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Lifestyle and Hypertension

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Lifestyle factors are critical determinants of blood pressure levels operating against a background of genetic susceptibility. Excess body fat is a predominant cause of hypertension with additive effects of dietary salt, alcohol, and physical inactivity. Controlled trials in hypertensives show blood pressure lowering effects of supplemental potassium, fibre, n-3 fatty acids, and diets rich in fruit and vegetables and low in saturated fats.[64] Some population studies show an inverse relationship between dietary protein and blood pressure levels. Regular coffee drinking raises blood pressure in hypertensives. The role of "stress" remains enigmatic, with "job strain" being a possible independent risk factor for hypertension. Am J Hypertens 1999;12:934–945

KEY WORDS: **Hypertension, lifestyle, behavioral factors, blood pressure.**

Lifestyle or behavioral factors critically determine the level of blood pressure in individuals and the prevalence of hypertension in populations.[1] Multiple and as yet largely unidentified genetic factors influence individual susceptibility to different aspects of diet and lifestyle: these will determine interindividual variations in blood pressure between subjects exhibiting common behavioral patterns that promote blood pressure elevation.

In this review we focus on recent data concerning the role of body fat, alcohol consumption, dietary sodium and potassium intake, complex dietary changes including fruit, vegetables, fats, fiber, n3 fatty acids, and dietary fish consumption, physical activity, psychologic factors, and some of the interactions between them.

BODY FAT

Excess body fat is the dominant factor predisposing to blood pressure elevation in cross-sectional and longitudinal population studies. The effect is apparent from infancy and childhood[2] through to the elderly, with a continuum of effect throughout the entire distribution of body fat.[3] Body fat excess, particularly central obesity, is associated with the so-called metabolic syndrome of impairment of insulin sensitivity, glucose intolerance, and dyslipidemia, which compounds with the effects of blood pressure elevation to increase the risk of cardiovascular disease.[4] Hence lifestyle determinants of obesity, along with cigarette smoking, are critical targets for public health campaigns against heart attack and stroke.

Obesity, blood pressure, and the associated metabolic syndrome track from childhood through to adult life, as has been shown in studies such as the Bogalusa Heart Study,[5] whereas adolescents show a clustering of behaviors predisposing to cardiovascular disease.[6] This suggests the need to focus on prevention of adverse health behaviors in childhood to reduce the patterns of excess body fat and gross obesity increasingly evident in societies such as the United States and Australia, both in their general and indigenous populations, and in developing countries. The key to this seems to be strategies designed to counteract the combination of the decline in physical activity and preference for energy-dense foods.

With a view to the childhood prevention of risk of

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hypertension and cardiovascular disease in adult life, we have carried out two randomized controlled trials of physical activity and nutrition programs in 11- to 12-year-old schoolchildren in Western Australia.[7,8] Each involved about 1000 children and used year-long home and school nutrition and physical activity programs conducted by the students' usual teachers. Both studies showed the potential to reduce the prevalence of obesity and to lower blood pressure and increase physical fitness, with girls responding better than boys.[7] In the second of these studies, overweight boys and girls who were targeted with a school-based physical activity enrichment program showed substantial improvement over a year, with some improvement sustained 6 months after the program ended.[8]

In overweight adults with established hypertension, calorie restriction and concomitant weight loss of around 5 kg can rapidly lower blood pressures.[9] Weight reduction programs have been the most successful elements of North American hypertension prevention programs.[10] Effects of concomitant lifestyle changes such as physical activity and moderating heavy alcohol consumption[11] are additive, resulting in falls in blood pressure as large as those seen with antihypertensive drug therapy.

PHYSICAL ACTIVITY AND FITNESS

Population studies show an inverse relation between physical fitness and blood pressure levels independent of all other risk factors for hypertension.[12] Similar relationships are seen between physical fitness or activity and cardiovascular morbidity and mortality.[13] Randomized controlled trials of the effects of exercise training show that blood pressure falls more consistently in those with established hypertension. Meta-analyses suggest reductions of around 7 to 11 mm Hg systolic in hypertensives and 3 mm Hg systolic in normotensives,[14] although the latter changes remain in dispute[15] and one relatively large study in hypertensives showed no effect on ambulatory pressures.[16]

Combining an exercise program with weight reduction had additive effects on blood pressure reduction in one study in hypertensives,[17] whereas another, involving a factorial design in obese subjects with high normal pressures, showed weight loss to have the dominant effect on ambulatory blood pressures, but effects were more sustained throughout the 24 h when weight loss and exercise were combined[9] A third study of independent and combined effects of weight loss and exercise showed no additive effects but lacked a true control group, as has been a feature of many trials in this field.[18]

The intensity of physical activity required to reduce blood pressure is also a subject of debate, with some evidence for loss of this effect with high-intensity anaerobic exercise involving more than 70% VO_2 max sustained for 40 min three times a week.[19] Cycling, brisk walking for 40 min three times a week, jogging, and swimming[20–22] have all been reported to lead to sustained blood pressure reduction in hypertensives. Regular exercise also improves insulin sensitivity,[23] and may be of particular value for hypertensives with non–insulin-dependent diabetes mellitus.[24]

