Predictive value of a short dietary questionnaire for changes in serum lipids in high-risk Utah families

Paul N Hopkins, Roger R Williams, Hiroshi Kuida, Barry M Stults, Steven C Hunt, Gary K Barlow, and K Owen Ash

ABSTRACT Dietary questionnaires and serum cholesterol, triglycerides, and high-density lipoprotein (HDL) cholesterol determinations were completed for 1239 subjects aged ≥ 20 at each of two separate screenings. The mean time between screenings was 2.5 y. After correcting for potential confounding variables, reduction of a measure of dietary cholesterol and saturated fatty acids assessed by two simple questions was a significant independent predictor of reduction in total cholesterol in serum (p < 0.005). Initial body mass index (BMI) and change in BMI were highly significant predictors of initial values and changes in total cholesterol, triglycerides, and HDL cholesterol in serum. Reduction of dietary saturated fatty acid and cholesterol was significantly correlated with initial serum cholesterol levels, which suggest that serum cholesterol screening may be an important motivating factor for dietary change. Important public health and research implications of these findings are discussed. Am J Clin Nutr 1989;50:292-300.

KEY WORDS Diet, dietary questionnaire, serum cholesterol, serum triglycerides, serum HDL cholesterol, body mass index, cholesterol screening

Introduction

Serum cholesterol is a major, causative risk factor for atherosclerotic coronary heart disease (CHD) (1, 2). Its acceptance as such has stirred a national effort to increase public and professional awareness both of the significance of serum cholesterol levels and the need to reduce levels in high-risk individuals (3). Screening programs are an important component of this program.

Diet is an important contributor to elevated serum cholesterol levels in Western populations (4). Controlled intervention trials in both inpatient and outpatient settings have demonstrated repeatedly the influence of diet on serum cholesterol levels (5, 6). However, demonstrating the expected relationship between diet and serum cholesterol has been more difficult in free-living people. Although a number of studies reported significant correlations between diet as assessed by questionnaire or diet history and serum cholesterol (7-16), reported correlations are usually quite small. Furthermore, several large, well-conducted studies were unable to demonstrate any such correlations (17-20).

In this study we screened more than 2800 individuals of all ages as part of an effort to understand cardiovascular disease in a number of families in Utah.

Methods

The Cardiovascular Genetics Research Clinic has been in operation since 1980. During studies on CHD and familial predisposition to hypertension, 2827 participants of all ages were seen at two different screening visits — 2.5 y apart. Participants were from four population groups: 1) prior hypertensive participants in the Hypertension Detection and Follow-up Program (HDFP) (21%), 2) normotensive HDFP participants (3%) and miscellaneous hypertension family referrals (3%), 3) stroke cluster pedigrees ascertained through the Utah Resource for Genetic and Epidemiological Research (22%), and 4) coronary-prone families from a population-based study of early myocardial infarction deaths in males (51%). Subjects were invited to the clinic by multiple phone and mail contacts. The response rate was 94% at the first 4-h clinic visit with a 91% return rate (second visit). The study was approved by the University of Utah Institutional Review Board and informed consent was obtained from each participant.

At these visits an extensive battery of tests was completed, which included a medical history and physical exam, a variety of investigational blood pressure tests, anthropometrics, an electrocardiogram, routine blood tests, red blood cell ion-flux tests; and a lipid profile including total cholesterol, triglycer-
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ides, and high-density lipoprotein (HDL) cholesterol in serum. Blood samples were always drawn between 0730 and 0800 after a 12-h fast and after subjects had been in a recumbent position for 5 min. There were 1239 subjects aged ≥20 y with completed cholesterol determinations from both visits.

Participants completed a questionnaire at each clinic visit, which included a section on dietary habits. Some of the participants did not complete all of the questions in the questionnaires, thus the different numbers reported in the tables. Details of the dietary section of the questionnaire are given in Appendix A. Questions regarding food choices were semiquantitative. For example, in the questionnaire a list of foods labeled as high in cholesterol or high in saturated fatty acids was followed by two questions, the first asking the last time such a food was eaten and the second how frequently a food item from the list was eaten. Although the results of such questions cannot be used to calculate actual fat or cholesterol intakes they can be useful for roughly rank ordering intakes and more importantly for documenting changes in reported behavior. Some questions on dietary habits were clearly more meaningful than others. Questions regarding fresh fruit and vegetable servings, snacking, and how frequently breakfast or other meals were skipped may have reflected some level of nutrition awareness but would clearly not be suitable for estimating or even ranking nutrient intakes. Not surprisingly, these questions did not contribute strongly to any changes in serum lipid levels.

