Effect of Antihypertensive Therapy on Weight Loss

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For the Trial of Antihypertensive Interventions and Management Research Group

We report the effect on weight changes of the type of antihypertensive medication prescribed in a trial of the relative efficacy of drug and dietary measures in mild hypertension. The Trial of Antihypertensive Interventions and Management studied 878 mildly hypertensive individuals randomly assigned, in a 3x3 design, to no diet change, weight loss, or a low sodium-high potassium diet and to placebo, 25 mg chlorthalidone, or 50 mg atenolol. The type of drug prescribed affected weight change with all diets. The drug effect on weight change, present in all groups at 6 months, was most pronounced in those randomly assigned to the weight loss diet, where the placebo group lost 4.4 kg, the atenolol group lost 3.0 kg, and the chlorthalidone group lost 6.9 kg. The group differences were attenuated but persisted at 24 months.

We suggest that the antihypertensive drug prescribed affects the success of a concomitant weight loss program and speculate that the difference between the drugs may be due to their intrinsic effects on the sympathetic nervous system and related metabolic changes. (Hypertension 1992;19:393–399)

KEY WORDS • mild hypertension • weight loss • diet • diuretic therapy • adrenergic beta receptor blockers • sympatholysis

Weight loss is often prescribed for hypertensive individuals who require antihypertensive medication. The available literature suggests that weight loss will increase the antihypertensive effectiveness of medications and decrease adverse effects.1-3 A study in Israel showed that weight loss, probably without change in the sodium intake, produced marked lowering of blood pressure in treated patients.4 Data from a large clinical trial, the Hypertension Detection and Follow-up Program, showed that individuals who lost weight during the course of the trial had better blood pressure control than controls whose weight did not change,5 and those who gained weight had poorer blood pressure control. Two studies (Dietary Intervention Study of Hypertension6 and Hypertension Control Program7) demonstrated that weight loss reduced the probability for recurrence of hypertension. The Trial of Antihypertensive Interventions and Management (TAIM), the study from which the present observations are drawn, showed that modest weight loss in the drug-treated patient increased the hypotensive effect of the drugs1 and reduced cardiovascular risk.2

The complementary question, whether antihypertensive medications affect the ability of patients to lose weight, does not seem to have been asked. We report observational data gathered in the course of a randomized and partially blinded trial on the combined effect of diet change and hypotensive therapy that suggest there is an interaction between the type of antihypertensive drug prescribed and the amount of weight loss.

Methods

TAIM was a multicenter trial using a 3x3 design to evaluate the efficacy of diet and drug treatment separately and in combination in reducing blood pressure. A brief description of the experimental design, which has been published in full,8 follows.

Participants were randomly assigned to one of three diets—no diet change, weight loss, or low sodium-high potassium and to one of three drugs—placebo, chlorthalidone (25 mg), or atenolol (50 mg). Individuals were enrolled who were between the ages of 21 and 65 years, weighed between 110% and 160% of their ideal weight, and had untreated diastolic blood pressures (DBP) of 90–100 mm Hg. Those on prior antihypertensive medication were taken off therapy for at least 2 weeks before entry. Those with major end organ disease or other complications were excluded. The study protocol was approved by the institutional review boards of all participating institutions, and participants gave informed consent by signature before randomization.
The goal for the weight loss group was a reduction of 10% of baseline weight or 4.5 kg, whichever was greater. Sodium and potassium goals were individualized by weight and ranged from 52 to 100 mmol/day for sodium (average 87 mmol/day) and from 62 to 115 mmol/day for potassium (average 103 mmol/day). To attain these goals, nutritional counseling oriented to behavioral change was provided in group sessions held weekly for 10 weeks. Thereafter, individual sessions were held every 6–12 weeks. This approach was a modification of the one used in the Dietary Intervention Study of Hypertension.

Participants who failed to achieve adequate blood pressure control were stepped up to additional therapy at 6 months or sooner if emergency failure criteria were met. The additional step-up therapy was administered in a double-blind fashion. Either 25 mg chlorthalidone or 50 mg atenolol was given to placebo failures (allocated in a 1:1 fashion); combined 25 mg chlorthalidone–50 mg atenolol was given to the chlorthalidone or atenolol failures. Medication was increased during the first 6 months if DBP was equal to or greater than 100 mm Hg for three visits at 2-week intervals, or equal to or greater than 105 mm Hg at any visit. If the additional step-up therapy did not adequately control the DBP, open-label therapy (known antihypertensive medication) was used. After 6 months, medication was increased as before for DBP greater than or equal to 100 mm Hg. In addition, if DBP was between 90 and 94 mm Hg at two visits with 3-month intervals, step-up therapy could be initiated. If the DBP was between 95 and 99 mm Hg at two visits with 2-week intervals, step-up therapy was initiated.

