Diet Modifications to Control Hypertension: The Multifactorial Approach

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Introduction

In the United States and other developed countries, approximately 1 in 4 adults has hypertension or high blood pressure, a major risk factor for coronary heart disease, stroke and renal disease [1]. The risk of developing hypertension increases with age, so the prevalence is even higher in the older age groups. The role of dietary factors in the etiology and treatment of hypertension continues to be an area of considerable interest, and results from numerous epidemiological and clinical studies form the basis of current knowledge in this area. Interpretation of results from cross-country comparisons and observational epidemiological studies, which are hypothesis-generating, has been considerably refined over the past few decades, based on findings from clinical trials and carefully controlled diet intervention studies.

An emerging concept is that several components of the diet and various lifestyle factors have independent effects on the development and management of high blood pressure, and that optimal control of high blood pressure is more likely to be achieved with a multifactorial approach. Further, dietary factors and food choices reflect an overall dietary pattern, and disentangling the effects of various components of that pattern can be difficult. Intakes of nutrients and other dietary constituents are typically inversely or directly correlated with one another, due to their distributions in foods.

This review summarizes the most important research efforts and results of clinical trials that provide the scientific support and rationale for current dietary guidelines for the prevention and management of hypertension. In addition to dietary factors, other lifestyle factors that affect risk and the management of hypertension include obesity, physical activity, smoking and...
alcohol intake. However, this brief review will focus on diet composition and specific dietary factors, and the reader is referred to other summaries that describe the evidence and current guidelines relating to the other lifestyle factors [1, 2].

**Specific Dietary Factors**

Numerous dietary factors have been hypothesized to play a role in the development and management of hypertension. These factors include minerals, such as sodium, potassium, calcium, and magnesium; macronutrients such as protein, fat, and carbohydrate; and other dietary constituents, such as n-3 fatty acids. Of these specific dietary factors, sodium has historically received the most attention.

Overall, cross-country comparisons provide fairly strong support for a direct relationship between sodium intake and the risk for hypertension. For example, in the INTERSALT study [3], the relationship between 24-hour urinary sodium excretion and blood pressure was examined in a large sample (10,079 men and women aged 20–59 years from 32 countries), and a significant, positive, independent linear relation was found. In a multivariate analysis adjusted for anticipated underestimation due to the assessment methodology that was used, a 100-mmol/day (approximately 2,300-mg/day) increase in sodium was associated with a 3- to 6-mm Hg increase in systolic blood pressure and a 0- to 3-mm Hg increase in diastolic blood pressure. However, studies of this type are confounded by probable differences in other factors that affect blood pressure and risk for hypertension, such as genetic factors, obesity and physical activity, across the populations examined.

In several randomized controlled studies, a reduction in sodium intake has been shown to reduce blood pressure. For example, the Trials of Hypertension Prevention phase I and II studies demonstrated a reduction in systolic (averaging −1.7 mm Hg) and diastolic (averaging −0.9 mm Hg) blood pressure in association with a reduction in sodium intake as measured by sodium excretion (−44 mmol/day) and prevention of hypertension in individuals with high normal blood pressures [4, 5]. However, the results of a meta-analysis that included data from 58 trials of hypertensive individuals indicate that the effects of reduced sodium intake on blood pressure, when this strategy is the sole focus of the intervention, are generally modest at best [6]. An average reduction in sodium excretion (to a mean of 118 mmol/day) was associated with an average change of −3.9 mm Hg in systolic and −1.9 mm Hg diastolic blood pressure in that meta-analysis.

An important individual characteristic that is known to influence blood pressure response to change in sodium intake is salt sensitivity. A subset of hypertensive individuals (estimated at approximately 50% of that population) and normotensives (approximately 25%) are salt-sensitive, as demonstrated
by an increase in blood pressure and body weight when changing from a low to high sodium diet [7]. This factor likely contributes to the variability in blood pressure response when sodium intake is reduced, and suggests that not all individuals with hypertension or at risk for hypertension will benefit from sodium restriction. Salt sensitivity is correlated with microalbuminuria and is more common in African-Americans (compared to whites) and the elderly. Notably, however, an easy and convenient method for identifying salt sensitivity has not been reported, although markers of this characteristic may eventually be identified. Thus, predicting responsiveness to sodium restriction is not a feasible strategy.