A physician examined the blood test results for each participant. A short warning, eg, "see your physician", was written on the lab report of some patients with high cholesterol levels. No further instructions or suggestions were given to the participants between the two visits.

All blood samples were analyzed by the University of Utah Medical Center clinical laboratory. Total cholesterol in serum was measured by a standard enzymatic assay on a Technicon SMA-1 auto-analyzer (21). Serum triglycerides were measured enzymatically with a Hitachi chemistry analyzer according to published methods (22). HDL cholesterol was measured in supernatant after precipitation of apolipoprotein B-containing particles by using sodium phosphotungstate according to published methods (23).

Accuracy and precision were carefully monitored throughout this study by using internal and external control samples. The University of Utah Clinical Laboratory has maintained acceptable performance since 1979 in the proficiency testing program of the College of American Pathologists. External control samples are sent quarterly with an accuracy requirement of ±7.5% for total cholesterol and triglycerides in serum and ±10% for serum HDL cholesterol. The laboratory is also standardized for total cholesterol, triglyceride, and HDL cholesterol in serum by using external control samples from the Center of Disease Control and the Northwest Lipid Research Clinic. Precision is monitored daily by using multiple control samples and has been maintained under 5% (CV for cholesterol and triglycerides).

Statistical calculations were performed on a DEC MicroVAX computer with use of BMDP statistical software (24). Tests included paired t test, simple regression, stepwise multiple regression, and factor analysis. Factor analysis was used initially to determine which dietary questions grouped together as single independent factors. A factor determined by this method is simply the weighted sum of several variables and behaves like a new variable. However, interpretation of results from factor analysis is often not straightforward. Therefore, rather than use the factors determined by factor analysis in other calculations, the two or three strongest contributing dietary questions were added together to form a new variable. These groupings were suggested naturally by the questions themselves and the grouped questions were all highly correlated. For example, the average weekly servings of fresh fruits was highly correlated with the average weekly servings of fresh vegetables in factor analysis and together were the only significant contributors to a single factor. Servings of fruit and vegetables were therefore added together to become a single variable. The dietary variables used in the analyses and their component questions are given in Appendix A. Results are reported as means ± SDs; ranges are also given.

Results

The age and sex distribution as well as other characteristics of study participants are presented in Table 1. Only results for subjects aged ≥20 y are reported here. Note the very low prevalence of current cigarette smokers; thus, cigarette smoking was not entered into the regressions.

Potential associations between the reported dietary variables and serum lipid levels were explored by using multiple-stepwise-linear regressions. The dietary factors analyzed include measures of dietary cholesterol and saturated fatty acid avoidance, dietary salt avoidance, weekly servings of fruits and vegetables, the usual number of cups of coffee each day, number of caffeinated soda drinks per week, difficulty limiting food intake, breakfast regularity, snacking, and degree of meal control. These dietary variables are explained in greater detail in Appendix A. Regression analyses also included age, sex, and body mass index (BMI) (wt/ht² in kg/m²).

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**Table 1**

<table>
<thead>
<tr>
<th>Population characteristics at initial visit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>Males (%)</td>
</tr>
<tr>
<td>Cigarette smokers (%)</td>
</tr>
<tr>
<td>CHD prevalence (%)</td>
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<tr>
<td>Hypertension prevalence (%)</td>
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<tr>
<td>Diabetes prevalence (%)</td>
</tr>
<tr>
<td>Those reporting to be on a cholesterol-lowering diet (%)</td>
</tr>
</tbody>
</table>

| Body mass index (kg/m² × 1000)                | 25.7 ± 5.0 |
| Range                                       | 15.7–50.8 |
| Serum cholesterol (mmol/L)                  | 5.22 ± 1.19 |
| Range                                       | 2.04–13.50 |
| Serum triglycerides (mmol/L)                | 1.38 ± 1.43 |
| Range                                       | 0.27–37.46 |
| HDL-cholesterol (mmol/L)                    | 1.25 ± 0.33 |
| Range                                       | 0.52–3.00 |
TABLE 2
Regression coefficients from multiple-stepwise regression for all subjects*

