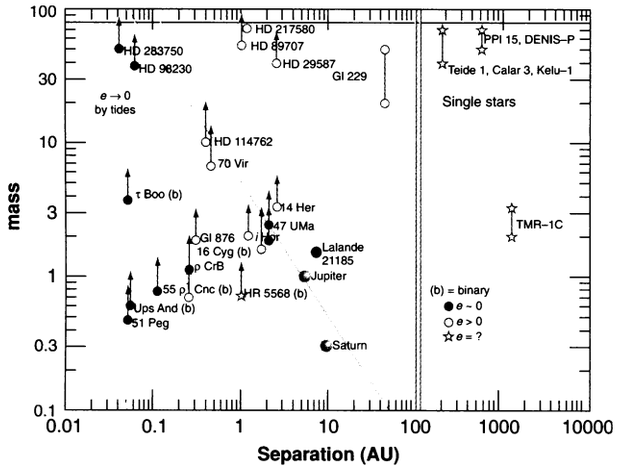

Other planets. Extrasolar planets and brown dwarfs discovered to date, shown as a function of their mass and separation from their central star. The objects to the right of the vertical double lines are not bound to any star. Eighty Jupiter masses is the dividing line between normal and brown dwarf stars. Objects detected by the radial velocity method generally have only lower limits on their masses. The oblique lines depict the approximate sensitivities of current searches by the radial velocity (light orange line) and astrometric (dark orange line) methods. The dividing line between brown dwarf stars and giant planets seems to fall at about 5 Jupiter masses. Objects to the left of the green line are so close to their primary star that they have lost all traces of any initial orbital eccentricity (*e*) because of tidal dissipation and resulting orbital circularization.

stars, whereas the giant planets found to date imply a frequency of at least 4%. The relative rarity of brown dwarf companions is consistent with the statistics for normal stars, which show that most stars have stellar companions no less than about 1/10 of their own mass; solar-type stars are about 12 times more massive than the most massive brown dwarf stars, making a solar-type star an unlikely host for a brown dwarf. The best place to find a brown dwarf star may be in orbit around another brown dwarf star. Gibor Basri (University of California, Berkeley) and his colleagues showed that the brown dwarf star PPl 15 is actually a double-lined spectroscopic binary system, the first such binary brown dwarf system discovered.

Twinned and multiple star systems are now known to be extremely common. Andrea Ghez (University of California, Los Angeles) pointed out that 50 to 80% of stars have stellar companions, with separations peaking at a distance of about 30 AU. The abundance of binary systems varies between star-forming regions, and in some regions the frequency is twice that of nearby solar-type stars. Five of the extrasolar planets discovered to date are in binary star systems—planets are not limited to single stars like the sun.

Although the rate of new discoveries has been fast and furious, the meeting underlined many gaps in our knowledge of planetary system formation and provided a sense of optimism that the next generation of telescopes will produce yet more fascinating results that will help us to understand the formation of our solar system and its relatives.

References and Notes

1. The Protostars and Planets IV conference was held at the University of California, Santa Barbara, from 5 to 12 July. Reviews presented at the meeting will be published as a part of the Space Science Series by the University of Arizona Press in 1999. The volume will be edited by V. Mannings, A. Boss, and S. Russell.
2. D. C. Jewitt and J. X. Luu, *Astron. J.* **109**, 1867 (1995).

PERSPECTIVES: BIOMEDICINE

Diet and Blood Pressure—The Paradigm Shift

David A. McCarron

Hypertension—a serious health problem for industrialized societies—contributes significantly to the risk of coronary heart disease, congestive heart failure, stroke, and kidney failure. Among vertebrates, humans are nearly unique in their natural propensity to develop elevated arterial pressure, a fact attributed to both genetic and environmental factors. Only certain highly inbred strains of rodents and genetically engineered animals also spontaneously exhibit hypertension. Public policy has generally recommended that blood pressure can best be controlled by restricton of sodium chloride intake and with medication (*1*). Recent research has, however, emphasized the powerful role of total diet in the etiology and treatment of hypertension, suggesting that the focus of current public policy regarding nutrition and blood pressure is too narrow (see News story on page 898).

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Limitation of sodium chloride in food has historically been the dietary mantra of those who set nutritional policy for hypertension. Nevertheless, the importance of salt in the pathogenesis of hypertension has long been debated (*2–4*) and remains undetermined (*5*). The intense focus on sodium began when early studies indicated that salt intake increased blood pressure. These studies, many conducted decades ago, included epidemiologic surveys in primitive societies, clinical trials in patients with kidney disease, and animal investigations in which sodium intake levels cannot be realistically extrapolated to humans (*6*). Many of these studies have since been discounted for design and methodologic flaws. But even where the methodology is sound, sodium intake cannot be linked to hypertension or higher population-wide blood pressure (*7*).