Participants were weighed in light clothing at monthly intervals. The data that we present are based on the weight change from baseline to the 6-, 18-, and 24-month visits.

Comparisons of baseline variables for all nine intervention groups were performed using χ² for categorical variables and one-way analysis of variance for continuous variables. Comparisons of 6-month changes for selected variables for the drug and diet groups were performed using two-way analyses of variance. Multiple linear regression analyses were also performed to examine the effect of β-blockade on 6-month weight change while controlling for several baseline factors.

Results

Baseline results have been previously described in detail. A summary of the baseline characteristics is presented here (Table 1). At baseline, all nine groups were comparable for almost all the major variables measured. There were no statistically significant differences between groups with regard to any of these variables except gender. There were more men than women in each of the nine treatment groups except the placebo/usual diet (40.0% male) and weight loss/chlorthalidone (47.0% male) groups.

Table 2 displays by treatment group, number of participants, mean weight change, mean pulse change, and mean kilojoule (kilocalorie) change and their standard errors at 6 months. The mean weight changes range from a loss of 6.9±0.5 kg in the chlorthalidone/weight loss group to a gain of 0.5±0.3 kg in the atenolol/usual diet group. The largest differences associated with the use of drug therapy were found in the participants randomly assigned to the weight loss group. The mean weight loss of those randomly assigned to the placebo group was 4.4±0.7 kg, those randomly assigned to the weight loss/chlorthalidone group lost 6.9±0.5 kg, and those randomly assigned to the weight loss/atenolol groups lost 3.0±0.4 kg. There were smaller but similar directional changes in those randomly assigned to the sodium-potassium diet group and to the usual diet group.

Within each diet group, the mean weight change of those assigned to receive atenolol therapy was the least. The only groups that showed a mean weight gain were those assigned to the atenolol/usual diet group and the atenolol/low sodium–high potassium diet group. There was a significant decrease in mean weight for those assigned to the weight loss diet versus those assigned to the usual or low sodium–high potassium diets (p<0.01) and for those assigned to chlorthalidone therapy versus those assigned to placebo or atenolol therapy (p<0.01).

The mean pulse rate changes range from a decrease of 12.0±1.2 beats per minute in the atenolol/weight loss group to one of 1.8±1.2 beats per minute in the placebo/usual diet group (Table 2). The largest decreases associated with the diets were found in the participants randomly assigned to atenolol therapy. The decrease in mean pulse rate for those randomly assigned to the usual diet group was 10.7±1.2 beats per minute; in those randomly assigned to the weight loss group, it was 12.0±1.2 beats per minute, and in those randomly assigned to the low sodium–high potassium diet group it was 9.9±1.1 beats per minute. There was a significant difference in mean pulse rate decrease for those assigned to atenolol therapy versus those assigned to placebo or chlorthalidone therapy (p<0.01).

Table 2 also shows the mean change in reported joule (calorie) intake at 6 months as assessed by diet diaries for those participants who had both diet diaries and measured weights at 6 months (n=685). The mean kilojoule (kilocalorie) changes range from a decrease of 1,869.0±281.0 kJ (445.0±66.9 kcal) in the atenolol/weight loss group to one of 210.4±317.9 kJ (50.1±75.7
kcal) in the atenolol/usual diet group. The smallest decreases associated with the use of drugs were found in the participants randomly assigned to atenolol therapy. The decrease in mean kilojoules in the weight loss group for those randomly assigned to the placebo group was 1,858.5±445.5 kcal (422.5±102.1 kcal); those randomly assigned to chlorthalidone therapy decreased their intake by 1,822.8±434.0 kcal (434.0±86.4 kcal); and those randomly assigned to atenolol therapy by 1,869.0±445.0 kcal (445.0±66.9 kcal). There was a significant decrease in those assigned to the usual low sodium–high potassium diet versus those assigned to the usual or low sodium–high potassium diet groups (p<0.01), but there was no significant difference by drug groups.