<table>
<thead>
<tr>
<th>Predictor variables</th>
<th>Initial serum lipids (n = 1061)</th>
<th>Change in serum lipids (visit 2 – visit 1) (n = 1042)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total cholesterol</td>
<td>Total triglycerides</td>
</tr>
<tr>
<td></td>
<td>(n = 1116)</td>
<td>(n = 1181)</td>
</tr>
<tr>
<td>Age</td>
<td>1.05 (0.32)‡</td>
<td>1.46 (0.16)§</td>
</tr>
<tr>
<td>Sex</td>
<td>-0.62 (NS)</td>
<td>-2511 (-0.10)†</td>
</tr>
<tr>
<td>BMI</td>
<td>0.30 (0.08)§</td>
<td>2.82 (0.15)§</td>
</tr>
<tr>
<td>Fat avoidance</td>
<td>0.94 (NS)</td>
<td>2.04 (NS)</td>
</tr>
<tr>
<td>Salt avoidance</td>
<td>1.12 (0.06)**</td>
<td>1.74 (NS)</td>
</tr>
<tr>
<td>Fruits and vegetables</td>
<td>-0.16 (NS)</td>
<td>-0.64 (NS)</td>
</tr>
<tr>
<td>Coffee</td>
<td>0.25 (NS)</td>
<td>-1.31 (NS)</td>
</tr>
<tr>
<td>Caffeinated soda</td>
<td>-0.18 (NS)</td>
<td>-0.41 (NS)</td>
</tr>
<tr>
<td>Difficulty limiting food</td>
<td>0.11 (NS)</td>
<td>0.65 (NS)</td>
</tr>
<tr>
<td>Breakfast regularity</td>
<td>-0.13 (NS)</td>
<td>-0.16 (NS)</td>
</tr>
<tr>
<td>Snacking</td>
<td>-0.20 (NS)</td>
<td>-0.46 (NS)</td>
</tr>
<tr>
<td>Meal control</td>
<td>0.13 (NS)</td>
<td>-0.48 (NS)</td>
</tr>
<tr>
<td>Intercept</td>
<td>137.3</td>
<td>-1.8</td>
</tr>
<tr>
<td>Multiple r</td>
<td>0.37</td>
<td>0.28</td>
</tr>
</tbody>
</table>

* Significant standardized-regression coefficients are in parentheses.
† For regressions with changes in lipids, changes in predictor variables were used (visit 2 – visit 1) for all variables except age and sex. For example, change in BMI was used where BMI is indicated. Initial age was used in all analyses.
‡ F > 40.
§ F > 20.
‖ p < 0.001, F > 10.8.
¶ p < 0.01, F > 6.6.
** p < 0.05, F > 3.8.

Results for the entire population of 1239 subjects are reported in Table 2.

Initial cross-sectional studies

Only age and BMI were shown to be strongly correlated with initial serum cholesterol levels. Greater care in avoiding salt was mildly associated with higher cholesterol levels as well. This association may reflect the higher prevalence of hypertensives among those with cholesterol levels above the 90th percentile (21% hypertensives among those above the 90th percentile vs 12% in the total group).

Initial serum triglycerides were highly dependent on age, sex, and BMI. Each decade of life was associated with a 0.165 mmol/L elevation in triglycerides. Females had levels 0.282 mmol/L lower than males. Increases in BMI were associated with higher triglycerides.

Initial serum HDL cholesterol levels correlated most strongly with sex, BMI, and age. Females had levels 0.247 mmol/L higher than did males on the average. In the cross-sectional analysis the effect of BMI on HDL was statistically highly significant but clinically minimal. A 9.1-kg weight gain in a man 1.78 m tall initially weighing 70 kg (increased BMI from 22.1 to 25.0) was associated with a lower HDL concentration of 0.036 mmol/L. Greater avoidance of saturated fatty acids and dietary cholesterol was mildly associated with lower HDL. However, most of the variance explained by the regression was due to sex difference, the first variable entered in the stepwise function (r = 0.38 with sex alone).