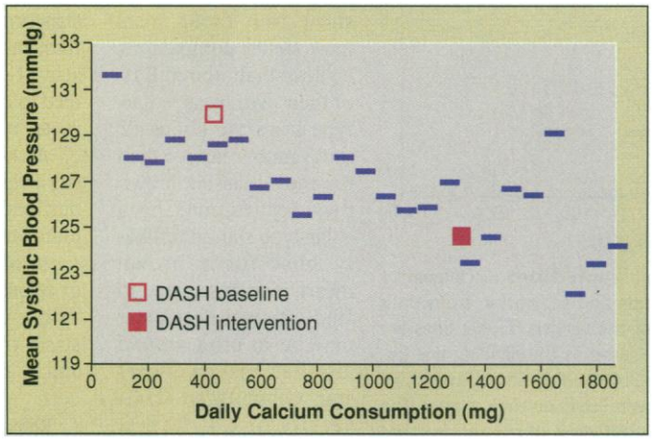
Two recent meta-analyses of randomized controlled trials—one examining the effects of sodium restriction (*8*) and the other of calcium supplementation on arterial pressure (*9*)—provide compelling evidence that adequate intake of minerals, rather than restriction of sodium, should be the focus of dietary recommendations for the general population. Assessing 56 trials of sodium restriction, Midgley *et al.* (*8*) concluded that individuals with normal blood pressure gained nothing from limiting sodium intake and that only older (>45 years) hypertensive people might benefit, a conclusion recently confirmed by other investigators (*5*). Midgley *et al.* also reported that the magnitudes of the blood pressure reductions were not consistently related to the amount of sodium intake, indicating that confounding factors were contributing to the changes in blood pressure. Indeed, in a meta-analysis of calcium supplementation trials, Bucher *et al.* (*9*) identified a small but consistent drop in blood pressure when normotensive and hypertensive persons consumed more calcium. They speculated that baseline calcium intake or increased biological need for minerals might be responsible for the blood pressure variability not otherwise accounted for in their analysis.

These two reports presaged the outcomes of two large clinical trials from the NIH, published in 1997 (*10, 11*). The Trials of Hypertension Prevention (TOHP) II is the largest and longest study ever executed

to test whether sodium restriction lowers arterial pressure and prevents the emergence of new hypertension cases (*10*). No benefit was detected for the primary endpoint diastolic blood pressure. Systolic blood pressure decreased minimally (0.7 mmHg), almost precisely the value that the Midgley (*8*) analysis projected. Furthermore, the TOHP II data demonstrated a dissociation between the extent of sodium restriction and the observed blood pressure reduction.

The second large-scale study was the Dietary Approaches to Stop Hypertension (DASH) trial published in the *New England Journal of Medicine* (*11*). As would be predicted by Bucher *et al.* (*9*) in their meta-analysis of calcium studies, in persons whose intake of dairy products (and therefore calcium and other minerals) was well below currently recommended levels, blood pressure decreased significantly when the recommended amounts were included in their diets. In the DASH diet that was rich in dairy products, fruits, and vegetables and lower in fat (with sodium held constant), blood pressure decreased 5 to 6 mmHg in subjects with normal blood pressure; in those with mild hypertension, this blood pressure reduction was doubled, to 11 to 12 mmHg. Reductions of this magnitude have been observed previously only with antihypertensive medications. A second DASH diet, which included increased amounts of fruits and vegetables but did not include dairy products, produced more modest but still significant systolic blood pressure reductions, easily surpassing those observed with sodium restriction in TOHP II.

DASH confirmed the meta-analyses as well as earlier indications from observational studies that dietary factors other than sodium markedly affected blood pressure (*3, 12*). For example, one of the earlier studies (*12*) identified inadequate calcium intake from dairy products as the dietary pattern most prevalent in individuals with untreated hypertension. Another study (*3*), in which nutrient intake was assessed from the first National Health and Nutrition Examination Survey (NHANES I), confirmed this dietary pattern in hypertensive individuals and identified the relative absence of fruits and vegetables in the American diet as the second best predictor of elevated blood pressure. These studies suggested that where intake of other critical nutrients is adequate, sodium intake at levels typically consumed in most societies might actually be associated with lower blood pressure. They also concluded that the absence of specific nutrients (calcium, potassium, and magnesium), resulting from low consumption of dairy products, fruits, and vegetables, is associated with hypertension in Americans (*3, 12*).



