Can Dietary Interventions Change Diet and Cardiovascular Risk Factors? A Meta-Analysis of Randomized Controlled Trials

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ABSTRACT

Objectives. This study evaluated the effectiveness of dietary advice in primary prevention of chronic disease.

Methods. A meta-analysis was conducted of 17 randomized controlled trials of dietary behavior interventions of at least 3 months' duration. Results were analyzed as changes in reported dietary fat intakes and biomedical measures (serum cholesterol, urinary sodium, systolic and diastolic blood pressure) in the intervention group minus changes in the control group at 3 to 6 months and 9 to 18 months of follow-up.

Results. After 3 to 6 months, mean net changes in each of the five outcomes favored intervention. For dietary fat as a percentage of food energy, the change was −2.5% (95% confidence interval [CI] = −3.9%, −1.1%). Mean net changes over 9 to 18 months were as follows: serum cholesterol, −0.22 (95% CI = −0.39, −0.05) mmol/L; urinary sodium, −45.0 (95% CI = −57.1, −32.8) mmol/24 hours; systolic blood pressure, −1.9 (95% CI = −3.0, 0.8) mm Hg; and diastolic blood pressure, −1.2 (95% CI = −2.6, 0.2) mm Hg.

Conclusions. Individual dietary interventions in primary prevention can achieve modest improvements in diet and cardiovascular disease risk status that are maintained for 9 to 18 months. (*Am J Public Health.* 1997; 87:1415–1422)

Introduction

Population dietary change is advocated to reduce the incidence of cardiovascular disease and certain cancers,[1–5] but there is uncertainty about the effectiveness of such strategies.[6–8] Dietary modifications are effective in modifying risk when adherence is high. For example, changes in the quantity and quality of dietary fat improve the lipid profile,[9] and blood pressure is lowered by reducing sodium[10] and increasing potassium intake.[11] These findings are based on trials involving well-motivated individuals, often in metabolic wards,[9] living in institutions,[12–14] or receiving treatment in a hospital clinic.[15] This study investigated whether such findings can be applied to the general population. A systematic search for randomized controlled trials of dietary advice designed for the primary prevention of chronic disease identified 17 suitable studies.[16–32] In this paper we use meta-analysis[33] to summarize the evidence on effectiveness.

Methods

Identification of Trials

We included all trials, including conference abstracts, published as of July 1993 and fulfilling the following criteria: (1) the subjects were free-living adults; (2) the trial involved primary prevention (i.e., less than 25% of subjects had diagnosed disease, including hypertension, under treatment before the start of the trial[34]); (3) the intervention group was encouraged to consume a diet aimed at changing patterns of fat, sodium, or fiber consumption; (4) subjects were randomized (or systematically allocated[17]) to an intervention or control group; and (5) the trial lasted at least 3 months.

A trial was excluded if a supplementation diet was used,[35] if the groups differed in ways other than the dietary intervention, if meals were provided for subjects,[12,14,36] or if a crossover design was used. Multiple-intervention trials[37,38] were also excluded, as was one trial[39] of diet vs exercise advice. Eight hundred seventy-seven references, including duplicates, were identified by computer and manual searches of databases and journals.[40] Abstracts were screened by Amanda Bristow according to the inclusion criteria; this screening identified 48 potentially suitable trials. Published reports of these trials were independently and systematically reviewed by Amanda Bristow, Eric Brunner, Margaret Thorogood, and Ian White. Seventeen trials were used in the final meta-analysis. Authors were contacted to obtain unpublished data for some of the studies.

