Diet, blood pressure and hypertension

Kjeld Hermansen

Department of Endocrinology and Metabolism, Aarhus Amtssygehus, Aarhus University Hospital, 8000 Aarhus C, Denmark

Prevention of hypertension, and control of blood pressure in patients with hypertension, are necessary for the reduction of cardiovascular morbidity and mortality. Lifestyle modifications are one of the most important tools for effective lowering of blood pressure. Most randomized controlled studies have shown that even a modest weight loss of 3–9 % is associated with a significant reduction in systolic and diastolic blood pressure of roughly 3 mm Hg in overweight people. Limitation of sodium chloride in food has historically been considered the critical change for reducing blood pressure. Changes in sodium intake do affect blood pressure in older persons and in patients with hypertension and diabetes, whereas its role in population blood pressure has proven controversial. Recent meta-analyses indicate that adequate intake of minerals, e.g. potassium and probably calcium, rather than restriction of sodium, should be the focus of dietary recommendations. Although epidemiological data point to a direct relation between the intake of saturated fat, starch and alcohol, as well as an inverse relationship to the intake of omega-3 fatty acids and protein, our knowledge about macronutrients and blood pressure is scanty. It may well prove more productive to look at food instead of placing emphasis on single nutrients. Thus the Dietary Approaches to Stop Hypertension (DASH) demonstrates that a diet rich in fruits, vegetables, low-fat dairy products, fibre and minerals (calcium, potassium and magnesium) produces a potent antihypertensive effect. Such a diet is not very restrictive and should not produce compliance problems. Further high-quality research on the influence of macronutrients and food will yield data for updated recommendations, enabling better prevention and control of the blood pressure problem.

Blood pressure: Hypertension: Diabetes: Obesity: Diet: Weight reduction: Minerals: Fat: Carbohydrate: Protein: Alcohol

Introduction

Hypertension is a major risk factor for coronary heart disease, congestive heart failure, stroke and renal disease. The incidence of hypertension becomes more prevalent with age (Burt *et al*. 1996) and hypertension is found in about 50 % of individuals above 55 years in many industrialized countries. Although the control of hypertension in Western Europe, USA, Japan and Australia has improved considerably in the second part of the 20th century, there are worrying signs that the rate of improvement has reached a plateau (Burt *et al*. 1996; Guidelines Subcommittee, 1999). Genetics plays a significant role in determining who will become hypertensive. As much as 20–40 % of blood pressure (BP) variations in the general population have been attributed to genetic factors (Ward, 1990). Also lifestyle factors such as dietary pattern are implicated as major contributors to the continued high prevalence of hypertension. A big challenge facing medical practitioners and public health authorities is the prevention and management of hypertension both in individual patients and at a population level (Anon., 1997; Guidelines Subcommittee, 1999). This should be achieved by the least intrusive means possible, including lifestyle modification, alone or with pharmacological treatment (Anon., 1997; Guidelines Subcommittee, 1999). In this context important questions to answer are: what is the evidence that dietary modifications influence BP regulation and the propensity to develop hypertension? How are weight reduction and changes in dietary sodium, potassium, calcium and magnesium intake linked to levels of BP? Does the intake of macronutrients and complex dietary changes affect BP and the risk of hypertension? I have tried to address these issues in humans, recognizing that there is no direct evidence that reducing BP through dietary measures reduces the risk of cardiovascular disease. It seems likely, however, that the benefits of BP reduction are determined primarily by the BP reduction *per se* rather than by the particular treatments.

Corresponding author: K. Hermansen, fax +45 89 49 7659, email Kjeld.Hermansen@aas.auh.dk