DIETARY SALT

The role of dietary salt in increasing population blood pressure levels and the rise in blood pressure with age is now well established.[25,26] However, there is still some dissension over the magnitude of the blood pressure fall with salt restriction. A metaanalysis of 32 randomized controlled trials of reducing salt intake estimated a blood-pressure–lowering effect of around 6 mm Hg systolic in hypertensives and around 2 to 3 mm Hg systolic in normotensives,[27] for a 100-mmol reduction in sodium intake. Another analysis of 56 trials reached more conservative findings of a fall of 3.7 mm Hg systolic in hypertensives, for a mean reduction in sodium intake of 95 mmol/day and a fall of 1 mm Hg systolic in normotensives.[28] On the whole, the better designed studies that have been blinded for sodium changes have shown falls of around 6 mm Hg in hypertensives. Most trials showing a blood-pressure–lowering effect of salt restriction have reduced sodium intake by 80 to 100 mmol per day, but a dose-response effect has been demonstrated with reductions between 50 and 100 mmol per day in older hypertensives.[29] Moreover, blood pressure falls were similar, at 8.2/3.9 and 6.6/2.7 mm Hg, respectively, in normotensive and hypertensive older subjects, reducing sodium intake from 10 to 5 g/day over 2 months in a well-designed double-blind trial[30] (Figure 1). The biggest problem is maintaining this degree of reduction in sodium intake in the long term[10] and in populations,[31] particularly given the high salt content of prepared foods.

A number of other issues remain under contention. First, not all people are salt sensitive, and indeed a minority may show a small rise in blood pressure with severe salt restriction,[32] presumably due to genetic or other constitutional factors. Some caution is needed in interpreting reports on so-called "salt sensitivity," because although blood pressure responses to changes in salt intake are normally distributed, this continuous variable is given an arbitrary cutoff point to define sensitivity. Furthermore, studies have mostly been very short term and involving extremes of salt intake or use of diuretic therapy. The conversion of qualitative responses to varied stimuli into categories of patients around an arbitrary dividing line is likely to maximize differences in findings between studies and may mislead as often as inform. Notwithstanding this problem, some interesting find-

ings have been reported on differences in urinary cortisol excretion[33] and endothelial dilator function[34] between salt-sensitive and salt-resistant subjects that may throw some light on possible mechanisms underlying the phenomena.

Salt sensitivity has also been reported to be an independent mediator of increased cardiovascular events in a study of 156 Japanese hypertensive patients followed for an average of more than 7 years.[35]

Differences in salt sensitivity have been used as an argument against public health measures to reduce the high sodium intake of most populations,[36] but this ignores the fact that blood pressure elevation has only been demonstrated independent of random variation with short-term extreme reductions of dietary sodium to around 20 mmol/day, ie, at levels four- to fivefold lower than those targeted in public health campaigns.

The second major area of contention concerns the effects of dietary sodium on cardiovascular outcome. A paper by Alderman and colleagues claimed that a low sodium intake was causally associated with an increase in total and cardiovascular mortality in a cohort of treated hypertensives studied prospectively.[37] The conclusions of this study can be criticized[38] on the basis that usual salt intake was never properly evaluated in these subjects, as patients were categorized according to 24-h urine sodium measurements obtained after 5 days of attempted dietary sodium restriction. Another paper reporting data on sodium intake and mortality from the United States, the NHANES 1 study, has added to the confusion.[39] The results of this study are very difficult to interpret, demonstrating increased or decreased mortality with low sodium intake depending on whether the data is analyzed according to absolute estimated dietary sodium or sodium/calorie ratio. The dietary records used in this study are even less accurate than measurement of urinary sodium excretion for assessing salt intake. Moreover, the results on mortality appeared to be confounded by ethnic and other lifestyle factors.

More reassuring are the results of the Trial of Nonpharmacologic Interventions in the Elderly (TONE) of the effects of sodium restriction and weight loss in older hypertensives.[40] In this study 585 obese subjects were randomized to either reduced sodium intake, weight loss, the two combined, or neither, whereas 390 nonobese subjects were randomized to reduced sodium intake or usual care. Withdrawal of antihypertensive therapy was attempted after 3 months' intervention. The main outcome measures (diagnosis of high blood pressure, treatment with antihypertensive medication, or a cardiovascular event during the 29-month median follow-up) were less frequent in those assigned to reduced sodium intake (relative hazard, 0.69) and in obese subjects assigned to weight loss (0.6) or weight loss combined with sodium restriction (0.47)

