A Novel Family-Based Intervention Trial to Improve Heart Health: FIT Heart
Results of a Randomized Controlled Trial

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Background—Family members of patients with cardiovascular disease (CVD) may be at increased risk due to shared genes and lifestyle. Hospitalization of a family member with CVD may represent a “motivational moment” to take preventive action.

Methods and Results—A randomized, controlled clinical trial was conducted in healthy adult family members (N=501; 66% female; 36% nonwhite; mean age, 48 years) of patients hospitalized with CVD to evaluate a special intervention (SI) with personalized risk factor screening, therapeutic lifestyle-change counseling, and progress reports to physicians versus a control intervention (CIN) on the primary outcome, mean percent change in low-density lipoprotein cholesterol (LDL-C), and other risk factors. Validated dietary assessments and standardized risk factors were obtained at baseline and 1 year (94% follow-up). At baseline, for 93% of subjects, saturated fat comprised \( \frac{7}{10} \) of total caloric intake, and 79% had nonoptimal LDL-C levels (of which 50% were unaware). There was no difference in the SI versus the CIN with respect to the mean percent change in LDL-C (1% versus 2%, respectively; \( P=0.64 \)), owing to a similar significant reduction in LDL-C in both groups (\( \frac{4.4}{dL} \) and \( \frac{4.5}{dL} \), respectively). Diet score significantly improved in the SI versus the CIN (\( P=0.04 \)). High-density lipoprotein cholesterol declined significantly in the CIN but not in the SI (3.2% [95% CI, -5.1 to -1.3] versus +0.3% [95% CI, -1.7 to +2.4]; \( P=0.01 \)). At 1 year, SI subjects were more likely than controls to exercise \( >3 \) days per week (\( P=0.04 \)).

Conclusion—The SI was not more effective than the CIN in reducing the primary end point, LDL-C. The screening process identified many family members of hospitalized patients with CVD who were unaware of their risk factors, and further work is needed to develop and test interventions to reduce their CVD risk. (Circ Cardiovasc Qual Outcomes. 2008; 1:98-106.)

Key Words: prevention ■ risk factors ■ trials
improve adherence to prevention guidelines, although proof of concept studies have shown that systematic evidence-based approaches have been successful in the setting of secondary prevention among hospitalized patients.15–17

The purpose of this study was to test a systems approach to primary prevention that identified family members of hospitalized cardiac patients; offered systematic screening, with results, education about national primary prevention goals, and lifestyle counseling; and supplied regular progress reports to primary physicians compared with the impact of a brief general prevention message on adherence to national prevention goals at 1 year. The primary outcome was the mean percent change in low-density lipoprotein cholesterol (LDL-C) between groups; prespecified secondary outcomes included changes in diet and lifestyle and other major CVD risk factors. A unique aspect of the program was the use of nonphysician, nonnurse, and English- and Spanish-speaking master’s level health educators who delivered the intervention at the time of hospitalization of a family member with CVD and had regular contact with intervention subjects for up to 1 year. The controlled design provided an opportunity to evaluate the added value of education related to personal risk factors and lifestyle counseling beyond the context of a potentially motivational period on changes in CVD risk factors at 1 year.

Methods

Design and Participants

The Family Intervention Trial for Heart Health (FIT Heart) was a 1-year randomized, controlled clinical trial that enrolled family members of cardiac patients admitted to New York Presbyterian Hospital/Columbia University Medical Center between January 2005 and June 2007 and conducted 1-year follow-up visits between January 2006 and June 2008. Participants were randomly assigned to either (1) a special intervention (SI) whereby they received personalized CVD risk factor assessment, National Cholesterol Education Program Adult Treatment Panel III therapeutic lifestyle change (TLC) counseling,18 regular contact with a health educator, and progress reports sent to their primary physicians or (2) a control intervention (CIN) in which they received a brief prevention message and a letter to their healthcare provider only if they had a critical threshold for a CVD risk factor level. All participants received a baseline and 1-year assessments of diet, lifestyle, and risk factors to compare the mean percent change in LDL-C in the SI versus the CIN (primary outcome) and changes in diet, lifestyle, and other CVD risk factors (secondary outcomes).