### Table 3. Multiple Linear Regression Analysis With 6-Month Weight Change as Dependent Variable

<table>
<thead>
<tr>
<th>Diet/drug group (n)</th>
<th>6-Month weight change in kilograms (SEM)</th>
<th>6-Month pulse change in beats per minute (SEM)</th>
<th>6-Month dietary intake change in kilojoules [kcal] (SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Usual diet</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (90)</td>
<td>−0.7 (0.4)</td>
<td>−1.8 (1.2)</td>
<td>−514.9 [−122.6] (285.6)</td>
</tr>
<tr>
<td>Chlorthalidone (87)</td>
<td>−1.5 (0.4)</td>
<td>−3.1 (1.1)</td>
<td>−541.8 [−120.9] (395.5)</td>
</tr>
<tr>
<td>Atenolol (87)</td>
<td>0.5 (0.3)</td>
<td>−10.7 (1.2)</td>
<td>−210.4 [−50.1] (317.7)</td>
</tr>
<tr>
<td><strong>Weight loss</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (89)</td>
<td>−4.4 (0.7)</td>
<td>−4.9 (1.0)</td>
<td>−1,858.5 [−442.5] (307.9)</td>
</tr>
<tr>
<td>Chlorthalidone (86)</td>
<td>−6.9 (0.5)</td>
<td>−4.2 (1.1)</td>
<td>−1,822.8 [−434.0] (249.1)</td>
</tr>
<tr>
<td>Atenolol (88)</td>
<td>−3.0 (0.4)</td>
<td>−12.0 (1.2)</td>
<td>−1,869.0 [−445.0] (281.0)</td>
</tr>
<tr>
<td><strong>Sodium–potassium</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (79)</td>
<td>−0.3 (0.4)</td>
<td>−2.7 (1.4)</td>
<td>−662.8 [−157.8] (355.3)</td>
</tr>
<tr>
<td>Chlorthalidone (89)</td>
<td>−1.0 (0.4)</td>
<td>−2.8 (1.3)</td>
<td>−1,046.6 [−249.2] (282.7)</td>
</tr>
<tr>
<td>Atenolol (90)</td>
<td>0.4 (0.3)</td>
<td>−9.9 (1.1)</td>
<td>−802.6 [−191.1] (313.7)</td>
</tr>
</tbody>
</table>

Table 3 presents a multiple linear regression analysis of 6-month change in weight as a function of the following baseline variables: drug group, diet group, age, race, sex, weight, diastolic blood pressure, exercise (0=≤average, 1=>average), smoking status (current/other), drinking status (≥1 drink/wk/other), prior use of antihypertensive drugs, pulse rate, dietary kilojoule intake, 24-hour urinary sodium and potassium, and glucose. Assignment to diuretic therapy resulted in a 1.4-kg weight loss (p<0.01), whereas assignment to β-blocker therapy resulted in a 1.1-kg weight gain (p=0.01). Assignment to the weight loss group yielded a 4.1 kg decrease in weight (p<0.01). The only other statistically significant variable was baseline weight (p<0.01). For every 5-kg increase in baseline weight, there was a 0.2-kg reduction in weight at 6 months.

Change in self-reported smoking and exercise status was further examined in the drug therapy groups to see if this might account for the weight gain in the atenolol-treated group. Twenty-two percent (25 of 112) of the baseline current smokers were non-smokers by 6 months, and in those assigned to atenolol treatment, this was 25% (9 of 36). Nine percent (55 of 587) of those participants who exercised at an average or below average level increased their amount of exercise by 6 months. In those assigned to atenolol therapy, the percentage was about the same (17 of 200).

Table 4 and Figure 1 present mean weight changes for those individuals who had weights measured at baseline, 6 months, 18 months, and 24 months (hereafter referred to as the 24-month weight loss cohort). The number of participants who had complete weight information at each 6-month follow-up visit was as follows: baseline, 878; baseline and 6 months, 785; baseline and 6 and 12 months, 675; baseline and 6, 12, and 18 months, 646; baseline and 6, 12, 18, and 24 months, 507. The weight changes for the 6-month, 12-month, 18-month, and 24-month weight loss cohorts were essentially the same within each treatment group at common time periods. All treatment groups tended to regain weight after 6 months. Note that the retrogression of weight in all drug groups on the weight loss diet appears to be at about the same rate (i.e., the slopes are similar). As a result, at 24 months mean weight in the atenolol group
TABLE 4. Mean Weight at Baseline and Weight Change From Baseline by Treatment Group for 24-Month Weight Loss Cohort