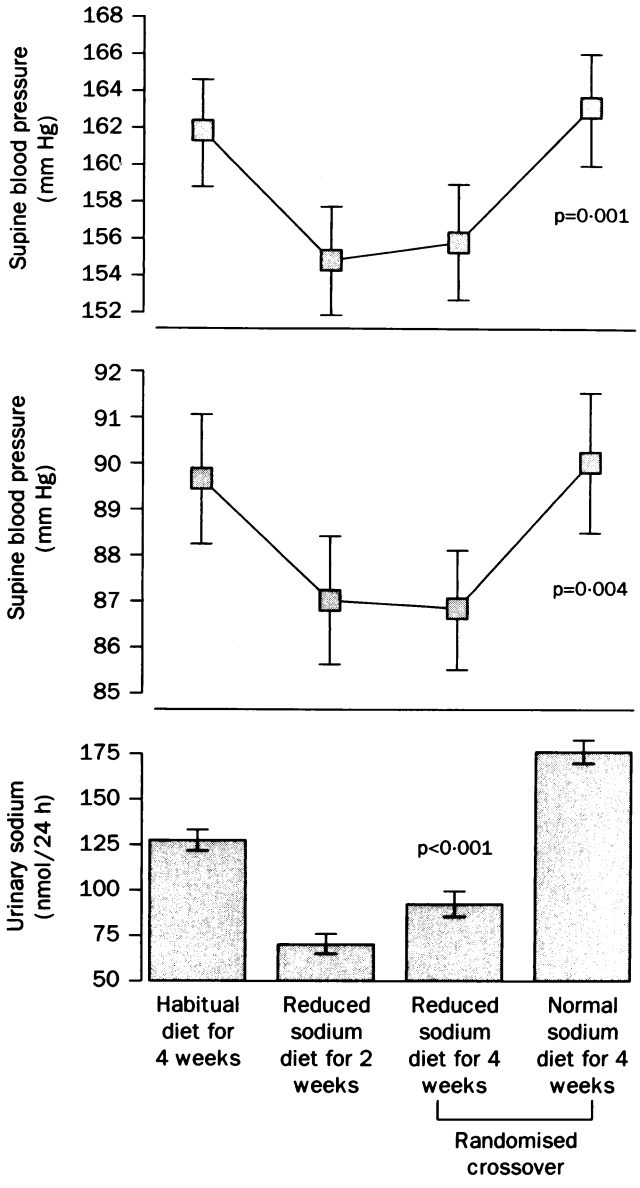

FIGURE 1. *Blood pressure and urinary sodium excretion at end of each dietary period in 47 participants. Statistical comparison by paired t test in crossover phase. Results expressed as mean and SE. SBP, systolic blood pressure; DBP, diastolic blood pressure. Reprinted with permission from Cappuccio FP et al. Double-blind randomised trial of modest salt restriction in older people. Lancet 1997;350(9081):850–854.*

(Figure 2). The frequency of cardiovascular events was similar across the six groups.

A third area of contention in relation to salt arose from studies reporting adverse changes in blood lipids in hypertensives subjected to extremes of sodium restriction (240 to 20 mmol/day) for 1- to 2-week periods.[41] However, trials using more moderate reductions in dietary sodium (180 to 70 mmol/day) over 8 weeks have not shown any such effects in normoten-

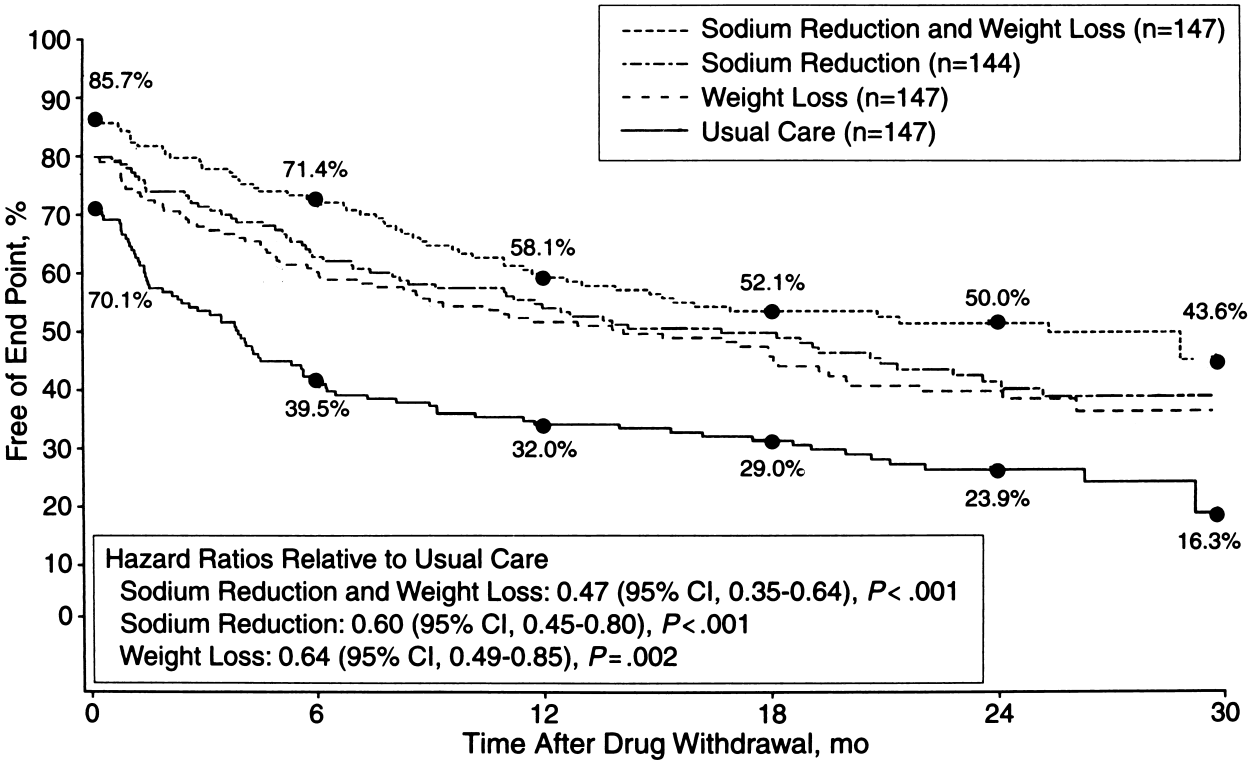

FIGURE 2. *Percentages of the 144 participants assigned to reduced sodium intake, the 147 assigned to weight loss, the 147 assigned to reduced sodium intake and weight loss combined, and the 147 assigned to usual care (no lifestyle intervention) who remained free of cardiovascular events and high blood pressure and did not have an antihypertensive agent prescribed during follow-up. CI, confidence interval. Reprinted with permission from Whelton PK, et al, and the Tone Collaborative Research Group. Sodium reduction and weight loss in the treatment of hypertension in older persons. A randomized Controlled Trial of Nonpharmacologic Interventions in the Elderly (TONE). JAMA 1998;279:839–846.*