The study sample comprised 501 adults (66% female; 36% racial/ethnic minorities; mean age, 48 ± 13.5 years) who were recruited at the time a family member (index case) was hospitalized with acute atherosclerotic CVD, including catheter-based procedures and coronary artery bypass grafting. English- and Spanish-speaking men and women between the ages of 20 to 79 years who currently lived within 3 hours of the medical center were eligible. A family member was defined as a blood relative, spouse, or other individual that currently lived with the index case or had been a cohabitant for a minimum of 1 year within the previous 5 years or for 10 years in the past. To avoid nonindependence of observations, only 1 family member per family was enrolled in the study and randomized; however, additional family members were welcome to attend intervention visits with the randomized participant. Exclusion criteria included current or planned pregnancy, established CVD, diabetes, active liver disease, chronic kidney disease, life expectancy <5 years, prescription of a special diet incompatible with the TLC diet, or participation in a clinical drug study within 3 months of randomization.

Once a subject met all inclusion/exclusion criteria, he or she was randomly assigned to either the SI or CIN. Research staff used a web-based program that generated a group assignment at the time of enrollment. Randomization was blocked based on sex and race/ethnicity using the PROC PLAN function in SAS (version 9.1, SAS Institute, Cary, NC).

All participants were required to give informed consent and were compensated $100 at the 1-year final visit to offset the costs of trial participation. The consent document informed subjects they would be participating in a study to test a personalized screening and education program. The study was approved by the Institutional Review Board of Columbia University Medical Center.

Intervention

Participants randomized to the SI received personalized CVD risk factor screening with immediate feedback (including lipid results) provided by a master’s level health educator with a minimum of 6 months of clinical experience and specific training in national primary prevention guidelines and TLC diet instruction by a registered dietician and a physician specializing in the prevention of CVD. The Transtheoretical Stages of Change model was used to determine each participant’s specific level of readiness to make lifestyle and/or dietary changes.19 Behavioral counseling was based on the 5 A’s construct (assess, advise, agree, assist, arrange) recommended by the US Preventive Services Task Force Counseling and Behavioral Interventions Work Group.20 SI subjects were taught lifestyle approaches to risk reduction based on national CVD prevention guidelines, including the Adult Treatment Panel III recommendations for improving blood cholesterol levels with a TLC diet.19 Education focused on avoiding foods that contain saturated fat, cholesterol, partially hydrogenated fats, trans fats, refined sugars, as well as recommendations to eat at ≥2 servings of fruits, ≥3 servings of vegetables, and ≥20 g of fiber per day. The counseling focused on foods rather than nutrient intake. Participants were encouraged to engage in moderate physical activity, such as brisk walking, for at least 30 minutes daily (60 minutes if weight loss was desired). Smokers were given educational handouts, encouraged to discontinue smoking, and referred to a hospital-based smoking-cessation program.

Regular contact between the SI participant and the health educator was in person or over the telephone at 2 weeks, 6 weeks, 3 months, 6 months, and 9 months. At each follow-up, lifestyle changes were reinforced and potential barriers to attaining risk factor goals were discussed. At the 6-week and 6-month follow-up, a validated dietary assessment of adherence to TLC diet was administered, and results were used to counsel SI subjects.21 In addition, at 3 months, 6 months, and 9 months, participants with previously abnormal lipid panels were offered measurement and immediate feedback of lipid levels using the same fingerstick technology used at the baseline visit, which has been validated to provide the same level of accuracy and precision as a standardized laboratory, meeting the total error guidelines set by the National Cholesterol Education Program.22 Visits were 30 to 60 minutes long. Risk factor results for SI subjects were given to their primary care providers in the form of progress reports sent via facsimile to physician offices. Those randomized to the CIN and SI received a 1-page handout to (1) avoid tobacco, (2) choose good nutrition, and (3) be more active. A report was sent to their healthcare providers if a critical threshold value for a CVD risk factor was determined (ie, blood pressure ≥140/90 mm Hg, LDL-C ≥190 mg/dL, high-density lipoprotein cholesterol [HDL-C] ≤25 mg/dL, triglycerides ≥500 mg/dL, total cholesterol >300 mg/dL).