A more recent food-based intervention study, discussed in detail below, demonstrated a distinct blood pressure-lowering effect of sodium restriction, when added to a major change in the overall dietary pattern, in adults with above-optimal blood pressure or in the lower range of stage-1 hypertension [8]. Nonetheless, promoting sodium restriction as an effective approach to the prevention and control of hypertension remains an unresolved issue, and the discussion is most contentious when this strategy is suggested as a general public health recommendation [9]. One of the biggest challenges in adhering to this recommendation is that a large amount of sodium ingested (estimated at 75% in the United States) is obtained from processed foods, so a substantial reduction in sodium intake can be difficult to achieve and maintain. The sodium content of processed foods is generally under the control of manufacturers, who are influenced by market forces and the taste preferences of the general population. Also, common foods that contain sodium, such as bread, are major contributors to sodium intake because of the quantity and frequency of consumption of these foods.

In cross-country and observational studies, the evidence suggesting that potassium intake is inversely associated with blood pressure and risk for hypertension is fairly consistent, and several intervention studies have tested the effects of potassium supplementation on blood pressure. In a meta-analysis based on data from 33 controlled studies that mainly targeted individuals with hypertension [10], potassium supplementation (median 75 mmol/day [approximately 2,925 mg/day]) promoted an average reduction of \(-3.11\) in systolic and \(-1.97\) in diastolic blood pressure. The beneficial effect was greater in African-American participants (versus whites) and in those with higher (versus lower) levels of urinary sodium excretion. Fruits and vegetables are major sources of potassium in the diet.

Although less consistent than the results of studies focusing on sodium and potassium intakes, calcium intake has been inversely associated with blood pressure and risk for hypertension in some observational studies [11]. Intervention studies testing this relationship have mainly utilized calcium supplementation (typically 1,000 mg/day), although a few studies have tested the effect of increasing calcium intake from calcium-rich foods on blood pressure. Results from the calcium supplement studies have shown modest
effects at best [12], and results from increasing calcium intake from foods as
the sole intervention strategy have been mixed [13]. Dairy foods are the rich-
est source of calcium in the diet, although selected vegetables and foods that
have calcium added during processing are also good sources.

Based on inverse associations between magnesium intake and blood pres-
sure in observational studies, the effect of magnesium supplementation on
blood pressure has been tested in several randomized studies, mainly involv-
ing subjects with high blood pressure. The majority of these studies did not
find beneficial effects [2]. Dietary magnesium is obtained from a wide variety
of foods, particularly vegetables, grains, nuts and dairy products.

Although high-protein diets are known to increase the glomerular filtration
rate and thus potentially increase the risk for renal failure, the majority of
epidemiological studies that have examined the association between dietary
protein intake and blood pressure suggest an inverse relationship [14].
However, intervention studies that have tested this hypothesis have been too
small or too confounded to support the hypothesis that protein intake per se
has independent effects on blood pressure under controlled conditions.

Results from epidemiological studies relating total fat intake to blood pres-
sure have not been consistent. In diet intervention trials aimed toward reduc-
ing total fat and saturated fat intakes, a reduction in blood pressure has been
observed in some but not all of these studies [15, 16]. In contrast, results from
a meta-analysis of 17 controlled clinical trials that examined the effect of n-3
fatty acid supplements on blood pressure found significant reductions in
blood pressure in individuals with hypertension (but not in normotensives)
[17]. The average reductions in individuals with hypertension were −5.5 for
systolic and −3.5 for diastolic blood pressure in response to n-3 fatty acid
supplementation (generally >3 g/day). However, side effects such as eructa-
tion and a fishy taste were often reported by treatment group subjects, and
administering this level of n-3 fatty acids is associated with other potentially
adverse medical consequences.

Only a limited number of observational and clinical studies have examined
the specific association between carbohydrate intake and blood pressure. In
a controlled feeding study, a high-carbohydrate (52% of energy) compared to
a low-carbohydrate (13% of energy) diet was observed to have a short-term
anti-natriuretic effect but no significant effect on blood pressure in normo-
tensive men [18].