sives[42] or in hypertensives,[43,44] despite reductions in blood pressure of around 6 mm Hg systolic. Indeed, the weight of evidence suggests that this or lesser degrees of sodium restriction will lower blood pressure and reduce antihypertensive requirements in a majority of patients.

POTASSIUM

Dietary potassium is inversely related to blood pressure levels in population studies such as Intersalt,[26] and low dietary potassium intake appears to enhance the pressor effect of a high salt intake. However, in population studies it is difficult to exclude confounding effects of other dietary constituents for which potassium may be a surrogate, such as fruit, vegetable, and fiber consumption. In this respect, neither the large prospective dietary study by Ascherio et al in adults[45] nor a cross-sectional study of children's diet and blood pressure[46] were able to detect any independent effect of potassium on blood pressure when other dietary factors were included in statistical models. In both studies, estimated fiber intake was inversely related to blood pressure. Similarly, in a longitudinal study of nutrient intake and blood pressure in 8- to 11-year-old children with raised low-density lipoprotein (LDL) cholesterol levels, the effects of potassium were lost when other nutrients were included.[47] In this study, only dietary calcium, fiber (inversely), and fat (positive relation) were associated with blood pressure when multiple nutrients were considered.

Randomized controlled trials of potassium supplementation in hypertensives have shown blood pressure reduction in proportion to the height of the blood pressure.[48] Studies in normotensives have generally shown little or no effect. However, a small effect was seen in a recent study in 300 normotensive women selected for a mineral supplementation trial on the basis that their usual intakes of potassium, magnesium, and calcium were in the 10th to 15th percentiles, averaging around 62 mmol potassium/day at baseline[49] (Figure 3). With 40 mmol/day potassium supplements alone, there was a significant reduction in ambulatory blood pressures of around 2.0 mm Hg systolic with 1.7 mm Hg diastolic, compared with placebo. Surprisingly, there was no effect of potassium when combined with calcium and magnesium supple-

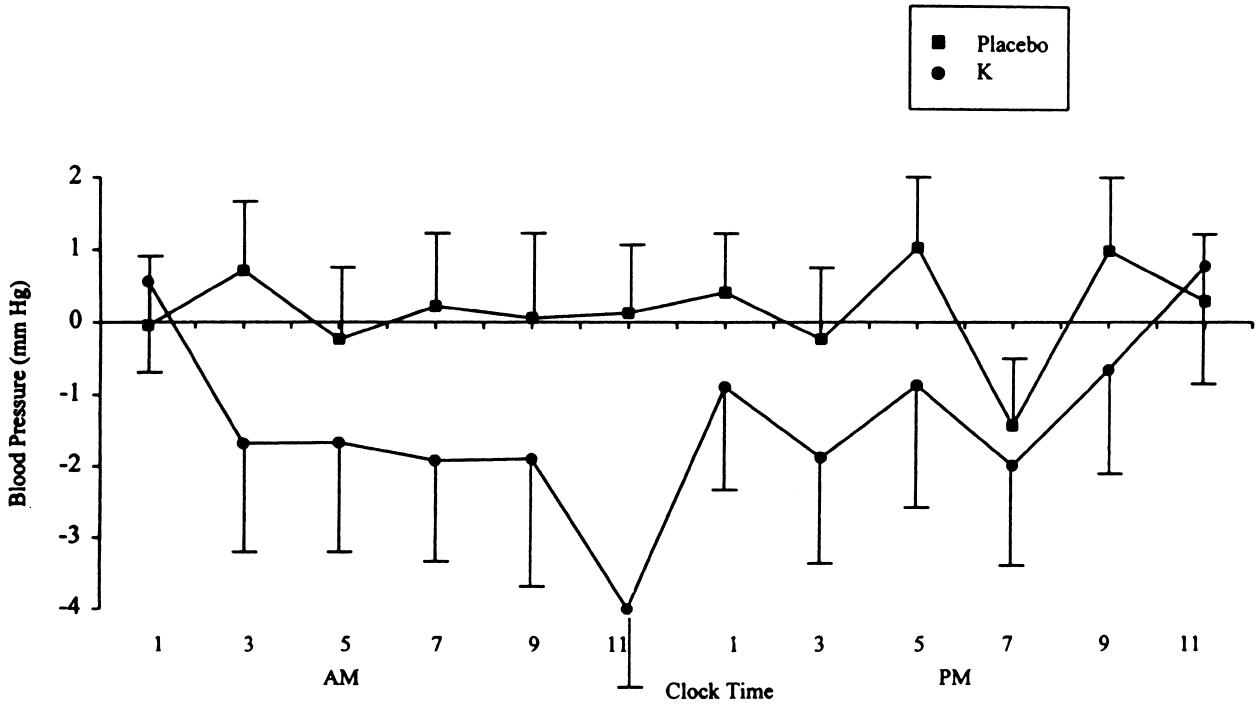

FIGURE 3. *Diastolic blood pressure changes in the potassium and placebo groups. Ambulatory blood pressure measurements were averaged for 2-h intervals with the data points indicating the median time within each 2-h interval. ●, potassium group (n = 49); ■, placebo group (n = 103). Data points show the mean changes from baseline averaging 8- and 16-week measurements during supplementation. Error bars show standard errors of the changes. Reprinted with permission from Sacks FM, et al: Effect of blood pressure of potassium, calcium, and magnesium in women with low habitual intake. Hypertens 1998;31:131–138.*