Risk Factor Measurements

Standardized CVD risk factor information was obtained for all subjects at baseline and 1 year, including demographics, medical and family history, medication usage, and lifestyle habits. Trained research assistants blind to group assignment collected the 1-year outcome data. Bilingual staff members were available to assist participants, and all forms were available in English and Spanish.
Systolic and diastolic blood pressure was measured in the Columbia University Clinical and Translational Science Award Center using a standard protocol, and standard definitions of hypertension were used.23,24 Height, weight, and waist circumference were measured using National Cholesterol Education Program Adult Treatment Panel III protocols, and body mass index was calculated.25 Physical activity level and smoking status were assessed using standardized questions adapted from the validated Behavioral Risk Factor Surveillance System questionnaire.26 Carbon monoxide monitoring was used to validate self-reported smoking behavior in 100% of participants.

Laboratory Procedures

Venous fasting blood samples were collected at baseline and 1 year on all subjects, stored at −70°C up to 2 weeks, and analyzed in the Columbia University Clinical and Translational Science Award Biomarker Laboratory (certified by the Centers for Disease Control and Prevention lipid quality control program). Plasma total cholesterol, HDL-C, triglyceride, and glucose values were determined spectrophotometrically on a Hitachi 912 chemical analyzer. Plasma LDL-C values were assessed using a direct homogeneous enzymatic colorimetric assay; additionally, LDL-C levels were determined on all participants with triglycerides <400 mg/dL. For educational purposes, SI subjects had fingerstick lipid measurements, and high-sensitivity C-reactive protein was measured using LDX desktop analyzers and supplies provided by Cholestech Corporation (Hayward, Calif).22

Diet Assessment

Adherence to the TLC diet was assessed using the MEDFICTS (Meats, Eggs, Dairy, Fried foods, fat In baked goods, Convenience foods, fats added at the Table, and Snacks) Questionnaire that quickly identifies nonadherence and has been validated in this population.21,27 Scores range from 0 to 216 points. A score of <40 is consistent with adherence to the TLC diet (≤7% of calories from saturated fat and <200 mg dietary cholesterol/day). Dietary intake of saturated fat, monounsaturated fat, polyunsaturated fat, trans fat, cholesterol, protein, carbohydrates, and fiber was assessed using the 1998 Gladys Block Food Frequency Questionnaire, a 110-item, validated food-frequency questionnaire developed based on National Health and Nutrition Examination Survey III data.28,29

Statistical Analysis

Descriptive data are presented as means or proportions. Between-group differences were analyzed with a t test of independent samples for continuous variables and a χ² test for categorical variables. For the primary outcome analysis, a t test was conducted to evaluate the difference between the SI and CIN groups in mean percent change from baseline to 1 year that included subjects with core laboratory baseline and 1-year LDL-C values. An intention to treat analyses was performed using the last observation (baseline) carried forward and also by imputing the mean within-group result for missing values (SI, 15; CIN, 17). All results were similar, and the true core laboratory measured results are presented. Analyses were also repeated for the primary outcome that restricted analyses to laboratory data collected centrally and eliminated the 1-year data points from outside laboratories (10%).

Paired t tests were used to assess within-group change in mean values from baseline to 1 year. Adherence to US prevention guidelines was defined as percent at goal according to Adult Treatment Panel III guidelines,18 and differences in proportions adherent to US prevention guidelines at baseline and at 1 year were assessed using χ² statistics. Linear regression was used to evaluate dose response between adherence to follow-up visits and change in lipids and diet. This study was designed to have >80% power to detect a 4% difference in mean recent change in LDL-C in the SI versus CIN groups. A 2-sided α error of <0.05 indicated statistical significance. Analyses were conducted using SAS software.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Figure 1 illustrates the number of participants screened for eligibility, excluded, randomized, lost to follow-up, and included in the analysis. Approximately 1 in 5 family members who were approached and eligible for the 1-year randomized controlled trial agreed to participate. There was a 94% rate of follow-up (232 participants with primary outcome measures in each group) at 1 year. In the SI arm, adherence with follow-up contact points ranged from 90% at 2 weeks to a low of 72% at 9 months. Characteristics of the participants are shown in Table 1. At baseline, there were no significant differences in demographics, CVD risk factors, or diet variables between the SI and CIN groups, suggesting that randomization was effective.