The outcome measures used were serum total cholesterol (mmol/L), diastolic blood pressure (mm Hg), urinary sodium (mmol/24 hours), and calories from fat as a percentage of total nonalcohol calories. Reporting bias in a trial of dietary intervention can be considerable,

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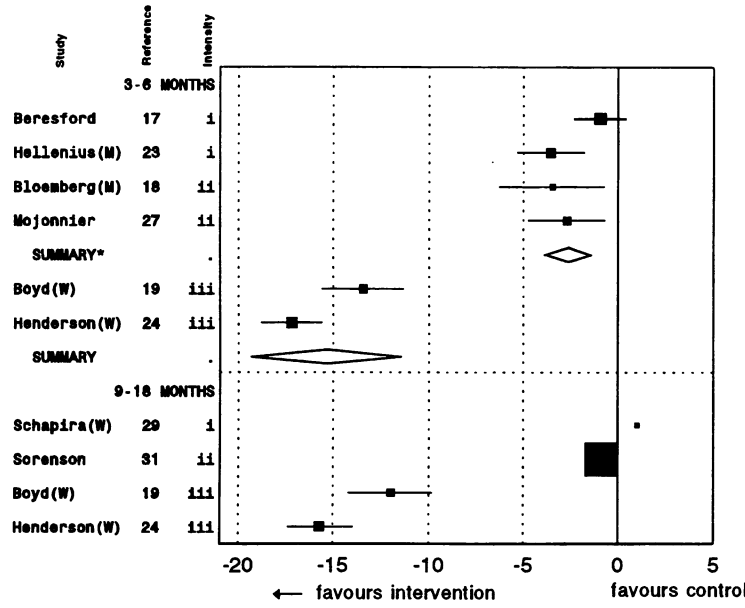


Note. Trials are ordered into three groups by frequency of contacts with intervention subjects. Diamonds indicate combined results. Net effect estimates for individual trials are shown as filled boxes of the area proportional to 1/(standard error),[2] along with 95% confidence intervals. Summary estimates, derived from a random effects model, are represented as diamonds; widths show 95% confidence intervals. Number of contacts are as follows: (i) 2–3, (ii) 5–9, (iii) 10–32. *Summary estimate excludes the two trials of women with raised breast cancer risk. M = trial with men only; W = trial with women only.

FIGURE 1—Estimate of effect of dietary advice on percentage of food energy from fat in trials of 3 to 6 months' and 9 to 18 months' duration.

particularly if intake is assessed by food frequency questionnaire.[41,42] Biomedical outcomes avoid this bias as well as being closer to the disease process than diet measures. When possible, two time points were used: the point nearest to 3 months in the range 3 to 6 months and the point nearest to 12 months in the range 9 to 18 months.

Statistical Methods

Intervention effects were estimated by comparing mean changes in the intervention and control groups.[43] We calculated this measure, together with its standard error, from the published paper or from the raw data that investigators supplied. In certain cases, we were forced to use less than optimal, but still unbiased, methods: estimation of treatment effects and standard errors from graphs[28,32] and comparison of follow-up values without use of baseline data.[21,24,27,29] In trials[21,22] involving three randomized groups, we compared the most intensive and least intensive interventions. A trial with a complex design[25] was analyzed within strata according to whether subjects received calorie restriction advice.

Pooled intervention effects were summarized by means of a random effects meta-analysis[33] that weighted by the inverse of the sum of the between-studies variance and the variance of the study intervention effect. Heterogeneity was assessed by the Q statistic, a weighted between-studies sum of squares. When heterogeneity is identified among trials, the summary statistic for the given outcome is, to an extent, dependent on the balance of trials included in the calculation (see Figure 1 for an example). We examined subgroups defined by factors potentially relevant to effectiveness: frequency of follow-up contacts in the intervention group (2 or 3, 5 to 9, 10 to 32 contacts); perceived level of risk (four categories: normal/population risk [four trials[16,17,22,31]], hypercholesterolemia [five trials[18,20,21,23,27]], hypertension [five trials[25,26,28,30,32]], and women with elevated breast cancer risk [three trials[19,24,29]]); proportion of missing data; blinding of blood pressure measurement; dietary assessment method; and proportion of female subjects.

Description of Trials

The 17 trials used in the analyses included 6893 participants (an estimated 51% of whom were women), with 3736 in the intervention groups. Of the participants, 3817 were randomized individually and 3076 were randomized by workplace.[31] Table 1 shows details, including dietary aims. The proportions of missing follow-up data were in the 1% to 30% range in 14 trials and in the 32% to 49% range in 3 trials.[20,29,30] Standard errors could not be calculated for two trial effects: dietary fat[29] and urinary sodium.[28] Dietary intake was assessed in 9 trials. Self-administered methods were used with the exception of one study[18] in which a diet history was taken by a dietitian. Five studies used diet records (2 used 3-day records,[19,27] 2 used 4-day records,[24,29] and 1 used a 7-day record[23]), and 3 used a food frequency questionnaire (two based on the Willett method[17,31] and one designed to assess dietary fiber only[16]). Only 2 trials involving the polyunsaturated–saturated fatty acid ratio were identified[18,27]; a meta-analysis was not performed on these trials.