ments. This trial provides some evidence supporting the need to maintain adequate levels of potassium intake in populations to minimize the risk of blood pressure elevation. This appears to be particularly important for lower socioeconomic groups. As discussed later, foods rich in potassium, such as fruit and vegetables, may have independent blood-pressure–lowering effects and would seem the best way to increase potassium consumption.

COMPLEX DIETARY CHANGES: FRUIT, VEGETABLES, FATS, AND FIBER

Vegetarians who consume diets rich in fruits and vegetables and fiber and low in total and saturated fat have lower blood pressures and less hypertension than the general population.[50] Randomized controlled trials in meat eaters have confirmed the blood-pressure–lowering effects of such vegetarian dietary patterns in both normotensive and hypertensive subjects.[51,52] Moreover, these effects were independent of changes in body mass or dietary sodium. Subsequent controlled trials failed to identify a specific dietary component, such as polyunsaturated fat, fiber, or vegetable protein, that was responsible for these effects and indicated that they were not dependent on the presence or absence of meat protein per se.[53] Thus a so-called prudent diet containing lean meat had a similar blood-pressure–lowering effect as a strict lacto-ovovegetarian diet in normotensives.[54] As a result of a series of such studies it was suggested that the blood-pressure–lowering effects of a vegetarian diet might depend on a combination of complex dietary changes including an increase in fruit and vegetable consumption and a reduction in total and saturated fat intake.[53] This hypothesis has been substantiated in the recent DASH study in the United States,[55] in which 459 subjects with mild hypertension (< 160 mm Hg systolic and diastolic 80 to 95 mm Hg) were randomized to either continue their normal diet or increase their fruit and vegetable consumption, or, in a third group, to reduce their total and saturated fat intake. The group increasing fruit and vegetable consumption showed falls in blood pressure of 2.8/1.1 mm Hg compared with controls, whereas those also reducing fat intake showed the greatest blood pressure reduction, 5.5/3.0 mm Hg (Figure 4). Among the 133 subjects with hypertension, pressures fell by 11.4/5.5 mm Hg. Although the dietary intervention only lasted 8 weeks, this study is important in demonstrating the antihypertensive effects of relatively modest dietary changes that are likely to be more acceptable to the general population than measures that are more rad-

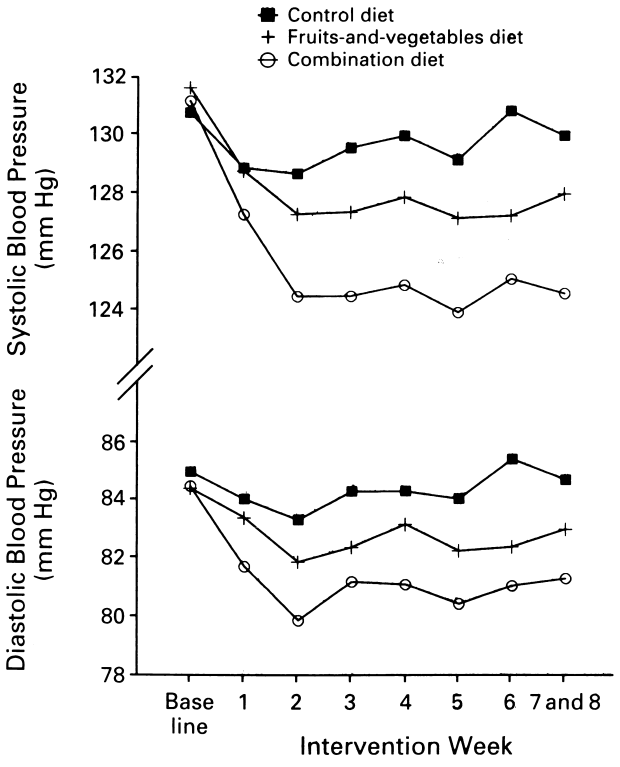

FIGURE 4. *Mean systolic and diastolic blood pressures at baseline and during each intervention week, according to diet, for 379 subjects with complete sets of weekly blood pressure measurements. Reprinted with permission from Appel LJ, et al: A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med 1997;336:1117–1124.*

ical. The dietary patterns involved in this study are also likely to reduce the risk of cardiovascular disease independently of effects on blood pressure.