Changes between visits 1 and 2

The average change in serum cholesterol between visits 1 and 2 was −0.091 ± 0.804 mmol/L (± SD) (range, −6.00 to +3.57 mmol/L). Correlations of changes in cholesterol levels with changes in reported dietary habits are also shown in Table 2. A change in BMI was the strongest predictor of a change in serum cholesterol. A 9.1-kg weight gain as above would be predicted to result in a 0.25-mmol/L increase in serum cholesterol.

The next strongest predictor of serum cholesterol change was the reported change in avoiding foods rich in cholesterol and saturated fatty acids. A theoretical change of +10 is possible for this variable; the largest change actually seen was 8. Such a change was associated with a decrease in cholesterol by 0.445 mmol/L.

The correlation coefficient for saturated fatty acid and cholesterol avoidance was 0.43 (p < 0.001) between visits 1 and 2. Saturated fatty acid and cholesterol avoidance increased from 3.84 ± 1.80 to 4.21 ± 1.94 (n = 1228) between visits 1 and 2, which was a highly significant change (p < 10⁻⁵, paired t test). Because males were coded as 1 and females 2, regression reveals that females were more successful at lowering cholesterol levels than were males.

Variables associated with change in triglycerides in-
cluded sex, change in BMI, and changes in the weekly number of servings of fruit and vegetables (Table 2). A decrease of BMI by 2.9 would result in a 0.287-mmol/L decrease in serum triglycerides. This influence of weight change was about twice that predicted by the cross-sectional analysis of visit 1. Females experienced an average 0.215-mmol/L decrease in serum triglycerides between visits 1 and 2. The decrease in triglyceride levels associated with eating more fruits and vegetables was of relatively low statistical significance.

Change in HDL cholesterol between visits 1 and 2 was only minimally explained by the regression (multiple $r = 0.17$). Female sex and increasing age were associated with slight decreases in HDL cholesterol. A change in BMI of 2.9 would result in a decrease of HDL cholesterol by only 0.031 mmol/L. This amount is similar to the change predicted in the cross-sectional visit 1 analysis. An increase in fat avoidance by eight units (maximum value observed) was associated with a 0.10-mmol/L decrease in HDL cholesterol.

To investigate the effects of baseline characteristics on reported change of diet, stepwise regression was performed with change in dietary cholesterol and fat avoidance as the dependent variables and age, sex, BMI, and initial serum cholesterol as predicting variables. Initial serum cholesterol level was the only factor entering the equation ($r = 0.10$, regression coefficient = 0.0044, $F$ ratio = 12.7). Higher baseline serum cholesterol did, therefore, appear to significantly influence behavior in this population as shown by increased avoidance of dietary saturated fatty acid and cholesterol.

Discussion

Renewed interest has emerged in serum cholesterol screening in populations since the National Institutes of Health (NIH) Consensus Conference statement "Lowering of Blood Cholesterol to Prevent Coronary Disease" was issued in 1985 (1). That statement boldly provided a redefinition of normal serum cholesterol levels and strongly urged active intervention in persons with cholesterol levels above the age-adjusted 75th percentile. Persons above the 90th percentile were considered to be at high risk for CHD and deserved dietary intervention and drug-lowering medications if necessary. This conference spawned the National Cholesterol Education Program Expert Panel with its subsequent recommendations (3) and a series of requests for applications for research in areas related to cholesterol screening and follow-up.