Results

In the figures, trials are ordered according to follow-up frequency. Net effect estimates for individual trials are shown as filled boxes whose areas represent the precision of each trial result (see figure notes).

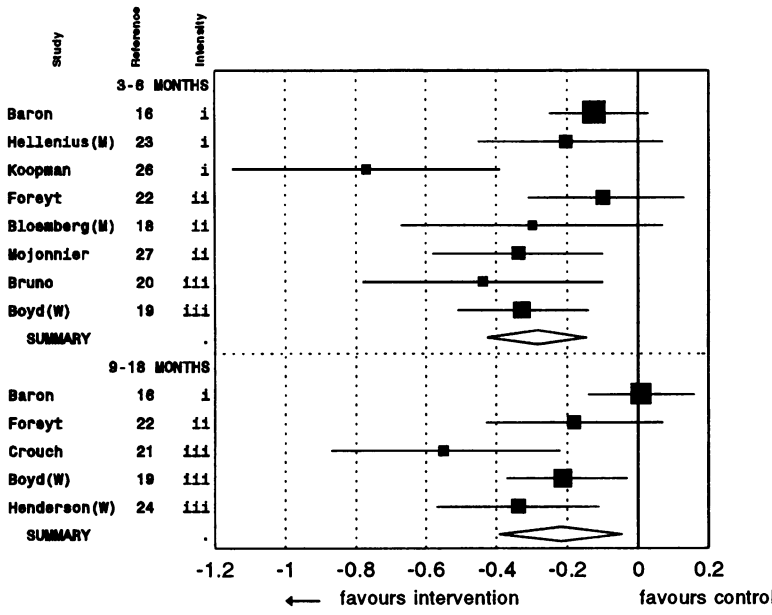
Reported Diet Outcomes: Dietary Fat

All six 3- to 6-month trials showed effects favoring intervention (Figure 1). Effect sizes varied considerably, with formal evidence of heterogeneity at 3 to 6 and 9 to 18 months of follow-up (both $Ps < .0001$). The first summary statistic in Figure 1 omits the trials in breast cancer prevention, which obtained the largest reductions in intake. The smaller trial effects over 3 to 6 months, involving adults without risk elevation[17] or with mildly raised cardiovascular risk,[18,23,27] were not heterogeneous ($P = .32$) and indicate a proportional net reduction of some 6% of energy from fat, as compared with 40% in the breast cancer prevention trials. No summary statistic was calculated for the 9- to 18-month trials. At 1-year follow-up, one of the three breast cancer prevention[29] trials obtained an

TABLE 1—Single-Factor Randomized Controlled Trials of Dietary Interventions: Subjects, Nature of Intervention, Dietary Aims, and End Points