The interpretation of the suggestive but inconsistent evidence linking
several specific dietary factors to the risk and management of hypertension
indicates that a more appropriate focus may be a focus on the overall dietary
pattern. Diets containing high levels of vegetables and fruits have been con-
sistently associated with a significantly lower risk for stroke in large cohort
studies [19]. The relationship between a dietary pattern and a hypertension-
related hard end point provides support for testing intervention approaches
focused on foods and the total diet. The ability to attribute response to
specific nutrients or other dietary factors with this type of intervention is not possible, because modifying the overall dietary pattern is inherently multifactorial by design.

**Food-Based Multifactorial Approaches**

Based on the concept that changing the overall dietary pattern will result in changes in the intakes of several nutrients and dietary factors, the Dietary Approaches to Stop Hypertension (DASH) diet study was conducted [20]. The DASH study was a randomized controlled feeding study involving 459 adults with untreated systolic blood pressure of <160 mm Hg and diastolic blood pressure of 80–95 mm Hg. Following 3 weeks of a control diet (containing 3.6 servings/day fruits and vegetables, and 0.5 servings/day dairy foods), subjects were assigned to receive 1 of 2 intervention diets or to continue the control diet for 8 weeks. A notable feature is that the DASH diet study was a feeding study, with all foods supplied to the subjects, and in addition to thorough oversight of intakes, this design provides assurance that adherence to the regimen was high. Weight and sodium intake were held constant during the study period.

One of the intervention diets was focused solely on increased intake of fruits and vegetables, with the target being 8.5 servings/day. The other intervention diet, termed the DASH ‘combination’ diet (referred to as the DASH diet), targeted 9.6 servings/day fruits and vegetables, 2.0 servings/day low-fat dairy foods (in addition to 0.7 servings/day regular dairy foods), and increased intakes of whole grains, poultry, fish and nuts. Percent energy from fat in the DASH diet was 26% (compared to 36% in the other 2 diets), and percent energy from saturated fat was 7% in the DASH diet (compared to 13–14% in the other 2 diets). The reduction in saturated fat intake was achieved with a reduction in meat intake, and intakes of sweets and sugar-containing foods also were limited compared to the control diet and the typical diet consumed in developed countries. The 2 intervention diets had similar and higher amounts of potassium (4,101 mg/day in the fruits and vegetables diet, and 4,415 mg/day in the DASH diet) and magnesium (423 mg/day in the fruits and vegetables diet, and 480 mg/day in the DASH diet), compared to the control diet (1,752 and 176 mg/day, respectively). However, the DASH diet provided more calcium (1,265 mg/day) than the fruits and vegetables (534 mg/day) and control (443 mg/day) diets.

Both the fruits and vegetables diet and the DASH diet promoted a reduction in blood pressure in both non-hypertensive and hypertensive subjects, but the effect of the DASH diet was substantially greater. In the 133 hypertensive subjects, the DASH diet reduced systolic and diastolic blood pressure by an average of −11.4 and −5.5 mm Hg, respectively, compared to the effect of the control diet. A less substantial effect was observed in response to this
Diet in the 326 non-hypertensive subjects, in whom systolic and diastolic blood pressures were reduced by an average of −3.5 and −2.1 mm Hg, respectively. Subgroup analysis revealed that the DASH diet reduced blood pressures significantly more in African-Americans than in whites, and in hypertensive subjects more than in non-hypertensive subjects [21].

As a result of the findings in this landmark study, the DASH dietary pattern was incorporated into the national practice guidelines in the United States as an appropriate strategy in the management of hypertension. Table 1 summarizes the guidelines and specific contents of the DASH diet.

