Fish Oil Supplementation and Risk of Ventricular Tachycardia and Ventricular Fibrillation in Patients With Implantable Defibrillators
A Randomized Controlled Trial

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Context  Clinical studies of omega-3 polyunsaturated fatty acids (PUFAs) have shown a reduction in sudden cardiac death, suggesting that omega-3 PUFAs may have antiarrhythmic effects.

Objective  To determine whether omega-3 PUFAs have beneficial antiarrhythmic effects in patients with a history of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF).

Design and Setting  Randomized, double-blind, placebo-controlled trial performed at 6 US medical centers with enrollment from February 1999 until January 2003.

Patients  Two hundred patients with an implantable cardioverter defibrillator (ICD) and a recent episode of sustained VT or VF.

Intervention  Patients were randomly assigned to receive fish oil, 1.8 g/d, 72% omega-3 PUFAs, or placebo and were followed up for a median of 718 days (range, 20-828 days).

Main Outcome Measures  Time to first episode of ICD treatment for VT/VF, changes in red blood cell concentrations of omega-3 PUFAs, frequency of recurrent VT/VF events, and predetermined subgroup analyses.

Results  Patients randomized to receive fish oil had an increase in the mean percentage of omega-3 PUFAs in red blood cell membranes from 4.7% to 8.3% (P < .001), with no change observed in patients receiving placebo. At 6, 12, and 24 months, 46% (SE, 5%), 51% (5%), and 65% (5%) of patients randomized to receive fish oil had ICD therapy for VT/VF compared with 36% (5%), 41% (5%), and 59% (5%) for patients randomized to receive placebo (P = .19). In the subset of 133 patients whose qualifying arrhythmia was VT, 61% (SE, 6%), 66% (6%), and 79% (6%) of patients in the fish oil group had VT/VF at 6, 12, and 24 months compared with 37% (6%), 43% (6%), and 65% (6%) of patients in the control group (P = .007). Recurrent VT/VF events were more common in patients randomized to receive fish oil (P < .001).

Conclusion  Among patients with a recent episode of sustained ventricular arrhythmia and an ICD, fish oil supplementation does not reduce the risk of VT/VF and may be proarrhythmic in some patients.

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hypothesis that omega-3 PUFAs have antiarrhythmic properties, we performed a prospective, double-blind, randomized, placebo-controlled trial of fish oil supplementation in patients with a recent episode of sustained ventricular arrhythmia.

METHODS

Patients

Patients at 6 medical centers in the United States were eligible for entry if they were receiving an implantable cardioverter defibrillator (ICD) for an electrocardiogram-documented episode of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) that was not the result of acute myocardial infarction or a reversible cause or who had a preexisting ICD and had received ICD therapy for an episode of electrocardiogram-documented VT/VF within the previous 3 months.

Patients taking class I or class III antiarrhythmic medications were excluded to prevent the inclusion of proven antiarrhythmic nonresponders and to prevent potential confounding problems related to antiarrhythmic drugs acting through similar mechanisms as fish oil. Patients who ate more than 1 fatty fish (salmon, Chilean sea bass, sardine, herring, or mackerel) meal per week or who had taken flaxseed oil, cod liver oil, or fish oil supplements in the last month were also excluded. Patients provided written informed consent to participate. The study conformed to the principles of the Declaration of Helsinki, was approved by the institutional review board at each site, and was monitored by an independent data and safety monitoring board.

Definition of race/ethnicity was required by the National Institutes of Health to determine any possible interaction with the treatment effect and to ascertain balance between the group assignments. Race/ethnicity was determined by asking participants to classify themselves.

Treatment

The computer-generated, blocked randomization scheme (in block sizes of 6) was stratified by arrhythmia at entry (VT vs VF) and by enrollment at the time of ICD implantation (yes vs no). Patients were randomly assigned to receive a total dosage of 1.8 g/d of fish oil, consisting of 42% eicosapentaenoic acid (EPA) and 30% docosahexaenoic acid (DHA), or placebo (olive oil: 73% oleic acid, 12% palmitic acid, 0% EPA/DHA). Oils were provided by Hoffman-LaRoche Inc (Nutley, NJ) as ethyl esters of the fatty acids. The dose of fish oil was based on preliminary studies that showed that it would increase the percentage of red blood cell membrane fatty acid made up of EPA and DHA to a level between 6% and 10%. Levels in this range have been shown to be associated with a low relative risk of sudden death in case-control studies.7,8 All patients received ongoing dietary counseling to not change their intake of fish and to follow the American Heart Association step 1 low-fat diet.