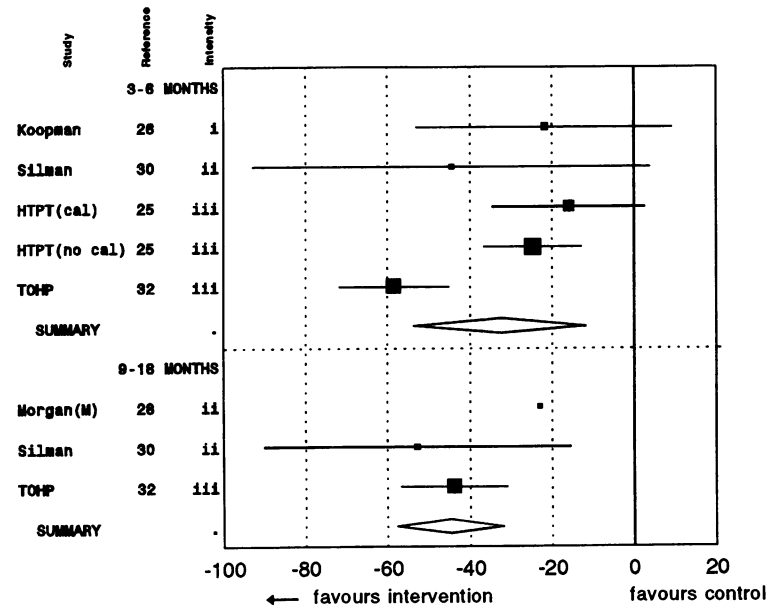
Study (Country)	Site	Subjects	INT, No.	CON, No.	Women, %	Missing, %	Nature of Intervention	Dietary Aims	End Point(s)
Baron et al.[16] (United Kingdom)	Primary care	Random sample 25–60 years of age	187	181	49	11	Nurse advice, small group or individual	Fat 30%–35% of energy; P:S ratio 0.4; increased fiber	Total cholesterol
Beresford et al.[17] (United States)	Primary care	Patients attending family doctor	120	122	73	22	Telephone reinforcement of self-help material	Fat 30% of energy; 20–30 g fiber/day	Total fat as % of food energy
Bloemberg et al.[18] (Netherlands)	Not stated	Screenees with cholesterol levels of 6.5–10 mmol/L	39	41	0	1	Individual dietitian advice, phone and mail support	Fat 30% of energy; P:S ratio 1.0; cholesterol 20 mg/MJ	Total fat as % of food energy; P:S ratio; total cholesterol
Boyd et al.[19] (Canada)	Breast clinic	Patients with breast dysplasia	148[a]	147[a]	100	30	Individual dietary advice	Fat 15% of energy	Total fat as % of food energy; total cholesterol
Bruno et al.[20] (United States)	Occupational health department	Nonobese employees with cholesterol levels of 6.9–9.1 mmol/L and BP <160/95 mm Hg	97	48	22	32	8-week group programs during lunch hour	Reduced fat, salt, sugar	Total cholesterol
Crouch et al.[21] (United States)	Clinic office	Screenees with cholesterol levels of 5.8–6.9 mmol/L	26	30	25	13	Face-to-face or mail/phone counseling	Reduced saturated fat and dietary cholesterol	Total cholesterol; BP
Foreyt et al.[22] (United States)	Special clinic	Volunteers recruited via newspapers	75	91	47	28	Diet booklet or nutrition education with or without behavior intervention	Fat 35% of energy	Total cholesterol; BP
Hellenius et al.[23] (Sweden)	Not stated	Patients with cholesterol levels of 5.2–7.8 mmol/L; triglycerides <5.6 mmol/L; glucose <6.7 mmol/L; diastolic BP <100 mm Hg	79	79	0	8	Individual advice from doctor and dietitian	Fat 30% of energy	Total fat as % of food energy; total cholesterol; BP
Henderson et al.[24] (United States)	Special clinic	Patients at increased risk of breast cancer	184	119	100	6	Regular group sessions with nutritionist	Fat 20% of energy	Total fat as % of food energy; total cholesterol; P:S ratio
HTPT[25] (United States)	Not stated	Volunteers with diastolic BP of 76–99 mm Hg	520	321	35	10	Weekly group counseling	(1) Reduced sodium; (2) reduced sodium and increased potassium	BP; urinary sodium
Koopman et al.[26] (Netherlands)	Primary care	Nonobese patients with diastolic BP of 90–110 mm Hg	17	18	54	16	Monthly visits with dietitian	PUFAs 10%–12% of energy; fiber 30 g; sodium 85–100 mmol; potassium 76–100 mmol daily	Total cholesterol; BP; urinary sodium
Mojonnier et al.[27] (United States)	Special study center	Screenees with cholesterol levels of >5.8 mmol/L	333[a]	84[a]	60	30	Weekly teaching sessions (self, group, or individual)	Fat 30% of energy; <10% saturates; <10% PUFAs; <300 mg cholesterol	Total fat as % of food energy; P:S ratio; total cholesterol
Morgan et al.[28] (Australia)	Hypertension clinic	Patients with diastolic BP of 95–109 mm Hg	34	33	0	7	Repeated individual dietary advice	Sodium intake 70–100 mmol/day	BP; urinary sodium
Schapira et al.[29] (United States)	Not stated	Patients at increased risk of breast cancer	60	60	100	49	Group discussion	Fat 30% of energy; 25–30 g fiber	Total fat as % of food energy
Silman et al.[30] (United Kingdom)	Primary care	Screenees with diastolic BP of 95–104 mm Hg	12	16	Not known	33	Individual advice	Sodium intake 100 mmol/day	BP; urinary sodium
Sorenson et al.[31] (United States)	Work sites	All employees at 16 work sites randomized by work site	1449[a]	1627[a]	52	30	Classes, demonstration, leaflets	Reduced fat; increased fiber	Total fat as % of food energy
TOHP[32] (United States)	Special clinics	Volunteers with diastolic BP of 80–89 mm Hg	327	417	29	13	Group and individual sessions	Reduced sodium intake	BP; urinary sodium