Primary Outcome and Other Risk Factors

There was no difference in the primary outcome (mean percent change in LDL-C) between SI and CIN groups from baseline to 1 year (−1% versus −2%, respectively; P=0.64; Table 2). LDL-C improved significantly within the SI (−4.4 mg/dL; 95% CI, −0.44 to −8.44; P=0.03) and the CIN (−4.5 mg/dL; 95% CI, −1.4 to −7.6; P=0.005) from baseline to 1 year. As shown in Table 3, there were no significant differences between groups in the proportion of participants meeting LDL-C goals of <130 mg/dL (<3.36 mmol/L) or <100 mg/dL (<2.59 mmol/L) at 1 year. Mean percent change in HDL-C declined significantly in the CIN but not in the SI (−3.2% [95% CI, −5.1 to −1.3] versus +0.3 [95% CI, −1.7 to +2.4]), and the between-group difference was significant (P=0.01).

Both groups showed a significant mean percent increase in blood pressure and waist size, and the between-group differences in mean percent change were not statistically significant (Table 2). At 1 year, there was similar adherence to US guidelines for body mass index (18.5 to 24.9 kg/m²) for SI versus CIN (38% versus 33%, respectively; P=0.14) and for waist circumference (59% versus 54%, respectively; P=0.14).18,25,30 as shown in Table 3. There was no significant difference in the proportion of SI versus CIN participants with a Framingham risk score <10% at 1 year (90% versus 92%, respectively; P=0.51).18

Lifestyle Factors

As shown in Table 4, the SI was associated with a significantly greater improvement in the mean percent change in MEDFICTS score compared with the CIN (18.4% [95% CI, −9.9 to −26.8] versus 5.0% [95% CI, +4.2 to −14.3]; P=0.04). At baseline, 93% of subjects overall had a saturated intake ≥7%. Both the SI and the CIN groups showed significant improvements in percent calories from saturated fat (−0.70 [95% CI, −0.4 to −1.0]; P<0.001 and −0.39 [95% CI, −0.06 to −0.72]; P=0.02, respectively), dietary cholesterol (mg) (−48.8 [95% CI, −32.1 to −65.5]; P<0.001 and −30.1 [95% CI, −14.6 to −45.5]; P=0.0002, respectively), and percent calories from trans fat (−0.024
[95% CI, −0.077 to −0.402]; *P*=0.004 versus −0.122 [95% CI, −0.248 to +0.0045]), with no statistically significant between-group differences in mean percent change in these dietary components.

In the SI participants, a dose response was observed between decreased saturated fat intake and the number of follow-up visits attended in which lipids were measured. Each additional follow-up visit with a fingerstick lipid measurement (range, 0 to 3) and immediate feedback of results to participants was associated with an average decrease in daily calories from saturated fat of 3.9% (*P*=0.01).

The SI and CIN groups each significantly increased the number of days of physical activity per week (+0.59 [95% CI, 0.29 to 0.88]; *P*=0.0001 and +0.35 [95% CI, 0.09 to 0.60]; *P*=0.009), and the mean percent change between groups was not significantly different. At 1 year, SI participants were more likely than CIN participants to engage in physical activity ≥3 days per week (33% versus 24%; *P*=0.02). The number of smokers in the SI decreased from 26 at baseline to 16 at 1 year and from 25 to 22 in the CIN; neither change was statistically significant.

