As with any major confounder, it is critical to adjust for these effects to demonstrate the observed relation. It may be important, however, to consider an interaction between a nutrient and aging, as nutrient intake (of, for example, calcium) and absorption frequently decrease with aging (*7*). This may be an important consideration when assessing the reasons for the increasing prevalence of hypertension in an older age group.

The intent of our article was to present an original analysis of the HANES I data base. Had we felt it was appropriate to "square" our conclusions with the abundance of population-based and experimental data suggesting that dietary sodium indeed plays an important role in hypertension, we would, of necessity, have included the now rather substantial body of newer information instead of the older information that has been the basis for the formulation of past policy. In fact, the lack of intrapopulation research indicating a positive association of sodium and blood pressure has often been noted (*8*). The most recent findings are consistent with those observed by us in HANES I. Development of high blood pressure in "salt sensitive" models of hypertension has been dissociated from the intake of sodium (*9*). In the most widely studied model of genetic hypertension, sodium restriction has resulted in growth retardation and possible acceleration of the hypertension (*10, 11*). In one of the studies (*11*), the level of sodium restriction was within the bounds currently recommended as the "safe" level of sodium reduction for the U.S. population (*12*). Finally, recently reported studies from abroad suggest no short-term benefits of moderate sodium restriction in hypertensive subjects studied under the tightest control reported to date (*13*).

We are encouraged by the acknowledgement of Feinleib *et al.* that "sufficient evidence has accrued to justify further experimental and clinical investigation of associations between dietary calcium and blood pressure." We trust that this portends a broadening from the narrow focus on sodium as the principle factor in the pathogenesis of hypertension. We hope that this new perspective will not simply encompass calcium, but will address the role of all nutrients, as well as the complicated interactions that characterize our diet. The complex issues we face in applying this information to our understanding of the pathogenesis of this common medical disorder should be a stimulus to intensify our research efforts rather than to formulate simplified and premature therapeutic recommendations to the public. Other established investigators in the research community share our perspective and have articulated it in recent public statements (*14*).

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Erratum: In the report "Cell sensitivity to gravity," by A. Cogoli *et al.* (13 July, p. 228), the legend for figure 1b should have read: "Glucose consumed by the lymphocyte cells during the experiment. The initial concentration of glucose in the medium was 1100 mg/liter; the glucose that remained in the medium after the experiment was measured by the glucose dehydrogenase method (*6*). The standard deviation of triplicate samples is shown."

Erratum: In the News and Comment article "Use of antibiotics in animal feed challenged" (12 Oct., p. 144) by Marjorie Sun, the rate of fatalities resulting from infections caused by drug-resistant *Salmonella* was incorrectly reported. The fatality rate resulting from these infections is 21 times higher than for disease caused by *Salmonella* strains that responded to conventional antibiotics. This finding was reported by Scott D. Holmberg *et al.* in *Science*, 24 Aug., p. 833.

Erratum: In two Research News articles by Arthur L. Robinson (24 Aug., p. 822; 14 Sept., p. 1137), the affiliations of three researchers were given incorrectly. Peter Smith and Thirumalai Venkatesan (24 Aug.) are with Bell Communications Research (Bellcore), not AT&T Bell Laboratories, as stated. David Hwang (14 Sept.) is also with Bellcore.