Weight reduction

In the nationwide Community Hypertension Evaluation Clinic (Stamler *et al*. 1978), screening of more than 1 million people showed an increase in frequency of hypertension in overweight persons aged 20–39 years and 40–64 years by 100 and 50 %, respectively. Upper-body obesity is especially associated with hypertension. A relationship between caloric restriction, weight loss and a decreased incidense of hypertension has also long been noted. Staessen *et al.* (1988) showed in a meta-analysis of randomized controlled intervention studies (RCT) conducted in obese hypertensive patients that on average each 1 kg decrease in body weight was associated with a reduction in systolic blood pressure (SBP) and diastolic blood pressure (DBP) by 1·2 and 1·0 mm Hg, respectively. In phase I of the Trials of Hypertension Prevention (TOHP I), participants aged 30–54 years who had a DBP of 80–89 mm Hg and were between 115 and 165 % of their desirable weight were randomly assigned to either an 18 months weight loss intervention ($n = 308$) or a usual-care control condition ($n = 256$) (Stevens *et al*. 1993). A significant mean net decrease in the intervention group occurred in SBP after 6 and 18 months of 3·8 and 2·9 mm Hg, respectively, and in DBP of 2·4 and 2·5 mm Hg, respectively (Stevens *et al*. 1993). Blood pressure reductions were greater for those who lost larger amounts of weight. Weight reduction of as little as 4·5 kg reduces blood pressure in a large proportion of overweight persons with high normal BP or hypertension (Trials of Hypertension Prevention Collaborative Research Group, 1997; Whelton *et al*. 1998). In TOHP II an average reduction occurred after 6 and 36 months in SBP of 3·7 and 1·3 mm Hg, respectively and in DBP of 2·7 and 0·9 mm Hg, respectively (Trials of Hypertension Prevention Collaborative Research Group, 1997). Although the effects on BP declined over time, reductions in hypertension incidence were achieved – the relative risk of hypertension at the end of the study being 0·79 (Trials of Hypertension Prevention Collaborative Research Group, 1997). The Trials of Nonpharmacological Intervention in the Elderly (TONE; Whelton *et al*. 1998) represents an attempt to discover whether a nonpharmacological approach to BP control such as weight loss is effective in older persons. The study subjects were between 60 and 80 years old, and were taking one or two antihypertensive drugs but were otherwise healthy. The average weight loss attained was 3·9 kg after 29 months. The incidence of endpoints that followed (hypertension, resumption of medication) was decreased by 25 % (Whelton *et al*. 1998). More recently, a meta-analysis encompassing 18 trials and involving 2611 overweight hypertensive persons has shown that weight-reducing diets (450–1500 kcal/d) for 2 weeks to 3 years can induce modest weight loss in the range 3–9 % of body weight (Brand *et al*. 2000). Comparison of weight-reducing diet versus non-intervention control in 361 participants suggests that such a weight loss produces an average reduction in SBP of 3·0 mm Hg and DBP of 2·9 mm Hg, respectively (Brand *et al*. 2000). However, since the pooled data do not reach statistical significance, it cannot be stated with 95 % certainty that weight reduction decreases BP (Brand *et al*. 2000). A comparison of weight-reducing diet versus antihypertensive medication showed that the latter produced a significantly greater reduction in SBP and DBP (6 and 5 mm Hg, respectively; Brand *et al*. 2000). Potential mechanisms of action of weight loss on BP are (a) hemodynamic effects via reduction in blood volume and cardiac output; (b) reduction in plasma renin activity which may be associated with a reduction in sympathetic nervous system activity; and (c) correction of hyperinsulinaemia with reduction in renal sodium retention.

Weight reduction appears to act as an effective tool in the prevention of hypertension, and may also decrease dosage requirements of antihypertensive medication in the presence of hypertension. The effect of weight loss on morbidity and mortality secondary to hypertension is not known. However it seems appropriate to prescribe to hypertensive patients who are overweight an individualized, monitored weight-reduction programme involving energy restriction and increased physical activity. On the other hand, it is unrealistic to expect a weight-reducing diet alone to achieve BP control in patients with severe hypertension or in patients unmotivated to lose weight.

Sodium intake

Epidemiological studies indicate a positive association between dietary salt intake, level of BP and prevalence of hypertension (Law, 1997). As shown in Table 1, three meta-analyses of RCT (Midgley *et al*. 1996; Cutler *et al*. 1997; Graudal *et al*. 1998) reveals that a reduction in sodium intake – over periods ranging from days to a few years – lowers BP. However, the individual BP responses to a reduction in sodium intake vary considerably among

Table 1. Meta-analyses of dietary sodium reduction (as measured by urinary sodium excretion/24 h), systolic blood pressure (SBP) and diastolic blood pressure (DBP) in hypertensive (hyper) and normotensive (control) persons

	Study					
	Cutler *et al.* (1997)*		Midgley *et al.* (1996)*		Graudal *et al.* (1998)†	
Blood pressure (mm Hg)	Hyper $n = 1043$	Control $n = 1689$	Hyper $n = 1131$	Control $n = 2374$	Hyper $n = 2161$	Control $n = 2581$
SBP	–5·7	–2·2	–3·7	–1·0	–3·9	–1·2
DBP	–2·7	–1·3	–0·9 NS	–0·1 NS	–1·9	–0·3 NS