**Medication Changes**

There was no significant increase in the use of cholesterol-lowering medications in either group. A total of 15 participants who were not on statins at baseline were on them at 1 year, and 7 subjects on statins at baseline were not on them at 1 year, with no significant between-group changes (15% of SI and 17% of CIN on statins at 1 year; *P*=0.56). If the 15 subjects initiated on statins were excluded from the primary analysis, the results were not statistically different from those obtained using the entire sample. Likewise, increases in antihypertensive use were small and nonsignificant both overall and between groups at 1 year (21% of SI and 24% of CIN subjects on antihypertensive medications at 1 year; *P*=0.55).

**Risk Factor Awareness**

At baseline, 79% of all participants had nonoptimal LDL-C levels (≥100 mg/dL), and 45% had LDL-C ≥130 mg/dL. Among participants with nonoptimal LDL-C (n=398), 50% reported that they were not aware of a history of diagnosis of hypercholesterolemia, abnormal lipid levels, or use of cholesterol-lowering medications. Of participants with LDL-C ≥130 mg/dL (n=226), 39% reported a similar lack of awareness of previous abnormality, diagnosis, or treatment. Additionally, of those with low HDL-C (<50 mg/dL in women; <40 mg/dL in men) or high triglycerides (≥150 mg/dL), 44% and 38%, respectively, were not aware they had dyslipidemia.
Table 1. Baseline Characteristics by Group Assignment (N=501)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Special Intervention</th>
<th>Control Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=250, n (%)</td>
<td>n=251, n (%)</td>
</tr>
<tr>
<td>Age (≥65 years)</td>
<td>30 (12)</td>
<td>30 (12)</td>
</tr>
<tr>
<td>Female</td>
<td>165 (66)</td>
<td>167 (67)</td>
</tr>
<tr>
<td>White race</td>
<td>161 (64)</td>
<td>162 (65)</td>
</tr>
<tr>
<td>Married/living with partner</td>
<td>161 (65)</td>
<td>167 (67)</td>
</tr>
<tr>
<td>Education high school or less</td>
<td>58 (23)</td>
<td>51 (21)</td>
</tr>
<tr>
<td>Unemployed</td>
<td>74 (30)</td>
<td>58 (23)</td>
</tr>
<tr>
<td>No health insurance</td>
<td>39 (16)</td>
<td>33 (13)</td>
</tr>
<tr>
<td>Family history of premature coronary heart disease</td>
<td>137 (55)</td>
<td>123 (49)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>26 (10)</td>
<td>25 (10)</td>
</tr>
<tr>
<td>Body mass index ≥30 kg/m²</td>
<td>78 (32)</td>
<td>90 (36)</td>
</tr>
<tr>
<td>Waist circumference ≥40 inches</td>
<td>87 (35)</td>
<td>102 (41)</td>
</tr>
<tr>
<td>Abnormal lipids*</td>
<td>171 (69)</td>
<td>176 (70)</td>
</tr>
<tr>
<td>Statin therapy</td>
<td>31 (13)</td>
<td>40 (16)</td>
</tr>
<tr>
<td>Hypertension†</td>
<td>85 (34)</td>
<td>86 (34)</td>
</tr>
<tr>
<td>Antihypertensive therapy</td>
<td>52 (21)</td>
<td>53 (21)</td>
</tr>
<tr>
<td>Framingham absolute risk ≥10%</td>
<td>21 (8.5)</td>
<td>23 (9.2)</td>
</tr>
</tbody>
</table>

*Abnormal lipids was defined as (1) LDL-C ≥130 mg/dL, (2) HDL-C <40 mg/dL (male) or <50 mg/dL (female), (3) triglycerides ≥150 mg/dL, or (4) on medication for lipid management.
†Hypertension was defined as blood pressure ≥140/90 mm Hg or on antihypertension medication.

Above-optimal blood pressure levels (≥120/80 mm Hg) were measured in 69% of all participants at baseline. Among those with nonoptimal blood pressure (n=345), 59% reported no prior awareness of abnormal levels, diagnosis, or treatment. Similarly, among those found to be hypertensive (≥140/90) at screening (n=112), 39% reported no history of diagnosed hypertension, abnormal blood pressure readings, or antihypertensive treatment previously. Among participants with nonoptimal LDL-C or blood pressure levels (92%), 4 of 5 participants (80%) were unaware of one or both of these conditions.