Strictly speaking, the population screened in visits 1 and 2 in the Cardiovascular Genetics Research Clinic was not a general population. Subjects belonged to pedigrees at high risk for hypertension (49%) or CHD (51%). Nevertheless, because unaffected relatives of probands were mostly screened, few subjects were affected with hypertension (12%) or CHD (3%). Thirteen percent of this study population had age-specific, total cholesterol levels in serum above the 90th percentile of the Lipid Research Clinic general population (1). Thus, higher cholesterol levels were somewhat more prevalent in this group than a general population. Those with higher lipid levels may have theoretically been more responsive to dietary changes thus making the trends observed more apparent. This would not constitute a bias, however, because all subjects filled out the same materials and none received intervention or training as part of their participation. The low prevalence of current smokers was consistent with the predominately Mormon population studied (> 90%). There is no reason to suspect, however, that this study population would respond differently to changes in diet than would other populations. Detailed genetic studies of the Mormon population in UT demonstrated that they are representative of a thoroughly mixed Northern European population with very little inbreeding (25, 26). The low incidence of CHD in Mormon populations compared with average rates in the United States has been attributed to low smoking prevalence, not differences in diet or serum cholesterol levels (27). These considerations suggest that our results apply primarily to white, nonsmoking Northern European populations.

An increasing number of studies have demonstrated a relationship between reported dietary variables and serum lipid levels (7–16). In an important review on statistical reasons for low correlations between diet and serum cholesterol levels, Jacobs et al (12) reported results from dietary intervention on 91 male participants from the Minnesota clinic of the Multiple Risk Factor Intervention Trial (MRFIT). They calculated dietary scores based on the Keys equation (6) from a 24-h dietary recall taken before a 6-wk intervention. At the end of the intervention a 3-d diet history was completed by each participant and his wife. Correlations between serum cholesterol and the dietary score were not significantly different from 0 in cross-sectional analyses both at the beginning and the end of the 6-wk intervention. However, the change in serum cholesterol was highly correlated with the change in dietary score ($r = 0.4$). Similar results were reported among 293 participants in the Chicago branch of the MRFIT (13) study. However, these investigators did not report the correlation coefficient between change in serum cholesterol and change in dietary scores.

Shekelle et al (14) reported a positive correlation between change in diet score and change in serum cholesterol concentration from the initial to a second examination 1 y later in the Western Electric Study. These investigators demonstrated a positive correlation between initial dietary score and initial base-line serum cholesterol levels.

In 1980, Kay et al (11) reported a positive correlation between percent calories from fat and serum cholesterol levels in a group of male staff and faculty from the University of Guelph in Canada. They felt that positive correlations were more easily demonstrated among their 200 participants because of a wide variety of dietary practices among the various ethnic and economic groups.
represented as well as differences in exposure to dietary advice.

During the 10-y follow-up of the Zutphen Study (15), serum cholesterol was significantly and inversely related to total energy intake per kilogram body weight. Changes in serum cholesterol at 5- and 10-y follow-ups were strongly correlated with changes in body weight and weakly correlated with changes in dietary cholesterol intake. Neither initial total fat intake, fat composition, nor reported changes in these variables correlated with initial serum cholesterol levels or changes in serum cholesterol in that study.

Most recently, cross-sectional analyses of correlations between dietary variables and total cholesterol, HDL cholesterol, and triglycerides have been reported for the Tromsø Heart Study (16). Multiple-stepwise regression coefficients were significantly positive for BMI, high coffee consumption, use of butter or hard margarine, nonselection of low-fat milk, and low bread consumption. Men and women who used butter or hard margarine had higher serum cholesterol levels of ~0.26 mmol/L than did subjects who selected soft margarine. Subjects who drank little coffee (<1 cup of coffee/d) compared with those with high coffee consumption (>9 cups of coffee/d) had cholesterol levels 0.59 mmol/L lower. HDL cholesterol was positively associated with use of butter or hard margarine and negatively associated with use of fruits and vegetables in men. However, findings for HDL were of marginal statistical significance. Subjects who did not use table fat and who ate fish frequently for dinner had significantly lower serum triglyceride levels. Most of these correlations were mild to modest with BMI the strongest predictor of serum cholesterol.

Studies that failed to find relationships between dietary variables and serum cholesterol included the Evans County Study (17), the Israeli Heart Study (18), the Framingham Study (19), and the Tecumseh Study (20). Potential confounding factors that tend to obscure the relationship between diet and serum cholesterol have been suggested and include 1) genetic determination and inherent variability of baseline serum cholesterol levels (12), 2) small interpersonal diet variability compared with large intrapersonal diet variability (12, 28), and 3) unreliability of dietary assessment.