Note. INT = Intervention; CON = Control; P:S ratio = polyunsaturated:saturated fatty acid ratio; MJ = megajoule; BP = blood pressure; PUFAs = polyunsaturated fatty acids; HTPT = Hypertension Prevention Trial Research Group; TOHP = Trials of Hypertension Prevention Collaborative Research Group.
[a] Our estimate.



Note. For explanation, see Figure 1 note.

FIGURE 2—Estimate of effect of dietary advice on serum cholesterol (mmol/L) in trials of 3 to 6 months' and 9 to 18 months' duration.



Note. For explanation, see Figure 1 note. HTPT = Hypertension Prevention Trial; cal = calorie advice; no cal = no calorie advice; TOHP = Trials of Hypertension Prevention.

FIGURE 3—Estimate of effect of dietary advice on urinary sodium excretion (mmol/24 hours) in trials of 3 to 6 months' and 9 to 18 months' duration.

increase of 1% in terms of percentage of energy from fat. In this trial, the control group also received dietary advice (one 2-hour education session), and mean intakes of energy from fat were lower in both the control group (28%) and the intervention group (29%). The large worksite trial[31] achieved a marginal reduction in mean percentage of energy from fat at 15 months.

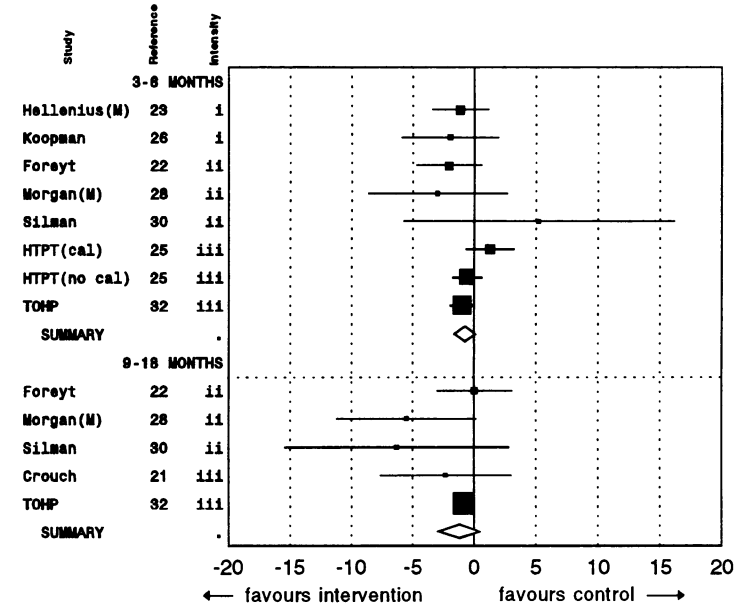
Biomedical Outcomes

Serum cholesterol. Of the eight 3- to 6-month trials (Figure 2), two[16,22] involved subjects at population levels of coronary risk. These trials obtained statistically nonsignificant reductions of approximately 0.1 mmol/L. A study of women with mammographic dysplasia[19] obtained a net change in cholesterol of −0.3 (95% confidence interval [CI] = −0.5, −0.1) mmol/L at 3 months (from a mean level at randomization of less than 5.0 mmol/L). There was significant heterogeneity ($P < .05$) resulting from a trial in mild hypertension,[26] involving monthly visits from a dietitian, that obtained the largest net decrease in serum cholesterol. The estimated overall mean net reduction in serum cholesterol was −0.28 (95% CI = −0.42, −0.15) mmol/L at 3 to 6 months. Five trials with a follow-up period of 9 to 18 months (Figure 2) yielded a summary effect of −0.22 (95% CI = −0.39, −0.05) mmol/L. Three of these trials[16,19,22] were among the trials described earlier; two obtained smaller reductions in serum cholesterol at a longer follow-up,[16,19] while in the third,[22] the reduction tended to be larger at 1 year than at 3 months. The heterogeneity test was significant ($P < .02$), reflecting the outlying nature of two studies.[16,21] Frequency of intervention contacts appeared to be associated with size of net effect in both sets of cholesterol results.

Urinary sodium. At 3 to 6 months, the Hypertension Prevention Trial[25] provided two data points: (1) the sodium/calorie restriction arm and (2) the sodium restriction and reduced sodium/increased potassium arms, which were pooled (Figure 3). The overall mean net reduction of some 32 mmol/24 hours was equivalent to 1.9 g NaCl, or a 20% reduction in salt intake. The heterogeneity test was highly significant ($P < .0005$) for the 3- to 6-month trials, because the net reduction of 59 (95% CI = 45, 72) mmol/24 hours[32] was an outlier result. At 9 to 18 months, the summary effect for the two trials[30,32] with standard errors was somewhat larger than at 3 to 6 months.