In a follow-up feeding study, the DASH-Sodium Trial, the effect of the DASH diet in combination with 3 levels of sodium intake was examined in 412 adults whose blood pressures exceeded 120/80 mm Hg, including those with stage-1 hypertension (defined as a systolic blood pressure of 140–159 mm Hg or a diastolic blood pressure of 90–95 mm Hg) [22]. Participants were provided the DASH diet or a control diet for 3 consecutive 30-day periods, during which sodium intake (approximately 65, 106, and 143 mmol/day) was varied in a randomly assigned sequence. Similar to the first DASH diet study, body weight was maintained throughout the study period, and all foods were provided. The higher sodium level that was targeted reflects the typical level of consumption in the United States.

At all 3 levels of sodium intake, subjects assigned to the DASH diet exhibited lower blood pressures than subjects assigned to the control diet. Reducing the sodium intake from the high to the intermediate level reduced the systolic blood pressure by −2.1 mm Hg in association with the control diet and −1.3 mm Hg in association with the DASH diet. Reducing the sodium intake from the intermediate to the low level caused an additional reduction of −4.6 mm Hg in association with the control diet and −1.7 mm Hg in association with the DASH diet. The effects of sodium reduction on blood pressure were greater in African-Americans than in whites and in hypertensive subjects compared to non-hypertensive subjects [23]. The DASH diet with low sodium content reduced systolic blood pressure by −7.1 mm Hg in non-hypertensive subjects and −11.5 mm Hg in hypertensive subjects, compared to the control diet with high-sodium content. Based on results from an ancillary study using data collected during the DASH-Sodium Trial (mean arterial pressure and urinary sodium excretion), the DASH diet was shown to exert a natriuretic effect [24]. Results from this study suggest that as an addition to the modified dietary pattern reflected in the DASH diet, a reduction in sodium intake can independently reduce blood pressure, and the effect is more pronounced in individuals with hypertension. Practice guidelines in the United States currently recommend that the management of hypertension include both adopting the DASH dietary pattern and reducing dietary sodium intake to approximately 100 mmol/day [1].

The PREMIER Study is the most recent food-based multifaceted diet intervention study aimed toward control of high blood pressure [25, 26]. This study
**Table 1.** The DASH diet (based on 2,000 kcal/day)

<table>
<thead>
<tr>
<th>Food group</th>
<th>Daily servings</th>
<th>Serving sizes</th>
<th>Examples and comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grains and grain products</td>
<td>7–8</td>
<td>1 slice bread, 225 ml dry cereal, 110 ml cooked rice or pasta</td>
<td>Whole wheat bread, English muffin, pita bread, bagel, cereal, grits, oatmeal, crackers</td>
</tr>
<tr>
<td>Vegetables</td>
<td>4–5</td>
<td>225 ml raw leafy vegetables, 110 ml cooked vegetables, 170 ml vegetable juice</td>
<td>Tomatoes, potatoes, carrots, green peas, squash, broccoli, spinach, green beans</td>
</tr>
<tr>
<td>Fruits</td>
<td>4–5</td>
<td>55 ml fruit juice, 1 medium fruit, 55 ml dried fruit, 110 ml fresh, frozen, or canned fruit</td>
<td>Apricots, bananas, dates, grapes, oranges, orange juice, grapefruit, mangoes, melons, peaches, pineapples, strawberries</td>
</tr>
<tr>
<td>Low-fat or fat-free dairy foods</td>
<td>2–3</td>
<td>227 ml milk, 225 ml yogurt, 43 g cheese</td>
<td>Fat-free or low-fat milk, fat-free or low-fat frozen yogurt, low-fat and fat-free cheese</td>
</tr>
<tr>
<td>Meats, poultry, and fish</td>
<td>2 or less</td>
<td>85 g cooked meats, poultry or fish</td>
<td>Select only lean; trim away visible fats; broil, roast, or boil, instead of frying; remove skin from poultry</td>
</tr>
<tr>
<td>Nuts, seeds, and dry beans</td>
<td>4–5/week</td>
<td>75 ml or 42.5 g nuts, 2 tbsp or 14 g seeds, 110 ml cooked dry beans</td>
<td>Almonds, filberts, mixed nuts, peanuts, walnuts, sunflower seeds, kidney beans, lentils and peas</td>
</tr>
<tr>
<td>Fats and oils</td>
<td>2–3</td>
<td>1 tsp soft margarine, 1 tsp low-fat mayonnaise, 2 tbsp low-fat salad dressing</td>
<td>Soft margarine, low-fat mayonnaise, low-fat salad dressing, vegetable oil</td>
</tr>
<tr>
<td>Sweets</td>
<td>5/week</td>
<td>1 tbsp sugar, 1 tbsp jelly or jam, 14 g jelly beans, 227 g lemonade</td>
<td>Maple syrup, sugar, jelly, jam, hard candy, sorbet, ices</td>
</tr>
</tbody>
</table>


