Optimal Dietary Strategies for Reducing Incident Hypertension

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Hypertension is a heterogeneous disease process in which both genetic and environmental factors play an important role. Historically, one of the most important environmental determinants is dietary sodium intake, consumed mostly as sodium chloride (salt). In humans, excessive dietary sodium intake is etiologically related to the epidemics of prehypertension and hypertension. Moreover, excessive dietary sodium intake has been associated with left ventricular hypertrophy and alterations in the structure and function of large arteries and renal vascular beds, in part, independent of the effect of sodium on blood pressure. There is also evidence that dietary sodium may affect arteriolar stiffness and, ultimately, systolic and pulse pressures. However, this is not true in all patients and may be related to not only genetic variations and how sodium is handled by the kidneys but also other dietary nutrients that may, in part, offset the effects of dietary sodium.

The study by Zhang et al suggests that another important environmental determinant of hypertension is dietary animal protein intake. The authors analyzed data from a prospective study of 87293 nurses followed for 14 years and provide interesting new information that a diet with a higher net acid load was independently associated with increased risk of incident hypertension. This association remains significant after controlling for dietary factors, such as sodium, magnesium, calcium, folate, protein, and potassium. Thus, we may have 1 more important factor to consider when treating hypertension: diet-dependent net acid load. These observations are certainly not without foundation, because other cohort studies have noted that patients with hypocitraturia or increased serum and anion gap are more likely to have higher levels of blood pressure.

The blood pressure–lowering effects of a diet rich in protein have been seen in other observational studies, as well as in clinical trials. In the OmniHeart Study, a randomized, crossover feeding study, a healthful diet where carbohydrate was replaced with protein lowered blood pressure. In part, this diet also reduced dietary salt, but it also markedly increased vegetable protein so that approximately half of the protein was from plant sources. Incidentally, the diet had also reduced animal protein intake. As a consequence, the acid protein:potassium ratio was likely reduced. Perhaps this explains the important blood pressure–lowering effects observed in this trial.

The study by Zhang et al also noted that results of analyses using the ratio of dietary animal protein:potassium paralleled observations of the relationship between the diet-dependent net acid load and incident hypertension. They theorized that these 2 measures showed similar results because there may be less between-person variability in vegetable protein intake as opposed to greater variability in animal protein intake. However, these considerations may not be true in all populations, and there may be other diet-related factors, such as the ratio of sodium:potassium and the effects of uric acid, that may alter this relationship. These factors were not considered in the analysis by Zhang et al.

The magnitude of the association between urinary potassium and blood pressure has been shown to be greater than that between urinary sodium and blood pressure. In the study by Hedayati et al, dietary potassium deficiency was independently associated with increased blood pressure in a multiethnic, population-based cohort study. The association was stronger in blacks than nonblack counterparts and was independent of demographics, estimated glomerular filtration rate, and cardiovascular risk factors. Additional evidence about the role of potassium comes from research by Xie et al, which suggests that potassium deficiency increases serine-threonine (with-no-lysine [K]) kinase (WNK-1) expression, which may create an imbalance between the activity between the renal outer medullary potassium channel (ROMK) and the sodium epithelial channel, which results in greater sodium retention. Perhaps the ratio of dietary sodium:potassium may be relevant as a dietary maneuver to reduce blood pressure.

Other diet-related factors not assessed in the study by Zhang et al study are precursors of uric acid. There are intriguing data that indicate that uric acid and hyperuricemia may be associated with incident hypertension. Although the mechanism of this relationship is unknown, there are theories that intracellular accumulation of uric acid may increase the risk of oxidative injury and proinflammatory effects. Might this be related to the development of subclinical metabolic acidosis as might occur with a diet higher in animal protein and deficient in potassium? Perhaps future evaluations of data from large, epidemiological cohort studies like the Nurse’s Health Study may provide an opportunity to explore relationships between dietary factors and uric acid. An interesting investigation would be whether excessive fructose consumption leads to higher levels of serum uric acid.
The study by Zhang et al\(^3\) adds important new information to the literature about diet and incident hypertension. Despite the study’s limitations (blood pressure was self-reported and not directly measured) and the use of food frequency questionnaires to estimate dietary intake, the large number of participants who were health professionals indicates that these results are likely trustworthy. Thus, we have one more dietary consideration (ie, protein intake) for preventing and controlling prehypertension and hypertension. These findings should be tested in randomized clinical trials to evaluate the use of these dietary manipulations that could not only reduce incident hypertension but also facilitate its treatment. Currently, total protein intake for US adults is above recommended levels, and typical US diets are composed of more animal than plant protein. In a nutrient-dense diet, plant foods can provide sufficient amounts of all of the essential amino acids and are likely to supply alkali and reduce the net acid load of the total diet. To reduce net acid load in the diet, Zhang et al\(^3\) suggest reducing dietary intake of animal protein and increasing intake of fruits and vegetables. This suggestion is not new, but now we have one more reason to adhere to this sound advice.

**Disclosures**

None.

**References**

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