DIETARY PROTEIN

Recent population studies suggesting that total dietary protein intake is associated with lower blood pressures are of interest. In the Intersalt study,[56] higher intake of protein was related to lower blood pressure, with an estimated 3.0/2.5 mm Hg difference between subjects whose protein intake was one-third above or one third below the population mean. An inverse association between protein intake and blood pressure was also reported from the Multiple Risk Factor Intervention Trial (MRFIT),[57] in the Dietary and Nutrition Survey in British Adults,[58] and in China.[59,60] In a longitudinal study, changes in blood pressure were inversely related to consumption of vegetable protein at baseline in subjects followed for 9 years.[61] However, it is not possible to conclude cause-and-effect relationships from these studies as many nutrients are found together in foods and are also related to other lifestyle habits that may influence blood pressure. Controlled dietary intervention studies are currently in progress in Western Australia to help resolve this issue.

FISH AND FISH OILS

The blood-pressure–lowering effects of n3 fatty acids of marine origin have been clearly demonstrated in randomized controlled trials in hypertensives and diabetics fed fish oil supplements containing $\geq$ 3.5 g/day of eicosapentanoic and docosahexanoic acids.[62] Metaanalyses suggest blood-pressure–lowering effects on the order of 3 to 4 mm Hg systolic.[63,64] The effects of these supplements on casual or clinic blood pressures in those with normal or high normal blood pressure appear to be marginal. Some recent studies suggest that greater attention should be given to dietary fish intake per se in relation to hypertension and cardiovascular disease. First, population studies continue to indicate that those eating fish are less prone to mortality from heart disease,[65] although not all concur.[66] Second, a study from Tanzania showed substantially lower blood pressures with aging in fish-eating Bantu compared with nearby vegetarian farmers.[67] Third, we have recently reported results of a randomized controlled trial demonstrating substantial independent and synergistic effects of a daily fish meal and weight loss on 24-h ambulatory blood pressure in 64 obese treated hypertensives (Figure 5), with falls of around 5/3 mm Hg in awake daytime pressures with the separate diets and 13/9 mm Hg with the two modalities combined.[68] Moreover, incorporating one fish meal a day into a weight-reducing regime resulted in the greatest improvement in lipid profile and glucose tolerance. Current dietary studies using highly purified n3 fatty acids suggest that the hypotensive effects seen with fish are due to these components of the fish diet rather than other dietary changes.[69]

ALCOHOL

The relation between regular alcohol consumption and blood pressure has now been established in populations drinking a variety of alcoholic beverages throughout the world.[70] In some studies alcohol ranked close to obesity for its effect on blood pressure.[71] The effect is seen in both genders, appears to increase with age, is additive to that of obesity, and may be aggravated by cigarette smoking. Randomized controlled trials have shown that heavier drinking patterns are an important and potentially reversible cause of hypertension.[72] Issues of particular interest are the nature of the dose-response effects, effects of drinking on ambulatory blood pressures, the pressor mechanisms involved, including the possible role of alcohol withdrawal in the phenomenon, and, most importantly, effects on cardiovascular and noncardiovascular morbidity and mortality. Several of these

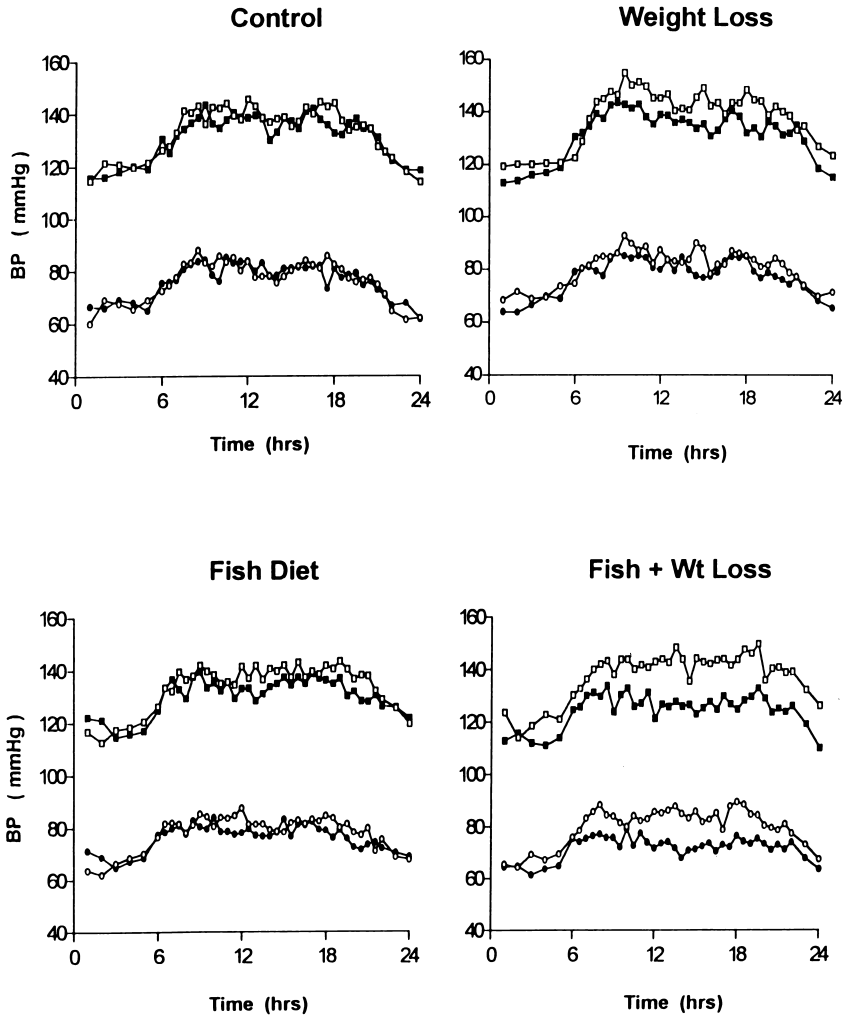

FIGURE 5. *Twenty-four-hour ambulatory mean unadjusted SBP and DBP at baseline and postintervention in the four treatment groups: □, baseline SBP; ■, postintervention SBP; ○, baseline DBP; ●, postintervention DBP. SBP, systolic blood pressure; DBP, diastolic blood pressure. Reprinted with permission from Bao DQ, et al: Effects of dietary fish and weight reduction on ambulatory blood pressure in overweight hypertensives. Hypertens (in press).*