tbsp = Tablespoon; tsp = teaspoon.
was a randomized, controlled trial that tested the effect of modifying several lifestyle factors, utilizing an intensive 18-month behavioral approach to intervention. Subjects (n = 811) were eligible if they had above-optimal through high-normal blood pressure (systolic 120–139 mm Hg and diastolic 90–95 mm Hg) or stage-1 hypertension (systolic 140–159 mm Hg or diastolic 90–95 mm Hg). The three study arms had similar targets for several lifestyle factors, with the following goals: lose ≥7 kg if overweight or obese (through increased physical activity and reduced energy intake), limit sodium intake to ≤100 mmol/day, and limit alcohol intake to ≤28 g/day for men and ≤14 g/day for women. These lifestyle factors were conceptualized as being established factors and thus standard care strategies. One of the groups (the advice only group) received this guidance in a single individual counseling session. The other 2 groups were enrolled in an intensive behavioral intervention program involving 14 group meetings and 4 individual counseling sessions during the initial 6 months. One of those behavioral intervention arms incorporated the DASH diet as an additional goal, added to the established lifestyle factor goals, so the two intervention arms consisted of the established and the established plus DASH diet groups. An important feature that differentiates this study from the 2 previous DASH diet studies is that the participants were not provided food or meals, and instead, they were provided guidance and counseling to help them translate these goals into behaviors and food choices.

In both of the behavioral intervention arms of the PREMIER Study, a significant reduction in body weight and improved fitness was achieved. Although urinary sodium excretion declined in all of the study arms, a significantly greater reduction in the two behavioral intervention arms (averaging −31.6 mmol/day for the established and −32.6 mmol/day for the established plus DASH diet groups) than in the advice only arm (averaging −20.6 mmol/day) was observed. The established plus DASH diet group also reported increased fruit, vegetable and dairy food intake, although the average level of fruit and vegetable intake achieved was somewhat less than the target (average intake increased from 4.8 to 7.8 servings/day in that group, compared to the intake of 9.6 servings/day in the DASH diet feeding studies). Possibly as a result of that lesser degree of change (compared to the feeding studies), the difference in blood pressure responses between the established plus DASH diet and the established intervention arms was less than expected, based on data from the DASH diet feeding studies. The mean reduction in systolic blood pressure at 6 months was −11.1 mm Hg in the established plus DASH diet group, −10.5 mm Hg in the established group, and −6.6 mm Hg in the advice only group. Both the established and established plus DASH diet behavioral interventions significantly reduced systolic and diastolic blood pressure compared to the advice only group, but the difference between the established and established plus DASH diet group was not significant.

A key finding from the PREMIER Study is that major changes in the overall dietary pattern, in addition to a reduction in sodium intake, can be achieved
in free-living subjects, and that these changes are associated with a substantial reduction in blood pressure. Notably, however, an intensive counseling effort, focused on developing behavioral skills to facilitate a sustained change in food choices and dietary intake, was a key component in the achievement of the dietary goals. In other studies in which the purpose is to promote cancer prevention or other health outcomes, similar intervention efforts have been shown to result in a substantial change in fruit and vegetable intake and in the overall dietary pattern [27, 28].

**Conclusions**

Based on the results of recent studies testing a food-based multifactorial approach, the combined effects of various dietary factors appear to have a much greater effect on blood pressure and control of hypertension than has been observed in studies in which the focus is on a single dietary factor. Further, there are other health benefits that may be derived from modifying the diet to include more vegetables, fruit and fiber, and less saturated fat, such as reduced risk for cancer and coronary artery disease [29, 30].