Diastolic blood pressure. The overall net change at 3 to 6 months was −0.7 (95% CI = −1.5, 0.0) mm Hg (P = .06) (Figure 4). Except for two trials,[25,30] the trial results favored intervention. In the five trials combined at 9 to 18 months, the net effect of −1.2 (95% CI = −2.6, 0.2) mm Hg was marginally significant (P = .09). There was no statistical evidence of heterogeneity between trials for either of the duration periods (3 to 6 months, P = .33; 9 to 18 months, P = .31). Results from the Trial of Hypertension Prevention[32] overshadowed the other effect estimates.

Systolic blood pressure. A meta-analysis involving systolic blood pressure was conducted with the same set of trials as for diastolic blood pressure (data not shown). The net effect at 3 to 6 months of −1.3 (95% CI = −2.4, −0.3) mm Hg was significant (P = .01). There was no statistical heterogeneity between effects (P = .27). At 9 to 18 months, the net effect was −1.9 (95% CI = −3.0, −0.8) mm Hg (P < .001), again with no evidence of heterogeneity (P = .82).



Note. For explanation, see Figure 1 note. HTPT = Hypertension Prevention Trial; cal = calorie advice; no cal = no calorie advice; TOHP = Trials of Hypertension Prevention.

FIGURE 4—Estimate of effect of dietary advice on diastolic blood pressure (mm Hg) in trials of 3 to 6 months' and 9 to 18 months' duration.

Potential Effect Modifiers

Summary statistics for the trials were ordered by the proportion of missing data, whether blood pressure measurement was blinded, method of diet assessment, and the proportion of female subjects. There was no relationship between any of these factors and effect size.

Public Health Impact of Dietary Interventions

Estimates, corrected for regression dilution bias, have been made of the effects of reductions in serum cholesterol[44] and diastolic blood pressure[45] on the incidence of coronary heart disease and stroke. On the basis of primary prevention trials, it has been estimated[44] that a 10% reduction (0.6 mmol/L) in serum cholesterol will reduce coronary heart disease by 25%. A 5-mm Hg reduction in diastolic blood pressure, based on cohort studies,[45] predicts a 21% reduction in coronary heart disease and a 34% reduction in stroke. Applying these estimates to our summary effects at 9 to 18 months, we calculated that dietary intervention could reduce coronary heart disease incidence by 14% (9% as a result of cholesterol lowering) and stroke by 8%. These estimates assumed that the observed changes in dietary habits would be sustained and that the reductions in risk attributable to the changes in cholesterol and diastolic blood pressure could be combined additively.