Diet assessment has been the subject of several reviews (29, 30). Much effort has been expended to validate dietary questionnaires or history-taking methods. A major shortcoming of most of these studies has been the absence of any gold standard or quantifiable, accurate, and reliable assessment of dietary intake. As noted by one reviewer (30) there is usually no way to determine the truth. Cumbersome 3- or 7-d diet diaries have been the standard by which more simple methods are judged. However, even 7-d diaries lacked accuracy after the first 3 d in a study of elderly subjects living in a home where actual intakes could be directly measured (31). Furthermore, it is not known whether or to what extent the act of recording food intake alters usual dietary practices.

Despite these difficulties a number of short dietary questionnaires have compared favorably with more extended methods (29, 32, 33). Semiquantitative questionnaires aimed at determining usual intakes (asking, for example, the usual number of cups of coffee per week) have been most promising (29, 33). The very short questionnaire used in the present study is of this type. Although our questionnaire could not be validated in the sense of comparison with more cumbersome methods, results suggest at least internal reproducibility and usefulness. Correlation coefficients > 0.50 for dietary variables in repeated assessments is one standard to judge adequate reliability (30, 33). In our population the correlation coefficient between visits 1 and 2 reported dietary fat and cholesterol avoidance to be slightly lower than this (r = 0.43). However, real changes in diet and long intervals between reevaluation may account for lower correlations even in well validated questionnaires or methods (30). Finally, the usefulness of our questions regarding cholesterol and saturated fatty acid-containing foods is suggested by the strong correlation observed between reported changes of dietary cholesterol fat avoidance and measured changes in serum cholesterol.

A fourth potentially major factor obscuring the relationship between reported diet and serum cholesterol was examined more recently. Investigators of the Chicago Western Electric Company Study (34) pointed out that at their first screening, the diet score, based on the formula by Keys et al (34), was positively associated with serum cholesterol levels. However, by the second examination the correlation between diet score and serum cholesterol had become significantly negative. This negative correlation was due entirely to a change in diet among subjects with cholesterol levels > 6.46 mmol/L. Interestingly, these investigators had noted that during their pilot nutritional intervention trial for the MRFIT study, subjects who had been invited to participate because of high cholesterol levels reported surprisingly low levels of dietary fat and cholesterol intakes. The investigators hypothesized that these low reported intakes were due to interventions already made by the participants on their own behalf (13). The fact that dietary histories were not obtained at the initial examination in the Tecumseh (20) and Framingham (19) studies is particularly significant.

For the Framingham Study, food frequency histories were obtained by trained dietitians at the fifth biennial examination in 1957–1960. In the Tecumseh Study, 24-h dietary recalls were not obtained until the third round of examinations in 1967–69. Hence, the same bias operating in the Western Electric study may have contributed to the lack of correlation between dietary variables and cholesterol in the Framingham and Tecumseh studies.

Further evidence for a confounding effect from prior screening may be found in our study. Although baseline serum cholesterol levels did not significantly correlate with dietary variables, the reported change in serum cholesterol was significantly and negatively correlated with changes in avoidance of foods rich in saturated fatty acids and cholesterol. The cross-sectional analysis of data from visit 2 reveals opposite findings. In multiple-step-
wise-linear regression, age, sex, and BMI were still primary determinants of cholesterol at visit 2 (standardized regression coefficients 0.291, -0.06, and 0.099, respectively with corresponding significance levels ($F = 108.5, 5.6, 12, 7$). However, dietary saturated fatty acid and cholesterol avoidance was now strongly positively correlated with serum cholesterol levels (standardized regression coefficient 0.11, $F = 15.4$). Furthermore, reported avoidance of foods rich in saturated fatty acids and cholesterol at visit 2 was correlated positively with serum cholesterol determined at visit 1. The fact that reported change in dietary cholesterol and fat avoidance also correlated positively with initial serum cholesterol levels affirms the importance of prior serum cholesterol screening on reported dietary behavior and subsequent serum cholesterol levels.