* Decrease in blood pressure for a 100 mmol/24 h reduction in daily sodium excretion.
† Decrease in blood pressure in hypertensive (reduced sodium excretion mean 118 mmol/24 h) and normotensive controls (reduced sodium excretion mean 160 mmol/24 h).

groups. Midgley *et al.* (1996) and Graudal *et al.* (1998) concluded that reduced sodium intake in normotensive persons had no impact on DBP, and that the results did not support current recommendations for universal dietary sodium restriction. TOHP II (Trials of Hypertension Prevention Collaborative Research Group, 1997) is the largest and longest RCT ever executed to test if sodium restriction lowers BP in a normotensive group of 30–54 years. After 6 and 36 months a reduction in SBP and DBP of 2·9/1·6 and 1·2/0·7 (NS) mm Hg, respectively, was observed in response to a decrease in the average sodium excretion of 50 and 40 mmol per 24 h, respectively. Although the effects on BP declined over time, an 18 % reduction in hypertension incidence was achieved. In TOHP II, combined weight loss and sodium restriction had limited additivity on BP which may be due to difficulty in maintaining a focus on both lower-energy and lower-salt foods simultaneously. The INTERSALT study (Stamler, 1997), an epidemiological study of 10079 men and women aged 20–59 years from 32 countries, indicated that an increase in sodium intake of 100 mmol per 24 h was associated with an increase in SBP/DBP of approximately 3–6/0–3 mm Hg. In the TONE study (Whelton *et al*. 1998), carried out in persons with hypertension aged 60–80 years, a moderate reduction in sodium intake of about 40 mmol per 24 h during 29 months elicited an approximately 30 % decrease in need for anti-hypertensive medication. Interestingly, the combination of weight loss and sodium reduction in this study had an additive effect. Although an inverse association between myocardial infarction and 24-h urinary excretion of sodium has been found in men (Alderman *et al*. 1995), there is no evidence from the studies included in the three meta-analyses (Midgley *et al*. 1996; Cutler *et al*. 1997; Graudal *et al*. 1998) to indicate that lower levels of sodium intake present a safety hazard. In salt-induced hypertension in humans, accumulation of sodium and water with expansion of the extracellar volume seems to precede the development of hypertension, whether the defect is intrinsic to kidneys and/or secondary to circulating factors. Changes in sodium intake are more likely to affect BP in African Americans, hypertensive, diabetic, obese and elderly people. However science has not yet provided a clearcut answer that reveals the putative benefits and drawbacks of sodium reduction for the common population (Midgley *et al*. 1996; Cutler *et al*. 1997; Graudal *et al*. 1998; McCarron, 1998; Taubes, 1998). The present official recommendations (Anon., 1997; Guidelines Subcommittee, 1999) support a moderate sodium reduction to a level no more than 100 mmol per 24 h, i.e. approximately 6 g of sodium chloride or 2·4 g/d sodium. This can most easily be achieved by avoiding added salt, obviously salted foods and in particular processed foods which contain large amounts of sodium.

Potassium intake

Observational studies have demonstrated an inverse relationship between potassium intake and BP (Langford, 1983; Intersalt Cooperative Research Group, 1988). Analysis of 24 h urinary electrolyte excretion and BP in the INTERSALT study (Intersalt Cooperative Research Group, 1988) showed that potassium excretion was negatively correlated with BP. In the TOHP I study (Whelton *et al*. 1995), a double-blind, placebo-controlled RCT of oral potassium chloride supplementation (60 mmol per 24 h) in 353 normotensive men and women, a potassium level increased by 44 mmol per 24 h following 3 months' therapy was associated with a reduction in DBP of 1·8 mm Hg. After 6 months, however, this apparent effect had virtually disappeared. Cappuccio *et al.* (1991) carried out a meta-analysis of data from 19 studies with oral potassium supplementation involving 586 participants. Results indicated that oral potassium supplementation significantly lowered SBP (–5·9 mm Hg) and DBP (–3·4 mm Hg). The reductions in BP were greater in hypertensive than in normotensive individuals. In a more recent meta-analysis (Whelton *et al*. 1997), including 33 RCT (2609 participants) in which potassium supplementation was the only difference between the intervention and the control conditions, potassium supplementation was associated (after exclusion of one outlier trial) with a significant reduction in average SBP and DBP of –3·1 and –2·0 mm Hg, respectively. Effects appeared enhanced in studies in which participants were concurrently exposed to a high intake of sodium. In addition to the natriuretic effects of potassium, effects on vascular smooth muscle cells and adrenergic nerve terminals may be important (Haddy & Overbeck, 1976; Haddy, 1988). Although most of the clinical trial experience to date emanates from studies in which potassium was administered in pill form as chloride salt, there is little reason to suspect a different outcome after dietary supplementation from food.