matters have been reviewed elsewhere[70]; however, the results of some recent studies are worth highlighting.

First, there has been some uncertainty as to whether the dose-response curve for the effect of regular drinking on blood pressure is linear or J-shaped and there have been few data detailing the full range of drinking habits. However, a recent report shows linearity for the effect of alcohol on blood pressure in both men and women, from the lightest to the heaviest drinkers, over a wider range of intakes than has been detailed previously,[73] with strikingly greater effects in women in this Brazilian community (Figure 6).

FIGURE 6. *Variation of systolic blood pressure (mm Hg) with the number of drinks (10 g alcohol/drink) consumed per day, for men (◆) and women (■), adjusted for age, educational achievement, and body mass index. Baseline blood pressure is that of nondrinking subjects. Reprinted with permission from Moreira LB, et al: Alcohol intake and blood pressure: the importance of time elapsed since last drink. J Hypertens 1998;16:175–180.*

Second, the large population studies of the effects of alcohol on blood pressure all refer to casual or clinic measures. Recent studies using ambulatory blood pressure measurement in 59 whites consuming an average of four to six standard drinks a day confirm a pressor effect throughout 24 h.[74] This effect was seen regardless of whether alcohol was consumed on a daily basis or taken predominantly on weekends, the latter akin to binge drinking. However, "weekend" drinkers showed higher ambulatory blood pressures on Mondays compared with Thursdays, suggesting a component of withdrawal hypertension with this pattern of drinking (Figure 7). This result is in accord with

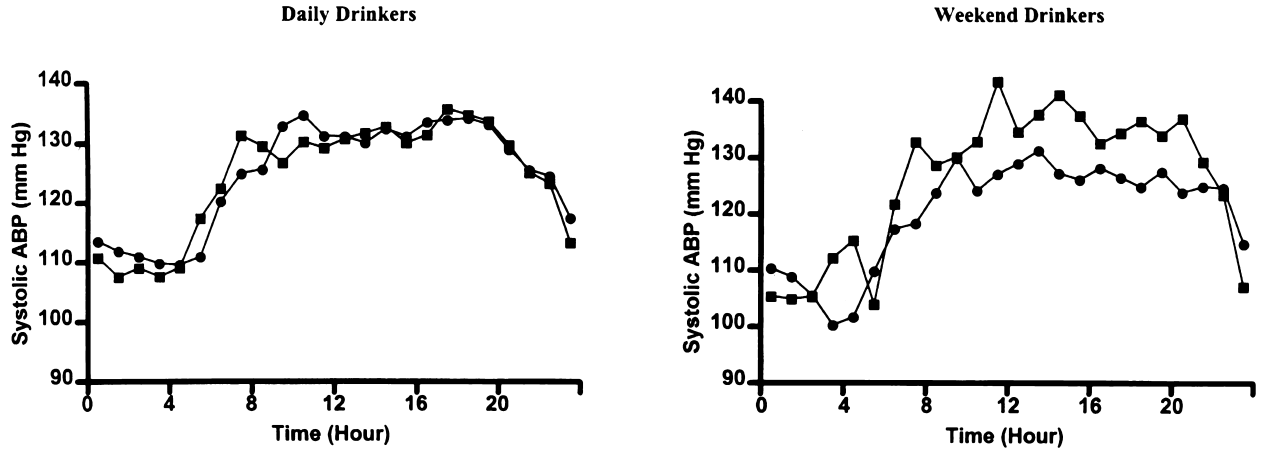

FIGURE 7. *The 24-h systolic ambulatory blood pressure (ABP) profile during week 4 of familiarization. Graphs show the difference in systolic ABP by day of assessment for daily and weekend drinkers. ■, Monday; ●, Thursday. Reprinted with permission from Rakic V, et al: Influence of pattern of alcohol intake on blood pressure in regular drinkers: a controlled trial. J Hypertens 1998;16:165–174.*

data indicating increased rates of diagnosis of hypertension in heavier drinkers seen by their general practitioners at the beginning rather than the end of the week.[75] Interestingly, Japanese drinkers who 'flush' appear more susceptible to hypertension.[76] Finally, data reviewed elsewhere[70] on the effects of alcohol on cardiovascular morbidity and mortality provide strong evidence for a reduction in coronary heart deaths and ischemic stroke with levels of intake up to around three standard drinks a day. This so-called benefit is counterbalanced by an increase in hemorrhagic stroke with more than two standard drinks.[77] Moreover, data from a prospective study of 490,000 adults in the United States show a progressive increase in deaths due to trauma, cancers, and cirrhosis with increased levels of consumption,[78] with a nadir in terms of survival rates at around one to two drinks per day in men and less than one drink per day in women. The net medical and social cost benefit of drinking alcohol will clearly be dependent on drinking patterns, age, and other risk behaviors[79] and needs to be considered in a broad context. Nevertheless, there is now ample evidence that drinking even moderate amounts of alcohol can make a substantial contribution to the prevalence of hypertension as well as increasing resistance to antihypertensive therapy.