Discussion

Main Findings

Our overview shows that dietary advice can lead to change in diet and to modest risk reduction among healthy adults. Weighted mean net changes over 9 to 18 months were −0.22 (95% CI = −0.39, −0.05; P < .01) mmol/L (3.7% proportional reduction) for serum cholesterol and −1.2 (95% CI = −2.6, 0.2; P = .09) mm Hg (1.4% proportional reduction) for diastolic blood pressure. Trials with serum cholesterol as an outcome suggest that increasing intensity of intervention has a small positive effect on dietary change. A higher perceived level of risk, as in the trials of breast cancer prevention, appears to motivate a greater reduction in dietary fat intake than that observed among groups with moderately elevated or unknown cardiovascular risk factors. We used biomedical variables, as well as reported diet, as markers of dietary change rather than as a test of the diet–risk factor relationship.

The evidence on change in cardiovascular risk achievable with reductions in serum cholesterol and blood pressure is now strong.[15,44,46–50] The question we have addressed is whether a diet strategy aimed at such changes is effective in the general population. Multiple-intervention trials were excluded in order to maximize the clarity of our findings. To our knowledge, no systematic study has been conducted of trials in which participants were free living, did not have symptomatic cardiovascular disease or other chronic disease, and were not taking cardiovascular drugs.

Data Quality

Long-term follow-up would be required to estimate the real effectiveness of preventive interventions, but the biomedical outcomes can be regarded as proxies for disease outcomes. Blood pressure, serum cholesterol, and urinary sodium are less subjective measures than reported diet and represent physiological changes attributable to the interventions of interest. It is plausible that some of the net differences in blood pressure may have been due to a relative decline in arousal in the intervention groups as a result of the

frequency of contacts with trial personnel. This consideration does not apply to serum cholesterol or urinary sodium.

Our summary statistics may be misleading as a result of publication bias,[51] which favors publication of small trials with positive results. Funnel plots[51] for 3- to 6-month trials involving cholesterol, urinary sodium, and blood pressure (data not shown) do not suggest that this was a serious problem. When respondents are unavoidably aware of the trial objective, the use of diet recall methods such as food frequency questionnaires in trials of dietary behavior interventions raises the question of reporting bias.[42] Self-reported diet methods have not been validated for use in intervention trials. Food frequency methods are relatively imprecise for individual dietary assessment but may be suitable for characterizing the intake of trial groups.[52]

The incompleteness of follow-up data excluded an intention-to-treat analysis and represents a source of bias. Loss of subjects after recruitment may overestimate effectiveness, since dropouts are likely to be less compliant. Missing follow-up data were at low levels in most trials. In three cases, summary statistics included trials with more than 30% of outcome values missing. The weights assigned to these trials in the calculations were small. The effect size obtained in a diet trial is dependent on willingness to alter diet, and the impact of an intervention in the population may be overestimated if trial subjects are selected for compliance, as was the case in some of the trials[20,24,32] included here. Impact may be underestimated if the control group knows about the intervention, as clearly occurred in one study.[29] These effects could not be controlled in our analyses. Related to this, our overview is restricted by the nature of evidence suitable for meta-analysis. Public health measures such as mass media campaigns are not readily amenable to testing in a trial format but may nevertheless influence national dietary patterns.

Motivation

Adherence to dietary advice may be influenced by factors such as perception of future disease risk and nature of the intervention. We were not able to test formally the effects of these factors as a result of lack of data. An effect of level of perceived risk was clearest in two trials[19,24] of dietary prevention of breast cancer for percentage of energy from fat. Using relatively unbiased diet record methods,[53] these studies each obtained approximately fourfold greater net reductions in reported fat intake than trials involving other subjects (Figure 1). The effect of the intensity of advice was assessed by ordering the trials according to the frequency of follow-up contacts in the intervention group. The data are not sufficient to estimate this effect, but they suggest that a low-intensity approach may be as effective as one involving considerable resources. In one trial,[18] for example, the intervention consisted of individual advice from dietitians at baseline, with eight telephone and postal follow-up contacts. In another,[23] advice was given by a physician at randomization, followed by a single visit to a dietitian and one phone contact after 3 months. Net changes in percentage of energy from fat and serum cholesterol were similar in these two groups of men chosen on similar entry criteria.

