium-rich diet [56]

Blood pressure lowering mechanism of potassium

Sodium and potassium balance is critical in endothelial-dependent vasodilation. Sodium retention reduces nitric oxide synthesis, an arteriolar vasodilator produced by endothelial cells, and raises blood levels of asymmetric dimethyl l-arginine, an endogenous inhibitor of NO production. The effects of sodium limitation are inverse [57]. Endothelium-dependent vasodilation is produced by hyperpolarizing the endothelial cell via sodium pump activation and potassium channel opening in response to a potassium-rich food and elevations in blood levels of potassium (even within the normal range). Endothelial hyperpolarization is transferred to smooth muscle cells in the vascular system, resulting in decreased cellular calcium and, thus, vasodilation. Potassium deficiency affects endothelial- dependent vasodilation in experiments [58]. Natriuresis, changes in sodium levels in cells and tonicity, baroreceptor sensitivity regulation, decreased vasoconstrictive sensitivity to norepinephrine and angiotensin II, elevated serum and urinary kallikrein, increased sodium/ potassium ATPase activity, and changes in DNA production and proliferation in vascular smooth muscle and sympathetic nervous system are all proposed mechanisms for potassium's impact on blood pressure [59]. Potassium can also enhance blood flow by inhibiting the sympathetic nervous system and boosting the absorption of norepinephrine into sympathetic nerve terminals. High potassium consumption, for example, prevented increased sympathetic vasoconstriction in

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juvenile salt- sensitive Dahl rats. As a result, potassium may have sympatholytic effects that protect the cardiovascular system from long-term harm [29] (Figure 3).

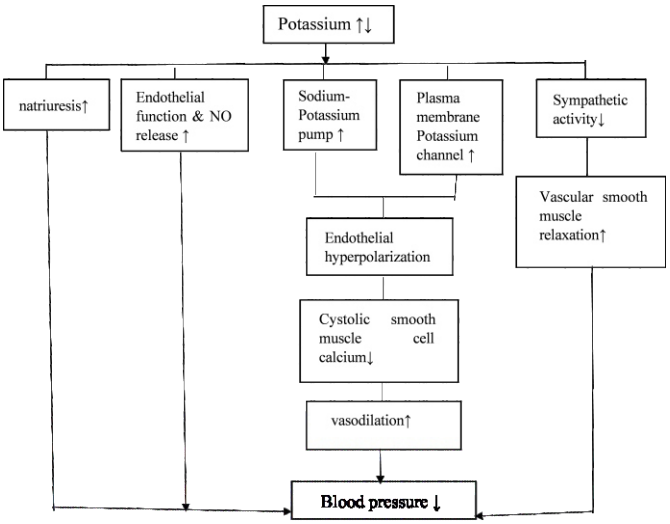

Figure 3: Mechanisms proposed for potassium's positive effects on blood pressure [29, 60]

CONCLUSIONS

In conclusion, the evidence suggests that non-pharmacological therapy such as increased dietary potassium consumption coupled with increased exercise, a healthy weight maintenance, restricted alcohol use, limited smoking and reduced sodium consumption may substantially help reduce hypertension. Numerous studies have demonstrated that increasing potassium consumption has a good impact on blood pressure regulation in a multitude of ways, as a natural vasodilator which relaxes and widens blood vessels, lowering blood flow resistance and blood pressure. It helps to reduce the harmful effects of excessive sodium ingestion by increasing sodium excretion. By limiting fluid retention and decreasing the volume of blood in circulation, it improves cardiovascular health and lowers the risk of hypertension by increasing endothelial function, increasing arterial compliance, and decreasing oxidative stress. Health organizations and medical practitioners should recommend increasing potassium consumption through a diet high in potassium-rich foods, such as fruits, vegetables, nuts, and legumes, in a controlled proportion, as a non-pharmacological means of treating and avoiding hypertension.

Authors Contribution

Conceptualization: AA

Writing-review and editing: MH, AR, AA, MS, HF, FA, TR, SA, ML

All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

Source of Funding

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