<table>
<thead>
<tr>
<th>Diet/drug group (n)</th>
<th>Baseline</th>
<th>6 Months</th>
<th>12 Months</th>
<th>18 Months</th>
<th>24 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Usual diet</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (61)</td>
<td>84.6 (1.5)</td>
<td>-0.5 (0.3)</td>
<td>-0.5 (0.4)</td>
<td>-1.0 (0.4)</td>
<td>-0.4 (0.5)</td>
</tr>
<tr>
<td>Chlorthalidone (52)</td>
<td>90.4 (2.0)</td>
<td>-1.9 (0.6)</td>
<td>-1.4 (0.5)</td>
<td>-0.7 (0.6)</td>
<td>-0.3 (0.5)</td>
</tr>
<tr>
<td>Atenolol (56)</td>
<td>89.0 (1.6)</td>
<td>0.3 (0.3)</td>
<td>0.6 (0.4)</td>
<td>1.2 (0.5)</td>
<td>0.6 (0.5)</td>
</tr>
<tr>
<td><strong>Weight loss</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (57)</td>
<td>89.1 (2.5)</td>
<td>-4.7 (0.9)</td>
<td>-3.7 (0.9)</td>
<td>-2.7 (1.0)</td>
<td>-1.9 (1.0)</td>
</tr>
<tr>
<td>Chlorthalidone (58)</td>
<td>87.8 (1.7)</td>
<td>-7.4 (0.7)</td>
<td>-5.6 (0.7)</td>
<td>-4.6 (0.7)</td>
<td>-3.4 (0.7)</td>
</tr>
<tr>
<td>Atenolol (54)</td>
<td>86.3 (1.8)</td>
<td>-3.5 (0.6)</td>
<td>-1.9 (0.6)</td>
<td>-0.9 (0.6)</td>
<td>-0.2 (0.5)</td>
</tr>
<tr>
<td><strong>Sodium-potassium</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (55)</td>
<td>86.9 (2.0)</td>
<td>-0.3 (0.5)</td>
<td>-0.6 (0.6)</td>
<td>0.3 (0.5)</td>
<td>0.4 (0.6)</td>
</tr>
<tr>
<td>Chlorthalidone (54)</td>
<td>85.9 (1.6)</td>
<td>-1.5 (0.5)</td>
<td>-0.9 (0.6)</td>
<td>-0.3 (0.6)</td>
<td>-1.4 (0.9)</td>
</tr>
<tr>
<td>Atenolol (60)</td>
<td>90.2 (1.8)</td>
<td>-0.1 (0.4)</td>
<td>0.5 (0.5)</td>
<td>0.5 (0.5)</td>
<td>0.4 (0.6)</td>
</tr>
</tbody>
</table>

Values are in kilograms (SEM).

had almost returned to baseline (−0.2 kg) but that in the chlorthalidone group was still at −3.4 kg.

Table 5 displays mean 6-month changes in weight, pulse rate, and dietary kilojoules in the weight loss group by race and sex. The least weight loss occurred in those receiving atenolol therapy for all race-sex groups. In two-way analyses of variance, there was a statistically significant effect for both drug (p<0.01) and race-sex (p=0.02) subgroups. The greatest decrease in mean pulse occurred in those receiving atenolol therapy in all the race-sex groups except black men. In two-way analyses of variance, there was a statistically significant effect for drug (p<0.01) but not for race-sex (p=0.02) subgroups. There was no consistent pattern or statistically significant results across the race-sex groups or drug therapy groups for change in mean dietary kilojoules.

**Discussion**

There were marked differences in success of the weight loss program at 6 months, with the chlorthalidone-treated patients losing 6.9±0.5 kg, the placebo-treated patients losing 4.4±0.7 kg, and the atenolol-treated group losing 3.0±0.4 kg. Although all the weight loss groups tended to regain weight with continued follow-up, the drug effect was marked enough so that only the atenolol group was above baseline by 24 months. Similar trends occurred in the patients randomly assigned to the regular diet group and to the low sodium–high potassium diet group although the results were not as marked.