Calcium intake

Low dietary calcium intake is associated with an increased prevalence of hypertension in most epidemiological studies (Allender *et al*. 1996). The pooled data from 26 RCT ($n = 1410$) assessing the effect of dietary calcium supplementation on BP (Allender *et al*. 1996) showed a change in SBP in normotensive (–0·53 mm Hg) and hypertensive participants (–1·68 mm Hg). Diastolic BP was not affected in either subgroup. A similar conclusion was reached in another meta-analysis (Bucher *et al*. 1996) including 33 RCT ($n = 2412$) focusing on the impact of calcium supplementation on BP. Calcium supplementation led to a small reduction in SBP (–1·27 mm Hg) but not DBP (Bucher *et al*. 1996). The mechanism of action of calcium administration on BP may be via natriuresis and diuresis. It is important to maintain an adequate intake of calcium for general health, e.g. as low-fat dairy products, however it is unlikely that there are important underlying effects of calcium supplementation in reducing BP in those with adequate calcium intake, whether normotensive or hypertensive.

Magnesium intake

The recent Atherosclerotic Risk in Communities study (Peacock *et al*. 1999), which included 7731 participants aged 45–64 years, free of hypertension, who were followed for 6 years, examined the relationship of serum and dietary magnesium with incident hypertension. An inverse

relationship between serum magnesium and incidence of hypertension was found; however no association existed between dietary magnesium intake and incident hypertension. This contrasts with the findings in the Multiple Risk Factor Intervention Trial (MRFIT; Stamler *et al*. 1997) where dietary magnesium was inversely related to SBP and DBP. Magnesium, like calcium, may affect BP, but the data from intervention studies with magnesium supplementation are conflicting and no convincing data currently justify recommending an increased magnesium intake to reduce blood pressure.

Macronutrients

With the exception of alcohol consumption, our knowledge about the relationships between macronutrient intake and BP regulation is lacking or deficient. This is primarily due to lack of clinical intervention studies, and even in the recent Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (Anon., 1997) very little discussion on the association between carbohydrates, fat and protein ingestion and BP perturbations is provided. One inherent problem in studying the effect of a change in a specific macronutrient is that the amount of one of the other macronutrients must be changed concomitantly to balance energy content. Consequently, the reason behind any outcome will often be dubious.

Alcohol

In MRFIT (Stamler *et al*. 1997) – a randomized, primary prevention trial conducted among more than 11 000 middle-aged men followed with nutrient data for trial years 1–6 (four or five 24 h dietary recall per man) – the alcohol intake was found to be significantly and positively related to both SBP and DBP, and a change in alcohol intake was followed by a change in BP. The relationship between alcohol intake and BP was also assessed in the INTERSALT study (Marmot *et al*. 1994) which involved 4844 men and 4837 women aged 20–59 years. There was a significant relation of heavy drinking (3–4 or more drinks per day) to BP. Men who drank >500 ml alcohol per week and women drinking >300 ml alcohol per week, as compared to non-drinkers, had an SBP/DBP of 4·6/3·0 and 3·9/3·1 mm Hg higher, respectively. Interestingly, a reduction in alcohol intake led to improved BP control (Puddey *et al*. 1987). The recommendation in JNC VI (Anon., 1997) for hypertension prevention and management is to limit daily alcohol intake to no more than 30 ml alcohol in men and 15 ml for women and lighter-weight people.