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Discussion

Dr. Allison: Thank you very much for an excellent presentation. The interest in
salt restriction, if we draw an analogy between obesity and diabetes, we are talking
about very long time scales and we are talking about the fact that once diabetes has
developed you are not going to cure it by diet alone, you might prevent it, so there is
a difference. I think cardiologists are a bit naïve about this business. They think that
if you give a low sodium diet for 3 weeks, it doesn’t work, so therefore sodium is not
important. If you look at much longer term studies of primary prevention, particularly
studies from India, between a rural population moving to the city, then the effects of
sodium are very much bigger. Of course there are other aspects of diet as well. If you
look at what were essentially your primary prevention studies, the slopes would continue to go down, and if you continue the study perhaps for another 5 years would they have continued to go down? Admittedly most of them were in a relatively normal rate. So we really need to look carefully at the time scale of these and whether it is primary or secondary prevention because secondary prevention it may be too late.

*Dr. Rock:* Generally what we do in behavioral interventions is we have a more stepwise approach where initial changes are made and working with the subject the dieticians will, for example, contract for a behavior change for 1 week or 1 day and see how that goes. So in terms of actual change in the diet, it is not like suddenly going on a drug, they are actually changing slowly over a period of time. That is one reason why you see a kind of gradual habituation, particularly in this kind of intervention. In fact we use that term when we talk about exercise interventions. How long it takes to get to that point varies a lot from one person to another. I would argue that, depending on the degree of hypertension, most of the people in especially these latter studies actually were hypertensive and they were taken off drugs, they weren’t starting on drugs before they were enrolled in the trial. So I would argue that this really relates to treatment in addition to primary prevention and for many people. We had an interesting discussion with Dr. Kopelman yesterday talking about compliance with drugs because people have to eat and if they get used to a food pattern and know what brand to buy and how to cook it, they do get habituated. In many ways if we do it the right way we can get better compliance with food choices than we can with taking a pill every day, and there are no side effects. You don’t get dry mouth, there are a lot of advantages.

*Dr. Go:* My comment is that dietary intervention not only for high blood pressure, but the same diet is applicable for obesity, for weight loss, for cancer prevention and for diabetes. So in a sense there is a commonality of all the diet intervention studies, and the results and application seem to go the same way. You already covered fat, minerals and vitamins. What is the role of phytochemicals in all of this? I don’t see any discussion even focusing on phytochemicals in diabetes. Fruits and vegetables are always in every one of the diets for all the metabolic syndromes that we talk about.

*Dr. Rock:* This is a very good comment and another reason why we shouldn’t be thinking too reductionist in terms of talking only about things we can quantify. Phytochemicals have largely only recently been isolated and identified. We don’t know bioavailability, we don’t know how much variability there is from one food source to another. So it is the other sort of ghost behind why these kinds of relationships, like the one I showed with fruit and vegetable intake, but we know it is something other than the vitamins that are in those foods that are the real mechanistic force. We are really just at the beginning of that kind of exploration, especially when it comes to blood pressure.

*Dr. James:* I am very intrigued by your data and analysis. I could not see from the back how to relate your cross-country into salt data with your intervention data. As I understand it, you are implying that the DASH study [1] in a sense was taking a different range for those with a polygenic sensitivity to salt. If you look at the Intersalt Study, I know that certainly in the second set of analysis they began to look explicitly at the age-related increase. I am reminded of the Dutch infant formula data showing that children on a lower infant formula salt intake had a statistically reduced salt intake 15 years later. It has been shown in epidemiological studies on hypertension that in hypertensive terms there seems to be an intrinsic amplification of blood pressure as you get older depending upon your preexisting blood pressure. If you look at the biology of it, elastin in the blood vessels has a molecular system such that you have a turnover of 40 years in the elastin that reduces the compliance. I am just wondering about your relatively short-term intervention data: do they really show a much more modest effect?
Dr. Rock: That is a good point and that is one reason why I included that, and Intersalt Study was of course one of the many studies to compare across countries. But the problem is that they are confounded by nature and sometimes they can be misleading. For an example, right now I am primarily involved in cancer research, and we can draw wonderful graphics of intakes of various dietary constituents and risk for cancers, but other characteristics must be taken into account, and I am not talking about genetics here, I am talking about other lifestyle characteristics and weight gain patterns and physical activity and alcohol intake and stress. But I do think you are right and that they are enlightening because they give us a window to look at lifetime exposure.