This report is not the first time that an effect of β-blocking agents on weight has been reported. The Heart Attack Primary Prevention in Hypertension trial reported that "the cause of the weight gain in the metoprolol-treated patients was not apparent." Rossner reported long-term weight gain in propranolol-treated, post–myocardial infarction patients.

There are several explanations that can be given for these somewhat unexpected results:

1. Although the differences are significant, the results could have occurred by chance. This is a post hoc analysis, and no hypothesis was entertained about diet–drug interactions as far as weight loss or gain was concerned at the initiation of the trial. However, the fact that the trends have continued as the trial has gone forward and the fact that β-blocking agents were associated with weight gain in the two studies noted above suggest that the finding is valid, at least as far as β-blocking agents are concerned.

2. Part of the difference may be related to fluid changes produced by drugs. Chlorthalidone does pro-
reduce sodium and water loss and β-blockers do not.11 Some investigators have shown decreases of extracellular fluid volume after initiation of diuretic therapy, but there was usually a return to the initial level, although not a complete return, after a few weeks or months of treatment.12-15 Plasma volume measurements after chronic diuretic therapy have given conflicting results. Some investigators have reported that the initial reduction was not maintained while others have reported a persistent reduction.16,17

3. Antihypertensive drugs could influence physical activity so that there may be secondary effects on weight loss. The lesser weight loss in participants randomly assigned to atenolol therapy could be due to unnoticed decreases in physical activity produced by the sense of tiredness that often accompanies β-blockade therapy, but this is usually not a permanent complaint with atenolol, nor was it in this study. However, the decrease in anxiety often noticed with β-blockers may decrease purposeless movement ("fidgeting"). Moreover, participants randomly assigned to the chlorthalidone/weight loss group lost more than those randomly assigned to the placebo/weight loss group. One would not anticipate increased physical activity as the result of chlorthalidone therapy, for if it produces fatigue, the patient should move less, not more.

4. The most likely explanation for the differences in weight loss seen here in the participants randomly assigned to various drug therapies can be related to the contrasting effects on catecholamines of diuretics and β-blockers. Studies of β-blocking agents have shown conflicting results with regard to increasing levels of catecholamines.18 However, some investigators have demonstrated sympathetic blockade with the use of these agents.19,20 If β-blockers prevent the action of catecholamines on fatty acid mobilization, this might decrease the availability of fatty acids and their feedback on appetite centers. However, as shown in Table 1, there was no significant difference by drug groups in reported joule (calorie) intake.

The greater weight loss in the thiazide-treated patients can be explained as the complement of the possible β-blocking effect. The volume depletion, and perhaps the potassium depletion, of thiazide therapy increases serum and urinary catecholamines, which could reduce appetite and increase fat mobilization. We suggest that the greater prominence of these differences among drugs in participants randomly assigned to the weight reduction group than in those randomly assigned to other diets is related to the fact that the other individuals were eating primarily from habit. Individuals attempting to change their weight were perhaps more susceptible to stimuli that affected appetite. However, appetite and fat mobilization may not be the only way that catecholamines affect weight. Increased circulatory catecholamine might lead to greater spontaneous activity and blocking catecholamines to less spontaneous movement.

The weight gain seen upon stopping smoking may be a similar phenomenon to the blunting of weight loss by β-blockers reported here. It has been reported that nicotine increases both the resting and the exercise metabolic rate.21 β-Blockade therapy and withdrawal of smoking both would be expected to lower metabolic rate. Campbell et al22 have shown that propranolol

