



## COMMENTARY

# Dietary changes and their effect on blood pressure: what lesson should we learn?

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The knowledge of a causal relationship between diet and blood pressure (BP) is an ancient one.<sup>1</sup> Furthermore, since the beginning of the 20th century it has been known that a reduction in salt intake<sup>2</sup> and an increase in potassium intake<sup>3</sup> would lower BP in people with hypertension. Over the past 25 years evidence that diet plays an important role in determining the level of BP within a population as well as causing a rise in BP has come from different sources—anthropological, epidemiological, intervention and migration studies, clinical trials and animal experiments. Whilst epidemiological studies are unlikely to prove a cause–effect relationship and are often limited by methodological problems, as for many studies of diet,<sup>4</sup> randomised clinical trials are the gold standard to test the efficacy and effectiveness of any intervention and are particularly suitable for studying the effects of nutrients or diets on BP levels.

The evidence available so far from randomised trials on the effect on BP of some nutrients can be summarised as follows: (a) in 32 trials on 2635 adult participants an average reduction in sodium intake of 76 mmol/day (equivalent to ~5 g of salt) significantly lowers BP in both normotensive people (by 1.9/1.1 mm Hg) and hypertensive patients (by 4.8/2.5 mm Hg);<sup>5</sup> (b) in newborn babies randomised to low-salt bottle feed for 6 months, the systolic BP rose significantly less (2.1 mm Hg) than in the control group.<sup>6</sup> After 15 years of follow-up the BP of the group exposed to a low sodium diet in the first 6 months of life remained significantly lower (3.6 mm Hg) than the control group;<sup>7</sup> (c) a reduction in salt intake of ~5 g in elderly people (over 60 yrs) significantly reduces both systolic and diastolic BP (7.2/3.2 mm Hg) and it does so in both normotensive (8.2/3.9 mm Hg) and hypertensive (6.6/2.7 mm Hg) people;<sup>8</sup> (d) in 32 trials on 2609 participants an increase in potassium intake of about 30–50% sig-

nificantly lowers BP by 3.1/2.0 mm Hg;<sup>9</sup> (e) in 28 active treatment arms from 22 trials on 1231 participants a 1 g calcium supplementation (double the average intake of Western populations) does not significantly modify BP (0.9/0.2 mm Hg).<sup>10</sup>

The results of these trials are accompanied by randomised trials where the effect on BP of changes in diet rather than nutrients have been studied. This is of particular importance since we eat foods rather than individual nutrients, so that dietary recommendations often refer to food items. In a randomised parallel-group trial Siani *et al*<sup>11</sup> studied hypertensive patients on drug therapy and good BP control. One group was given a diet enriched in potassium-rich foods (increasing potassium intake from 2.76 to 4.35 g per day) whereas the control group was followed up without special dietary recommendations. After 1 year of follow-up the intervention group had a 36% reduction in the use of antihypertensive medication without loss of BP control. More recently, the results of the Dietary Approaches to Stop Hypertension (DASH) randomised feeding trial<sup>12</sup> carried out in 459 subjects with BPs below 160/95 mm Hg at entry showed that an 8-week period of a diet rich in fruit and vegetables significantly reduces BP by 2.8/1.1 mm Hg.

In this issue of the *Journal of Human Hypertension* Grossman *et al*<sup>13</sup> report the results of a trial of the effects of combining low-sodium, high-potassium and high-calcium diets on the BP of patients with mild hypertension. Overall, 36 patients with hypertension were studied on three different dietary regimens including reduced sodium, increased potassium and increased calcium intake. The order with which these diets were administered varied. It is concluded that "... only low sodium diet may be advantageous in patients with mild essential hypertension". Let us examine the evidence on which they base their conclusions, the reliability of their results and the possible reasons for their cautious interpretation.

The allocation of subjects to one of the three dietary groups does not appear to be randomised. This explains why Group 1 is made of leaner participants

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with lower BP than the other two groups. Almost 50% of the patients were on drug therapy whilst changing their diet. This is an important confounder in assessing BP response, particularly to a low salt diet, since its efficacy varies in the presence of different antihypertensive agents.<sup>14</sup> In spite of the counselling and monitoring, it is apparent that the participants did not achieve any significant change in their diet. The low salt diet achieved a 35 mmol/day change from 183 to 148 mmol/day in contrast to an aimed level of 50 mmol/day. The high potassium diet achieved an 11 mmol/day increase in contrast to aimed levels of 30–60 mmol/day. Finally there was only an estimated increase in calcium intake of 281 mg/day in contrast with the aimed 1000 mg/day. This clearly suggests either the failure of the researchers in achieving good compliance in their study group or the inadequacy of the dietary recommendations. Taking these three points into consideration, it is not surprising that no significant changes in BP occurred in the overall sample. Inability to achieve dietary changes and the very small sample size are the likely explanations for the authors' findings. They then proceed with subgroup analyses only to discover that amongst those who complied to the low salt diet, by play of chance, some had a fall in BP (salt-sensitive) and some had not (salt-resistant). BP response to salt restriction may well be heterogeneous. However, if compliant, everyone reducing salt intake will lower BP.<sup>8</sup> Furthermore, their discussion fails to acknowledge that a reduction in salt intake can only be achieved if food items with reduced salt are made available by modifying food processing at source.

What have we learnt from yet another trial? Why would someone carry out another study, not randomised, confounded by drug therapy, with inappropriate sample size, lacking the intervention it purposed, unable to show any change in BP, to then agree with what was already known? And why would someone want to read the results of such a trial? The answers are hard to find.

Considering the evidence,<sup>15</sup> national and international scientific bodies still recommend a moderate reduction in the intake of salt in the diet and an increase in foods rich in potassium not only as a first step for the management of people with hyperten-

sion, but more importantly, as a population-based strategy for the prevention of hypertension and its related complications.

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