### Discussion

The trial failed to achieve the primary outcome of a difference in mean percent change in LDL-C between the groups. The lack of effectiveness of the SI to reduce LDL-C beyond the reduction observed in the CIN supports the possibility that self-directed changes in lifestyle during a “motivational moment” are not insignificant, and that a more powerful intervention than what was used in this study may be necessary to further reduce LDL-C. The trial also showed a significant reduction in HDL-C in the controls but not in the SI group. The mean percent change was small and may have been due to chance. This finding deserves further investigation and may be related to the significantly greater improvement in overall diet score and adherence to physical activity goals as well as the combined trends in less saturated fat intake, increased monounsaturated fat intake, smaller increases in abdominal adiposity, and less smoking at 1 year in the SI compared with the CIN. Despite the improvements in lifestyle, some CVD risk factors worsened in both groups over time, including blood pressure and adiposity, suggesting a more comprehensive and intensive intervention may be prudent. It is noteworthy that improvements in lifestyle may have beneficial effects for CVD and chronic disease prevention beyond the risk factors measured in this study. Overall, there was substantial nonadherence to national prevention goals and poor adherence to lifestyle at baseline and follow-up among family members at risk.

We observed a smaller improvement in LDL-C than in other studies, which may be related to lower mean baseline

Table 2. Change in Measured CVD Risk Factors by Group Assignment

<table>
<thead>
<tr>
<th></th>
<th>SI, Mean (95% CI)</th>
<th>CIN, Mean (95% CI)</th>
<th>Between-Group Difference in Mean Percent Change, P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline 1 Year</td>
<td>Percent Change</td>
<td>Baseline 1 Year</td>
</tr>
<tr>
<td>LDL-C, mg/dL</td>
<td>128.5 (124.1 to 132.9)</td>
<td>123.7 (118.9 to 128.4)</td>
<td>−1.0 (−4.3 to 2.3)</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>202.7 (197.9 to 207.4)</td>
<td>202.2 (196.8 to 207.6)</td>
<td>0.8 (−1.4 to 3.0)</td>
</tr>
<tr>
<td>HDL-C, mg/dL</td>
<td>58.5 (56.2 to 60.7)</td>
<td>58.7 (56.1 to 61.3)</td>
<td>0.3 (−1.7 to 2.4)</td>
</tr>
<tr>
<td>TC/HDL-C ratio</td>
<td>3.6 (3.6 to 3.9)</td>
<td>3.7 (3.6 to 3.9)</td>
<td>2.0 (−0.6 to 4.5)</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>115.0 (106.7 to 123.3)</td>
<td>113.5 (102.6 to 124.4)</td>
<td>4.0 (−1.5 to 9.4)</td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>97.9 (96.0 to 99.8)</td>
<td>98.1 (96.1 to 100.1)</td>
<td>1.2 (−1.0 to 3.4)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>126.7 (124.8 to 128.6)</td>
<td>129.7 (127.3 to 132.0)</td>
<td>3.1 (−1.6 to 4.7)</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>77.9 (76.5 to 79.3)</td>
<td>79.0 (77.5 to 80.6)</td>
<td>2.8 (1.2 to 4.5)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>27.6 (27.1 to 28.5)</td>
<td>27.7 (26.9 to 28.5)</td>
<td>0.5 (−0.2 to 1.2)</td>
</tr>
<tr>
<td>Waist circumference, inches</td>
<td>35.6 (34.9 to 36.3)</td>
<td>36.2 (35.5 to 37.0)</td>
<td>2.4 (1.6 to 3.3)</td>
</tr>
</tbody>
</table>

TC indicates total cholesterol.
Table 3. Adherence to CVD Primary Prevention Goals at Baseline and 1-Year