These findings have important public health implications. In MRFIT, the regular-care-group managed to lower their serum cholesterol to an extent similar to that of the special-care-group, thereby eliminating the power to demonstrate any beneficial effect of dietary intervention on the special-care-group (35). This may well have been due to the effect of screening serum cholesterol levels in the participants of that study. Although a screening effect may confound results in an intervention trial such as MRFIT, the benefit to individuals may be substantial. Screened participants do respond by changing their diets when their cholesterol levels appear to be high. Screening programs may be justified simply because they supply cholesterol levels to participants. Sufficient information should be supplied to allow participants to compare their levels with others. Cholesterol screening may thus provide a means whereby a realistic appraisal of risk is supplied to participants. Several studies demonstrated that unrealistic optimism in regard to risk status is prevalent among large segments of the population (36-39). This unrealistic optimism was corrected when an objective assessment of risk in comparison with others was supplied to the study subjects (39).

Results from regressions of HDL and triglyceride levels are consistent with findings of other investigators. Significant negative correlations between initial BMI and HDL cholesterol were found in this study and by others (16, 40-44). Few studies report long-term relationships between weight change and change in HDL cholesterol in free-living subjects. There are several reports, some with conflicting results, that deal with HDL changes after weight loss (42-48). A mild but statistically significant negative correlation between the reported change in avoidance of foods rich in saturated fatty acids and cholesterol and the change in HDL cholesterol was seen in this population. This finding may be consistent with studies reporting mild to moderate decreases in serum HDL, along with greater decreases in LDL, when diets designed to lower total cholesterol have been instituted (49-52). Also, subjects who increase their saturated fatty acid and cholesterol intake have been reported to experience increases in serum HDL levels along with increases in low-density lipoprotein (LDL) cholesterol levels (53-57). These changes in HDL may not necessarily be beneficial (58-60). Monounsaturated fatty acids when substituted for carbohydrates either do not affect or tend to increase serum HDL levels (61-65). Nevertheless, results from the present study suggest that a reduction in BMI would be a more effective means of increasing the HDL level than would the alteration of dietary fat intakes. Triglyceride levels would also be favorably influenced by decreases in BMI.

In conclusion, we demonstrated significant correlations between changes in serum cholesterol levels and changes in dietary saturated fatty acid and cholesterol intake assessed by two short questions from a dietary questionnaire in a free-living population. Initial BMI and changes in BMI were important predictors of initial levels and changes in serum lipids. Dietary changes were strongly influenced by initial cholesterol levels suggesting that serum cholesterol screening may provide important motivation to improve dietary habits. These results have important public health implications.

References


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APPENDIX A

The following are questions from the questionnaire used to characterize subjects’ usual diet. Several questions were grouped as single variables as noted below. All the groupings were suggested by factor analysis (see Methods).

A. Dietary Cholesterol and Saturated Fat Avoidance. Questionnaires included the list below (labeled as foods high in cholesterol or saturated fat as shown) and subjects responded to the two questions following the list. Responses (1 to 6) to the two questions were added together. Observed values at Visit 1 among 1,207 subjects, age 20 and over with completed questionnaires ranged from 2 to 12 with mean 3.84, S.D. = 1.81.

List of Foods High in Cholesterol or Saturated Fat:

- Bacon or other Pork
- Hot Dogs
- Sausage
- Marbled and Fatty Meats (beef, pork, lamb)
- Spare Ribs
- Fish fried in shortening
- Liver or other organ meats
- Shrimp
- Hamburger
- Luncheon Meats
- Egg yolks
- Cakes and Pies
- Sweet Rolls
- Butter
- Shortening
- Coconut Oil
- Potato Chips
- French Fries
- Other Fried Foods
- Lard
- Whole Milk
- Cream and Ice Cream
- Cheese
- Butter Rolls
- Donuts
- Egg Noodles

1. When was the last time you ate eggs, whole milk, pork or other high cholesterol foods from the above list?
   1) Within the last 3 meals
   2) A day ago
   3) 2–5 days ago
   4) A week ago
   5) Over a week ago
   6) Over a year ago

2. How often do you eat any of the foods listed above, such as egg yolk, pork, whole milk, butter, cream or other of these foods?
   1) 2 or more times a day
   2) Once a day
   3) 2–5 times a week
   4) Once a week
   5) Less than once a week
   6) Almost never

B. Dietary Salt Avoidance. Responses (1 to 6) to the two questions referring to a table of salty foods were added together. Visit 1 values range from 2 to 12 with mean = 4.33, S.D. = 2.32.