Carbohydrates

Little attention has been given to possible influence on BP regulation of amount and type of dietary carbohydrate. The MRFIT study (Stamler *et al*. 1997) showed a direct relation between starch intake and both SBP and DBP. In addition, two small clinical studies (Ahrens, 1975; Israel *et al*. 1983) suggested a small positive correlation of sugar consumption with high BP. This may be ascribed to salt and water retention and subsequent BP increase after ingestion of sugar. Furthermore, an inverse relation between fibre and BP seen in MRFIT (Stamler *et al*. 1997) stems in part from many reports that vegetarians have lower average BP (Anderson, 1983; Sacks & Kass, 1988). The studies on fibre intake and BP are, however, difficult to interpret since fibre-rich diets differ in other respects that might influence BP, e.g. are lower in energy and fat content as well as having a differing mineral content. Thus no firm conclusion can be drawn about the causal relationship between carbohydrates and BP, and further research is needed to clarify these issues.

Protein

The cross-sectional study INTERSALT (Stamler *et al*. 1996*b*) indicated that higher dietary protein intake (assessed by total nitrogen and urea nitrogen in 24-h urine) has favourable influences on BP. On average the SBP/DBP were 3·0/2·5 mm Hg lower among those eating on average 81 versus 44 g per 24-h dietary protein, respectively. Also MRFIT (Stamler *et al*. 1996*b*, 1997) supports the finding that dietary protein intake has the least association with elevated BP, but on the contrary is inversely associated to BP. This seems to present a paradox, as high protein consumption has been correlated with progression of renal damage and impaired renal function with hypertension. Furthermore, people ingesting lacto-ovo-vegetarian diets that contain low amounts of protein have low BP (Rouse *et al*. 1983; Margetts *et al*. 1985). However, if levels of dietary fats, fibre, energy, sodium, potassium, magnesium, calcium and carbohydrates were kept constant, BP was not influenced by addition of meat protein (Prescott *et al*. 1987). The potential mechanisms by which protein may reduce BP may have different causes. Thus protein may replace fats or sugars that are maintaining a higher BP. Lower BP may also be attributed to increased natriuresis, or certain of the amino acids in the dietary protein may cause vasodilation through enhanced endogenous production of nitric oxide.

Fat

Morris (1994), reviewing the evidence from cross-sectional studies (35268 participants), biochemical studies (6422 participants) and controlled clinical trials (2323 subjects), found no support for an association between average total fat intake and average BP. Some of the discrepancies among studies may be explained by differential effects of different types of fat. In MRFIT (Stamler *et al*. 1996*a*, 1997) the main findings in the multivariate analysis of the 6-year data revealed significant independent positive relations of dietary cholesterol with SBP and DBP, between dietary saturated fat and DBP as well as an inverse relation of poly-unsaturated fat/saturated fat to DBP. No difference in diurnal blood pressure was observed by us in a small RCT in response to the two quantitatively most important saturated fats, stearic and palmitic acid (Storm *et al.* 1997). A meta-analysis of 31 placebo-controlled trials (1356 subjects) showed a dose–response effect of fish oil on BP of –0·66/–0·35 mm Hg per g omega-3 fatty acids (Morris *et al.* 1993). An amount of omega-3 fatty acids ⩾3·3 g per 24 h

was needed to be associated with an effect on BP. The average reduction in BP in response to fish oil was –3·0/–1·6 mm Hg (Morris *et al*. 1993). In the Lugalawa study (Pauletto *et al*. 1996), fish consumption (300–600 g daily) was associated with raised plasma omega-3 fatty acids and lower BP. However, the effect of omega-3 fatty acids on BP appeared exclusively in individuals with hypertension, hypercholesterolaemia and atherosclerotic disease, and not in healthy normotensive individuals (Morris *et al*. 1993). The role of monounsaturated fat in BP is uncertain. In five trials in normotensive persons, no evidence of a BP-reducing effect of monounsaturated fat was seen (Morris *et al*. 1993). In a small group of hypertensive women, a diet rich in monounsaturated fat from olive oil had beneficial effects on BP (Ruitz-Gutierrez *et al*. 1996). Using ambulatory BP monitoring with repeated measurements over 24 h, we found a diet rich in olive oil to cause an average reduction in SBP/DBP of 4–5/3 mm Hg in normotensive type 2 diabetic subjects (Rasmussen *et al*. 1993; Thomsen *et al*. 1995), while such an effect could not be picked up in a group of insulin-treated type 2 diabetic subjects with microalbuminuria (Nielsen *et al*. 1995). Further studies are needed to confirm the potential of monounsaturated fat-rich diets to lower BP. A number of theories exist regarding the BP modulating effect of dietary fats. Incorporation of unsaturated fat into lipid membranes increases membrane permeability thereby stimulating the sodium and cation transport. Another explanation is that polyunsaturated fat converts to prostaglandins which reduces BP via effects on arterial vasodilatation, electrolyte balance, renal renin release and/or pressor hormones.