STRESS AND PSYCHOLOGICAL FACTORS

The role of stress in sustained elevation of blood pressure remains far less clear than the lifestyle factors discussed earlier, largely due to difficulties in definitions, perceptions, and consequent measurement issues, as have been discussed elsewhere.[80,81] Using the Karacek model of job stress and control, Pickering and colleagues have reported an association between high job strain and ambulatory blood pressures in blue-collar workers, which was restricted to men who were heavier drinkers.[82] In a prospective study they found that the same job strain pattern was as numerically powerful as body mass index at predicting a rise in blood pressure over 3 years, after adjusting for confounding factors.[83] Using a different job stress model, a West Australian study of 800 men and women working in a busy government tax office suggested that ways they coped with stress were more important in determining resting blood pressure levels than the subjective experience of stress itself. Moreover, these coping mechanisms appeared to operate through lifestyle factors already known to have a direct impact on blood-pressure–regulating mechanisms such as diet, exercise, smoking, and drinking habits[84] (Figure 8). Clearly there is a need for new paradigms and more research in this field, using varied and better ways of assessing stress, coupled with ambulatory blood pressure measurements recorded in a variety of situations at work and at home.

SUMMARY AND CONCLUSIONS

A variety of lifestyle factors have been shown to directly influence blood pressure levels at both an individual and population level. Of these, the most important are excess body fat, alcohol consumption, physical activity, and a variety of dietary constituents including salt, potassium, and a complex of fruits, vegetables, and saturated fat as well as n3 fatty acids. Moderate changes in combinations of some of these factors have additive effects on blood pressure reduction in all grades of hypertension. Effects are often as large as those seen with antihypertensive drug therapy, but with a greater potential to simultaneously

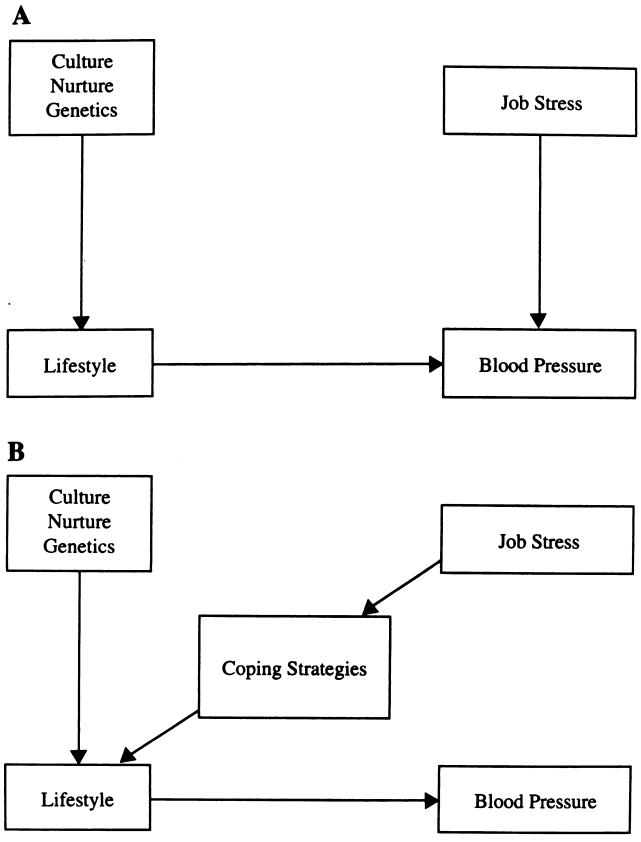

FIGURE 8. *Conceptual model of job stress, coping, lifestyle, and blood pressure relationships.* **A**, *Job stress directly influences long-term blood pressure regulation in tandem with obesity and other lifestyle factors (diet, alcohol, exercise, smoking).* **B**, *Job stress has no direct effect on resting blood pressure levels. Coping strategies in response to perceived stress influence lifestyle factors that directly modulate long-term blood pressure regulation. Reprinted with permission from Linquist TL, et al: Influence of lifestyle, coping and job stress on blood pressure in men and women. Hypertension 1997;29:1–7.*

reduce the risk of cardiovascular disease by mechanisms other than blood pressure reduction. The role of stress in long-term blood pressure elevation remains enigmatic other than through possible influences of unhealthy coping mechanisms that determine dietary, drinking, exercise, and smoking habits. Lifestyle changes have a major role to play in the prevention and management of high blood pressure and associated cardiovascular disease and in the reduction of requirements for antihypertensive drug therapy. Cook et al,[85] using data from observational studies and randomized controlled trials, estimated that reducing the average diastolic pressure in a population by as little as 2 mm Hg through lifestyle changes would decrease the prevalence of hypertension by 17%, with a 6% reduction in the risk of coronary heart disease and a 15% reduction in the risk of stroke and transient ischemic attacks. Improved methods of achieving changes towards a healthier lifestyle remain a high medical and social priority.

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