List of Salty Foods:

- Bacon or Ham
- Hot Dogs
- Sausage
- Bologna and Luncheon Meats
- Chipped or Corned Beef
- Smoked or Salted Meats
- Herring, Sardines
- Potato Chips
- Pretzels
- French Fries
- Salted Snacks (popcorn, nuts, etc.)
- Salted Crackers
- Seasoning Salts (celery, garlic, onion)
- Pickles
- Sauerkrout
- Bouillon
- Catsup
- Canned Soups
- Dried Soups
- Chili Sauce
- Mustard
- Olives
- Relishes
- Meat Tenderizers
- Sauces (soy, steak, etc.)

1. How often do you salt your food from a shaker at the dinner table or eat salty foods such as potato chips, bacon, or other foods listed above?
1) 2 or more times a day
2) Once a day
3) 2–5 times a week
4) Once a week
5) Less than once a week
6) Almost never (I'm on a special low salt diet)

2. When was the last time you used a salt shaker on your food or ate one of the salt foods listed above?
   1) Within the last three meals
   2) A day ago
   3) 2–5 days ago
   4) A week ago
   5) Over a week ago
   6) Over a year ago

C. Weekly Servings of Fruits and Vegetables was the sum of the following two questions. Responses at Visit 1 ranged from 0 to 46, mean = 10.0, S.D. = 6.84.
   1. In an average week, how many servings of fresh fruit are you likely to eat?
   2. In an average week, how many servings of fresh vegetables, including salads, do you eat?

D. Cups of Coffee Per Day. Range for this variable at Visit 1 was from 0 to 45, mean = 0.60, S.D. = 2.31.
   How many cups of non-decaffeinated coffee do you drink in an average day?

E. Caffeinated Soda Drinks Per Week. This variable did not group with cups of coffee in factor analysis. Values reported at Visit 1 ranged from 0 to 35, mean = 4.96, S.D. = 6.16.
   How many drinks per week do you have of Coca Cola, Pepsi, Dr Pepper, Mountain Dew, Tab or diet cola?

F. Difficulty Limiting Food Intake. The following three questions grouped together as a unique factor in factor analysis. Responses were added together for this variable. Visit 1 range was 0 to 21, mean = 4.51, S.D. = 5.14.
   1. During the last week, how many days did you experience difficulty in limiting candy eating? 1 2 3 4 5 6 7 0
   2. During the last week how many days did you experience difficulty in limiting your eating of breads or pasta? 1 2 3 4 5 6 0
   3. During the past week, how many days did you experience difficulty in limiting your eating of fatty foods? 1 2 3 4 5 6 0

G. Breakfast Regularity was estimated by subtracting the second of the following two questions from the first. Values from Visit 1 responses ranged from −31 to 7, mean = 7.21, S.D. = 12.65.
   1. In an average week, how many mornings do you eat a breakfast that includes some form of protein such as cereal and milk, eggs, cheese, fish or meat?
   2. In an average month, how many times do you skip breakfast?

H. Snacking was the sum of the following two questions. Range was 0 to 60, mean = 5.09, S.D. = 4.29.
   1. On weekdays, how many snacks do you usually have that are not part of a planned meal?
   2. On weekends, how many snacks do you usually have that are not part of a planned meal?

I. Degree of Meal Control was the sum of the following three questions which grouped as a single factor in factor analysis. Values ranged from 0 to 21, mean = 7.13, S.D. = 4.95.
   1. During the past week, how many days did you plan what you would eat at the start of the day? 1 2 3 4 5 6 7 0
   2. During the past week, how many days did you eat your meals at set times? 1 2 3 4 5 6 7 0
   3. During the past week, how many days did you decide not to eat a snack that you wanted, even though the food was available? 1 2 3 4 5 6 7 0