Can calcium in the diet lower blood pressure? The relationship between dietary calcium intake and systolic blood pressure, assembled from data in (*2*) and (*10*).

The TOHP II study adds to the substantial body of literature that challenges the public health emphasis on sodium restriction as the primary nutritional means of improving blood pressure. The issue is further complicated by concerns raised in several recent reports (*5, 13, 14*) that the long-term effect of sodium restriction on cardiovascular morbidity and mortality may be the opposite of what has always been assumed. The DASH study provides a clear rationale for focusing our public health strategy on adequate intake of low-fat dairy products and fruits and vegetables.

The consistency of the accumulated data is evident when the blood pressure changes seen with the DASH diet (*11*) are superimposed on the blood pressure profile of Americans as a function of calcium intake from (*3*) (see the figure). According to these data (*3, 11*), if the intakes of calcium and other nutrients obtained through low-fat dairy products, fruits, and vegetables were increased to the amounts readily achieved in the DASH study, the percentage of Americans with moderately severe hypertension (160/100 to 179/109 mmHg) would be decreased by nearly half, from approximately 9% to 5%. For the millions more with borderline elevations, the benefits are likely to be at least as great.

Whether hypertension is genetic or environmental in origin, control of dietary mineral intake has a place in its management and prevention. As a society, we will not achieve the dramatic reversal in hypertensive heart disease that DASH and other studies clearly show is possible until we direct our efforts to the nutritional factors and dietary patterns that are actually relevant to this condition. In addition, diets low in fat but high in calcium, fruits, and vegetables are not prevalent in the subgroups of society at greatest risk for hypertensive cardiac disease—the elderly and African Americans. An added plus: A diet low in fat but high in calcium, fruits, and vegetables is also consistent with reduction of two other major public health problems, osteoporosis and cancer (*15, 16*).

The emphasis on sodium as the single dietary culprit is counterproductive to our significantly reducing cardiovascular risk for most of us (*5*) and diverts attention from the issues we need to address (*17*). "Food products" such as snacks and soft drinks added to our diets in recent years have supplanted nutrient-rich foods such as fruits, vegetables, and milk. This shift in dietary patterns, and simultaneous suboptimal nutrient intake, is also far more likely to contribute to the prevalence of hypertension than salt, which has always been a component of the human diet. Humans may be unique in our propensity to develop hypertension simply because we are the only species with the capacity to manipulate our diets to our own detriment.

References

1. "The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure," *Arch. Intern. Med.* **157**, 2413 (1997).
2. G. Kolata, *Science* **216**, 38 (1982).
3. D. A. McCarron, C. D. Morris, H. J. Henry, J. L. Stanton, *ibid.* **224**, 1392 (1984).
4. J. D. Swales, *Br. Med. J.* **297**, 307 (1988).
5. N. A. Graudal, A. M. Galløe, P. Garred, *JAMA* **279**, 1383 (1998).
6. M. Muntzel and T. Drüeke, *Am. J. Hypertens.* **5**, 1S (1992).
7. Intersalt Cooperative Research Group, *Br. Med. J.* **297**, 319 (1988).
8. J. P. Midgley, A. G. Matthew, C. M. T. Greenwood, A. G. Logan, *JAMA* **275**, 1590 (1996).
9. H. C. Bucher *et al.*, *ibid.*, p. 1016.
10. Trials of Hypertension Prevention Collaborative Research Group, *Arch. Intern. Med.* **157**, 657 (1997).
11. L. J. Appel *et al.*, *N. Engl. J. Med.* **336**, 1117 (1997).
12. D. A. McCarron, C. Morris, C. Cole, *Science* **217**, 267 (1982).
13. M. H. Alderman, S. Madhavan, H. Cohen, J. E. Sealey, J. H. Laragh, *Hypertension* **25**, 1144 (1995).
14. M. H. Alderman, H. Cohen, S. Madhavan, *Lancet* **351**, 781 (1998).
15. NIH Consensus Development Panel on Optimal Calcium Intake, *JAMA* **272**, 1942 (1994).
16. National Research Council Food and Nutrition Board, *Diet and Health: Implications for Reducing Chronic Disease Risk* (National Academy Press, Washington, DC, 1989), p. 665.
17. T. G. Pickering, *Arch. Intern. Med.* **157**, 596 (1997).