Dr. Wasantwisut: I have a question about the effective period. The diet, the PREMIER study [2], that plus behavior, how long does it take for you to see such an effect? I was interested by the fact that those diets are applicable to other conditions. Do you measure the other things that are of benefit to the population and can you sustain those things? I mean, once you do not intervene, what happens?

Dr. Rock: The PREMIER study was a large multicenter study that was actually run out of the National Institutes of Health, but I can tell you data from other studies with which I am involved. In the Women's Health Eating and Living study we have over 3,000 women who we randomized to being in intensive diet intervention and basically focused on eating more vegetables and fruits and less fat, and we are collecting blood at specific intervals. In the 3-year data we actually still see a higher intake of vegetables and fruits; in the blood sample we measure much higher levels of carotenoids, we get most of those from vegetables and fruits. But your point is well taken, you don't just cut people off. We are following women for years and we taper the interaction depending on where the person is in her behavior change. When you mention how long before we see an effect: in the other large studies we are involved in we usually see a peak effect in about 6 months and then there is some recidivism which you would expect. But if you have done the intervention right, by the time they hit 6 months, they have achieved their maximum in terms of behavior change, they are reinforced enough with that lifestyle and those choices that they have developed some of the tools for getting through the tough times that they had. So we expect to see people fall off a little bit but if we have done the intervention right they internalize the skill. It is not me telling them what to eat or the doctor telling them what to do, it is that in a collaborative counseling arrangement they say this is what I am willing to do, this is where we are working my lifestyle. This philosophy goes back to the Diabetes Control and Complications Trial since we are talking about diabetes, that in the old days with diabetes we would say here is your diet and here is your insulin schedule, and what we learned from the Diabetes Control and Complications Trial is you sit down with the patient and say 'what is your day like, at what time do you get up in the morning, what do you like to eat'. That was really the first study that showed how crucial it was to do individualized intervention. So we make the changes fit the person rather than the person fit the goal.

Dr. Biolo: As a physician I would like to make a comment. When hypertension is already established, we know that with the diet it is very difficult to be able to treat hypertension. You have shown that there is a decrease of no more than 10 mm Hg with that diet alone, but we know for sure that the interaction is very important between diet, in particular a low-sodium diet, and antihypertensive drugs, not only diuretics, and we know that using them is very powerful when you are taking a low-sodium diet. I would like to underline this very important point.

Dr. Rock: I totally agree with you, that is why I said adjuvant because if someone is making these changes and you still have to start a drug, hopefully it will be at a much lower dosage, lower risk for adverse effects and greater likelihood that they will continue to be compliant. It is not necessarily either or.
**Dr. Marette:** I am glad you mentioned protein as one nutrient that can regulate the blood pressure. We did some studies that have shown that different types of dietary proteins, can dramatically affect insulin sensitivity [3–6]. So I was wondering if you are aware of any studies that have shown that the type or the source of dietary proteins can affect blood pressure?

**Dr. Rock:** Among this amount of smallish studies that have been done, I don’t think they are of good enough quality that I would draw any conclusion, they usually don’t have a control group or there are other things that are changing in the diet in addition to the protein. One is the phytochemicals, if they switch to, for example, soy foods then there are other things that are being administered besides the different protein. But it is interesting if you look at what has been effective in the DASH diet and the PREMIER diet, there is clearly a shift away from animal protein, included in the summary and also in the manuscript that will be published are the actual foods that make up the DASH diets, and you can see them.

**Dr. Marette:** In the studies where you looked at fish oils, I don’t remember if it was actually your studies or some other investigators, but did you or they show any effect of fish oils on insulin sensitivity?

**Dr. Rock:** They were randomized placebo-control trials, they basically gave about 3 g of fish oil compared to none, and they were very short-term studies which just focused on looking the effects on the blood pressure.

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