There is little evidence on maintenance of change following dietary intervention beyond 18 months. The Women's Health Trial follow-up[54] found that reductions in reported fat intake (from 40% to 27% of energy) were maintained 4 years after baseline, 1 year after the trial ended. A small trial of salt restriction in mildly hypertensive subjects[28] showed blood pressure reductions maintained over 2 years. In a larger trial,[25] mean sodium excretion initially decreased by some 45 mmol/24 hours from baseline but then returned to 20 mmol/24 hours below baseline at 3 years. Multifactorial trials provide further evidence on long-term changes. The Stanford Project[55] showed scant evidence for the effectiveness of 2.5- to 5-year communitywide interventions assessed at 6 years with cardiovascular risk factor surveys. On the other hand, the Multiple Risk Factor Intervention Trial[38] found persisting net reductions of serum total and low-density lipoprotein cholesterol some 3 years after the end of the 6- to 8-year trial, and the Oxcheck study[56] demonstrated net benefits over 3 years.

Dietary Sodium

Current United Kingdom recommendations[1] call for a reduction in the average adult intake of sodium from approximately 150 to 100 mmol/24 hours. Our analyses suggest that a reduction in salt intake of some 30 mmol or 2 g/24 hours is achievable among normotensive[25,32] as well as mildly hypertensive[26,28] individuals. We did not identify any trials involving measurements of urinary sodium that used a low-intensity intervention design. Whether one or two advice sessions, rather than more frequent contacts, would be effective remains an open question.

Population Diet Strategy

Reduction in the incidence of premature coronary heart disease by dietary modification continues to be a key public health target.[1–5] Strategies for dietary change may target the whole population by means of education, mass media, and economic policy, or they may involve individual advice. Trial evidence for the effectiveness of individual advice in reducing saturated fatty acid intake is sparse[18,24,27] but suggests that the 10% population average target is achievable. An increase of some 60% in the polyunsaturated–saturated fatty acid ratio and a reduction of 10% in saturated fatty acid content in the British diet appear to have taken place during the 1980s,[57] indicating that national dietary habits are not fixed. Our analysis suggests that dietary changes might be achievable with an individual strategy if resources are sufficient. Estimates of the cost-effectiveness of cholesterol lowering by means of screening and dietary advice vary between $4500[58] and $18 000[59] per life-year gained, amounts considerably higher than the estimated $18 for a population strategy based on education and mass media advertising.[59]

An informal review[6] of five trials in primary prevention estimated a mean reduction in cholesterol of about 2% over 6 months to 6 years in high-risk men and concluded that "the ethics of seeking out healthy individuals, measuring cholesterol concentrations, and offering intervention of such limited efficacy needs to be reconsidered." Since then, two trials of screening and multifactorial intervention have been reported. The Family Heart Study[7] (at 1 year) and the Oxcheck study[56] (at 3 years) obtained net reductions, respectively, of 2% and 3% in serum cholesterol and 4% and 2% in diastolic blood pressure. Our systematic overview showed similar net reductions of 0.22 mmol/L (3.7%) in cholesterol and 1.2 mm Hg (1.4%) in diastolic blood pressure. When reduction in blood pressure as well as cholesterol is considered, and regression dilution bias is taken into account,[44,45] we estimate a reduction in coronary heart disease risk of 14%, a decrease considerably greater than the previous estimate of 3%.[6]

Conclusions

With the reservation that we have evaluated the effects of dietary change on

blood pressure and cholesterol rather than on mortality, our estimate of the reduced coronary heart disease incidence achievable through individual dietary intervention would represent some 35% of the United Kingdom's Health of the Nation target. Dietary advice from health care or health promotion personnel appears to be effective in achieving modest dietary change and accompanying cardiovascular risk reduction. Dietary advice in primary care, together with public health and other populationwide policies, may present the most cost-effective strategy for prevention.[60] □

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