<table>
<thead>
<tr>
<th>Prevention Goals</th>
<th>Baseline</th>
<th>1-Year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Special Intervention, %</td>
<td>Control Intervention, %</td>
</tr>
<tr>
<td>Total cholesterol &lt;200 mg/dL</td>
<td>51</td>
<td>47</td>
</tr>
<tr>
<td>LDL-C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;130 mg/dL</td>
<td>56</td>
<td>53</td>
</tr>
<tr>
<td>&lt;100 mg/dL</td>
<td>22</td>
<td>20</td>
</tr>
<tr>
<td>HDL-C ≥40 mg/dL</td>
<td>89</td>
<td>87</td>
</tr>
<tr>
<td>Triglycerides &lt;150 mg/dL</td>
<td>79</td>
<td>78</td>
</tr>
<tr>
<td>Glucose &lt;100 mg/dL</td>
<td>62</td>
<td>58</td>
</tr>
<tr>
<td>Blood pressure &lt;140/90 mm Hg</td>
<td>77</td>
<td>78</td>
</tr>
<tr>
<td>&lt;120/80 mm Hg</td>
<td>30</td>
<td>32</td>
</tr>
<tr>
<td>Waist circumference</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤35 inches (female)</td>
<td>61</td>
<td>56</td>
</tr>
<tr>
<td>≤40 inches (male)</td>
<td>73</td>
<td>67</td>
</tr>
<tr>
<td>Body mass index 18.5 to 24.9 kg/m²</td>
<td>38</td>
<td>35</td>
</tr>
<tr>
<td>Nonsmoking</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>Physical activity &gt;3 days/week</td>
<td>25</td>
<td>18</td>
</tr>
<tr>
<td>Saturated fat &lt;10% kcal/day</td>
<td>43</td>
<td>39</td>
</tr>
<tr>
<td>Dietary cholesterol &lt;300 mg/day</td>
<td>73</td>
<td>72</td>
</tr>
</tbody>
</table>

LDL-C levels in our subjects, a less intensive intervention, suboptimal diet adherence in the SI arm, and/or a greater-than-expected benefit of the CIN. A meta-analysis of the effects of dietary intervention programs on CVD risk factors showed a significant correlation between decreased intake of saturated fat and reductions in LDL-C and HDL-C. Well-controlled diet studies have also shown that HDL-C levels decrease with low saturated fat intake. Our controls reduced saturated fat intake and had a decline in HDL-C. Although physical activity levels increased in the CIN, the improvement was 3-fold greater in the SI. The lack of significant change in HDL-C in the SI may have been blunted by increased physical activity levels.

Few other studies have evaluated the impact of lifestyle interventions on family members of patients hospitalized with CVD. In a community-based risk factor intervention trial targeted to blacks with a family member hospitalized with cardiac disease, brief screening and lifestyle counseling by a nurse practitioner was associated with a 2-fold greater likelihood of achieving target LDL-C goals compared with an enhanced primary care model. The greater impact of the intervention on reducing LDL-C may have been related to a significant increase in use of pharmacotherapy and access to an exercise room, neither of which were used in the current study. In a recent family-based CVD prevention study, patients and their families were recruited in hospitals, an intervention for lifestyle and CVD risk factors was delivered by a multidisciplinary team, and results were compared with usual care at 1 year. Similar to our findings, the intervention was associated with healthier diets including reductions in saturated fat intake and more physical activity in participants. Control of LDL-C also improved in the intervention and usual care groups and was greatest in the high-risk patients, but similar to our study, mean changes from baseline were small.

Prospective data that have shown that small changes in lipids may have a significant impact on CVD risk. The mean decrease in HDL-C in our control group was 3.2%, which translates to a >2-mg/dL decrease in >1 year. A 1-mg/dL (0.026 mmol/L) increment in HDL-C was associated with significant reductions in fatal CVD in men and women (3.7% and 4.7%, respectively). Small changes in rates of chronic diseases, such as CVD, may translate to substantial reductions in numbers of deaths. Barter et al have shown that HDL-C levels are predictive of major CVD events even when LDL-C levels are low. These data suggest that interventions with a modest effect on lipids may be important in reducing the burden of disease when applied to large populations.