<table>
<thead>
<tr>
<th>Drug Group</th>
<th>Weight loss in kilograms</th>
<th>Prolonged change in beats per minute</th>
<th>Diastolic pressure change in kilogram (diuretic)</th>
<th>Diuretic use</th>
<th>Chlorthalidone/weight loss</th>
<th>Chlorthalidone/placebo</th>
<th>Others</th>
<th>Others</th>
<th>Others</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>placebo</td>
<td>-3.4 (1.2, 4.0)</td>
<td>-0.3 (0.0, 0.6)</td>
<td>-0.4 (0.0, 0.8)</td>
<td>-0.4</td>
<td>-0.5 (0.0, 0.8)</td>
<td>-0.6 (0.0, 0.8)</td>
<td>-0.7</td>
<td>-0.7</td>
<td>-0.7</td>
<td>-0.7</td>
</tr>
<tr>
<td>atenolol</td>
<td>-2.8 (1.0, 4.0)</td>
<td>-0.3 (0.0, 0.6)</td>
<td>-0.4 (0.0, 0.8)</td>
<td>-0.4</td>
<td>-0.5 (0.0, 0.8)</td>
<td>-0.6 (0.0, 0.8)</td>
<td>-0.7</td>
<td>-0.7</td>
<td>-0.7</td>
<td>-0.7</td>
</tr>
<tr>
<td>chlorthalidone</td>
<td>-3.3 (1.6, 3.7)</td>
<td>-0.3 (0.0, 0.6)</td>
<td>-0.4 (0.0, 0.8)</td>
<td>-0.4</td>
<td>-0.5 (0.0, 0.8)</td>
<td>-0.6 (0.0, 0.8)</td>
<td>-0.7</td>
<td>-0.7</td>
<td>-0.7</td>
<td>-0.7</td>
</tr>
</tbody>
</table>

Values given in parentheses are SEM and number of subjects.
lowers the postprandial increase in metabolic rate. Studies in the Pima Indians have shown that there are individual differences in metabolic rates that correlate with the tendency to become obese. An atypical \( \beta \)-adrenergic receptor agonist, BRL26830A, has been shown to be effective in promoting weight loss. It seems reasonable to assume that blocking \( \beta \)-adrenergic receptors, in the case of atenolol, would have the opposite effect, and that increasing \( \beta \)-agonist secretion, in the case of a diuretic, would have the same effect as the agonist in promoting weight loss.

The association of atenolol with decreased weight loss was more marked in black women than in whites. One explanation for this may be the "volume-expanded" state of many black hypertensive patients, which should lead to decreased sympathetic activity. \( \beta \)-Block- ing therapy, therefore, could suppress more completely sympathetic drive, with attendant effects on weight. Alternatively, with a lower sympathetic drive, sympathetic blockade would have less effect.

These results should not be used to suggest that \( \beta \)-blocking agents should be avoided in obese hypertensive individuals but to suggest that weight loss may be more difficult and will require greater attention from the patient, the nutritionist, and the physician. Previous reports from TAIM1-2 have shown that weight loss plus atenolol therapy did quite well in lowering DBP and cardiovascular risk.

**Appendix**

**Trial of Antihypertensive Interventions and Management Participating Institutions and Investigators**

- **Birmingham Clinical Center** (University of Alabama School of Medicine): Albert Oberman, MD (Principal Investigator); Neil Rappaport, PhD; James M. Raczynski, PhD; Heidi Hataway, MD, RD; Gary R. Cutter, PhD

- **Jackson Clinical Center** (University of Mississippi Medical Center): Herbert G. Langford, MD (Principal Investigator) (deceased); Sheila Corrigan, PhD; Lori Danks, BS, RN; Stephanie Jennings, MS, RD

- **New York Clinical Center** (Albert Einstein College of Medicine): M. Donald Blaupox, MD, PhD (Principal Investigator); Maureen Magnani, RN; Judy Stern, BS, RD; Gail Miller, RN

- **Nutrition and Behavioral Core** (Albert Einstein College of Medicine): Sylvia Wassertheil-Smoller, PhD (Director); Judith Wylie-Rosett, EdD, RD; Charles Swencionis, PhD; Yvonne P. Raiford

- **Coordinating Center** (University of Texas School of Public Health): C. Morton Hawkins, ScD (Principal Investigator); Barry R. Davis, MD, PhD; Neal Zimbaldi, BS; Maura O'Connell Knerr, MS

- **Central Laboratory** (University of Minnesota School of Public Health): Sue Bird

- **24-Hour Urinalysis Laboratory** (University of Mississippi Medical Center): K.T. Holder, CLS(C)

- **Plasma Renin Activity Laboratory** (Albert Einstein College of Medicine): Hye-Bok Lee, MS

- **Policy Advisory Committee**: Lloyd Filer, MD (Chairman) (University of Iowa); W. Gordon Walker, MD (Johns Hopkins University); Max Haiperin, PhD (George Washington University) (deceased); Elisabeth McSherry, PhD, MPH (VA Medical Center, Boston); Pat Elmer, PhD (University of Minnesota); Thomas P. Blaszkowski, PhD (NHLBII)

**References**


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