Complex dietary changes

As mentioned above, current guidelines recommend weight control, reduced intake of sodium, reduced alcohol consumption and increased dietary potassium and calcium as some of the nutritional approaches to control BP. Other dietary factors and overall dietary patterns, such as vegetarianism, may also have beneficial effects on BP. Changes in overall dietary habits may, however, be more valuable than supplementation with individual nutrients. Accordingly, the DASH (Sacks *et al*. 1995; Appel *et al*. 1997; Svetkey *et al*. 1999) was conducted to study the impact on BP of dietary patterns of nutrients as they occur together in food, rather than the effects of individual nutrients. In this multi-centre study, 459 adults with mean SBP/DBP of 131/85 mm Hg, respectively, were included. For 3 weeks they consumed a control diet low in fruits and vegetables (four servings per day) and dairy products (0·5 servings per day), and similar in fat content to the typical US diet. The subjects were then randomly assigned to one of three diets for 8 weeks: (a) the control diet; (b) a diet rich in fruits and vegetables (8–10 servings daily) providing potassium and magnesium approximately the 75th percentile of US consumption and 31 g fibre daily; or (c) a combination diet rich in fruit and vegetables (10 servings daily) and low-fat dairy products (three servings daily) and low in saturated fat and total fat, providing potassium, magnesium and calcium at approximately the 75th percentile of US consumption. Sodium intake and body weight were maintained at constant levels. As seen in Table 2, the DASH combination diet and the fruit-and-vegetable diet reduced SBP and DBP more than the control diet. The impact on hypertensive subjects was even greater (Table 2). The BP reduction appeared within 2 weeks of taking the combination diet.

Table 2. Influence of complex dietary changes on BP – the DASH trial (Appel *et al.* 1997): mean changes (Δ mm Hg) in BP between the combination diet (combi), the fruit-and-vegetable diet (FV) and the control diet (C)

Blood pressure	Δ Combi–C	Δ Combi–FV	Δ FV–C
SBP, all (n=459)	–5·5	–2·7	–2·8
SBP, hypertensive (n=133)	–11·4	–4·1	–7·2
DBP, all (n=459)	–3·0	–1·9	–1·1
DBP, hypertensive (n=133)	–5·5	–2·6	–2·8

Combi: a combination diet rich in fruit and vegetables and low-fat dairy product and low in saturated and total fat; high content of potassium, calcium, magnesium and fibre.
FV: diet rich in fruit and vegetables and low in dairy products; high content of potassium, magnesium and fibre.
C: control diet low in fruits and vegetables and dairy products and high in fat; low in calcium, magnesium and potassium.

Conclusions

The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (Anon., 1997) recommends the following lifestyle modifications to prevent hypertension: lose weight if overweight; limit daily sodium chloride intake to less than 6 g; maintain adequate intake of dietary potassium (approximately 90 mmol/d) and adequate intake of dietary calcium and magnesium for general health; limit alcohol intake to no more than 30 ml/d for men and 15 ml/d for women; increase aerobic physical activity (30–45 min most days of the week); stop smoking and reduce intake of dietary saturated fat and cholesterol for overall cardiovascular health. The DASH study (Sacks *et al*. 1995; Appel *et al*. 1997; Svetkey *et al*. 1999) suggests that complex dietary changes, such as in the combination diet, may have more potent effects on BP than single-nutrient supplementation or restriction. This may be ascribed to interactions and more potent effects on BP of dietary factors. Moreover, diets such as the DASH combination diet are not very restrictive and consequently may not produce major compliance problems. Our knowledge on how the three key macronutrients – protein, carbohydrates and fats – influence BP is relatively scanty. Further high-quality research on the problems discussed here will yield data for updated recommendations, enabling better prevention and control of the BP problem. In this context it is notewothy that lifestyle changes undertaken to influence blood pressure levels may have a wide range of additional beneficial effects on the metabolic aberrations related to the metabolic syndrome. This is not true for most antihypertensive drugs, which sometimes may even worsen the metabolic abnormalities. In a direct comparison of the effects on BP, this ‘added’ value of the non-pharmacological treatment should not be forgotten.

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