It is important to note that in our study of family members of patients with CVD, >90% of subjects had a Framingham score <10%, and overall adherence to lifestyle and several other primary prevention recommendations was suboptimal. Less than half of subjects were consuming a diet with <10% of energy from saturated fat, and less than one third reported physical activity >3 days per week. These findings highlight the importance to provide specific information and guidance regarding lifestyle and CVD risk to a vulnerable population at a captive moment. Additionally, we documented that a significant proportion of participants were unaware of their elevated blood pressure or cholesterol levels, underscored the rationale of a hospital-based screening program for patient family members who may not be aware of their risk. Secondary prevention guidelines committees might consider the addition of a recommendation to systematically identify and refer family members of CVD patients for screening and lifestyle counseling where appropriate.
The strengths of our study include the randomized controlled design, the substantial representation of women and racial/ethnic minorities, and the 94% 1-year follow-up rate. There are also several important limitations. The dietary data were based on food-frequency questionnaires and may not reflect consumption as accurately as dietary food records. Physical activity was self-reported, and there may have been misclassification of true levels. The finding that HDL-C was significantly higher in the SI versus the CIN is biological support for the validity of self-reported changes. Our results may not generalize to other populations, because our participants may have been motivated to improve lifestyle because they accepted an invitation to participate in a 1-year trial, they had a family member hospitalized with CVD, and they were provided $100 to offset their expenses to return for a 1-year follow-up visit. It is unknown whether study participants differed from nonparticipants in ways that could limit the generalizability of our findings.

In conclusion, a special screening and educational intervention was not more effective in lowering LDL-C than the CIN in the trial. Both groups had similar reductions in LDL-C after hospitalization of a family member with CVD. The intervention was associated with a significantly greater improvement in diet score and beneficial effects on changes in HDL-C compared with the CIN, although these findings may be due to chance and should be confirmed in future studies.

Our data also showed that adherence to primary prevention recommendations is suboptimal, and many individuals were unaware they had abnormal cholesterol or blood pressure levels. Systematic screening of family members of patients hospitalized with CVD may represent an opportunity to identify individuals at risk before a cardiovascular event occurs. Additional research is needed to determine cost-effective approaches to improve adherence to lifestyle and primary prevention guidelines for relatives of patients with CVD.

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Disclosures
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Mosca et al. FIT Heart Trial

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**CLINICAL PERSPECTIVE**

Many individuals at heightened risk of cardiovascular disease (CVD) because of genetic or lifestyle factors may not be aware of their risk, which impedes preventive efforts. Teachable moments take advantage of naturally occurring events (eg, a new diagnosis) when patients may be motivated to make behavioral changes. The Family Intervention Trial for Heart Health (FIT Heart) was a 1-year randomized, controlled trial to test the effectiveness of a hospital-based educational intervention among 501 family members of patients hospitalized with CVD. A special intervention (SI) that included personalized risk factor screening, lifestyle counseling by certified health educators at regular intervals, and progress reports to physicians was compared to a control intervention (CI) in which participants received a general prevention message at baseline. There was no difference between the SI and the CI at 1 year in the primary end point mean percent change in low-density lipoprotein cholesterol; both groups had similar significant reductions. The SI showed a significant improvement in diet score, a secondary end point, compared to the CI. The CI showed a significant reduction in high-density lipoprotein cholesterol, but not the SI. Both groups significantly improved their intakes of saturated fat and cholesterol, and the SI was more likely to exercise >3 days/week at 1 year. The changes in secondary end points were small and may be attributable to chance. However, the findings are plausible, and small improvements in lifestyle could be clinically important independent of lipid effects. An important finding was that many family members were unaware of their risk factors. Physicians should routinely ask about recent family cardiac events and consider screening family members during this “motivational moment.”
A Novel Family-Based Intervention Trial to Improve Heart Health: FIT Heart: Results of a Randomized Controlled Trial
Lori Mosca, Heidi Mochari, Ming Liao, Allison H. Christian, Dana J. Edelman, Brooke Aggarwal and Mehmet C. Oz

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