Follow-up

After randomization, patients were followed up for as long as 2 years with monthly clinic visits at the enrolling center for the first 3 months and every 3 months thereafter. At each visit, participants were asked to report anticipated, unanticipated, and serious adverse events. FIGURE 1 shows the flow of patients through the study. Implantable cardioverter defibrillator programming was left to the discretion of the attending cardiologist, and the slowest heart rate programmed to trigger ICD therapy was tracked. At all visits, the ICD memory was checked for occurrence of episodes of ICD therapy. Blood was drawn for lipid analysis at baseline and months 1, 2, 3, 6, 12, 18, and 24. Fatty acid levels in plasma and red blood cells were analyzed by methods that have been previously published9 and the results were expressed as percentage of total fatty acids.

ICD Electrogram Review

A printout of each episode of ICD therapy was reviewed by the local investigator and by a member of the electrogram committee, both of whom were blinded to the treatment assignment of the patient. Episodes of ICD therapy were classified as VT, VF, atrial fibrillation, supraventricular tachycardia, oversensing, or unknown using methods previously reported.10,11 When there was disagreement between the investigator and the committee member on the interpretation of the tracings, the tracings were reviewed by the entire committee and classified by consensus. Only episodes of ICD therapy determined to be due to VT or VF are included as end points in this article.

Electrophysiologic Study

Forty-nine patients at 2 participating centers who were enrolled at the time of ICD implantation underwent electrophysiologic testing through the ICD at implantation and again 3 months later. Testing consisted of (1) effective refractory period determined at drive train cycle lengths of 400 milliseconds and 600 milliseconds using a single extra stimulus decremented at 10-millisecond intervals at 2× threshold; (2) up to 3× extra stimuli at both drive train cycle lengths; and (3) defibrillation threshold measurement using the step-up, step-down method, starting with an initial energy of 12 J and using 3-J steps, with defibrillation...
threshold defined as the minimum energy leading to successful defibrillation.

**End Points**

The primary end point was the time to the first episode of VT/VF leading to ICD therapy. Predetermined secondary analyses were performed in subgroups of qualifying arrhythmia (VT vs VF), history of coronary artery disease, and ejection fraction. Based on an estimated 75% incidence of these arrhythmias in placebo patients during 2 years of follow-up and a 15% dropout rate, we calculated that 100 patients per group or 200 total would be required for 92% power to detect a 33% reduction in event rate with treatment using a 2-tailed α level of .05. Pre-defined secondary end points were: (1) time to days with recurrent episodes of VT/VF leading to ICD therapy; (2) time to first use of antiarrhythmic medication; and (3) change in defibrillation threshold, inducibility of VT or VF, and the ventricular effective refractory period from baseline to 3 months.

**Statistical Analysis**

All analyses were performed based on intention to treat. The baseline characteristics of patients randomized to receive fish oil vs placebo were compared using the t test and the χ² test as appropriate. Differences in percentage of total plasma and red blood cell membrane fatty acids over time were determined using separate mixed-model analysis of variance models. The initial value was used as a covariate to control for any differences at baseline, with the most appropriate covariance structure selected using the Akaike information criterion. Least square–adjusted means were estimated and compared for all analysis of variance effects; a Tukey adjustment was applied within analyses to account for multiple comparisons. All analyses were performed with SAS software, versions 8 and 9 (SAS Institute Inc, Cary, NC).

Actuarial analyses were performed using the Kaplan-Meier method, and the statistical significance of observed differences was determined using the log-rank test. Survival is presented as percentage (standard error). For the primary analysis of time to first arrhythmia and the secondary analysis of time to first VT/VF analysis in patients with VT at study entry, a plot of the natural logarithm of the negative of the natural logarithm of survival vs the natural logarithm of survival was reviewed to assess the proportional hazards assumption.

As a secondary analysis, a Cox proportional hazards model was used to assess the significance of the primary outcome controlling for other baseline characteristics. Variable selection was performed with these baseline characteristics using all possible regression models with the score statistic and stepwise addition of variables. Treatment group was then added to the best model to determine if it was a significant predictor after controlling for significant baseline characteristics. To determine if compliance modified the primary analysis, all repeated measurements of plasma and red blood cell membrane omega-3 PUFA levels were analyzed as time-dependent covariates using the Cox proportional hazards model. The Anderson-Gill application of the Cox proportional hazards model was used to assess the effect of treatment group on the number of days with recurrent VT/VF events. That is, each day on which a patient had VT or VF was modeled as an event. For example, if a patient experienced arrhythmias on days 1, 3, and 5, each of these 3 arrhythmias was included in the model with the time measured from the last recorded arrhythmia.

**RESULTS**

Patients were enrolled from February 1999 until January 2003. Follow-up was completed July 2003, with a median follow-up duration of 718 days (range, 20-828 days). Patients assigned to receive fish oil were followed up for a median of 720 days (range, 20-828 days) and patients assigned to receive placebo were followed up for a median of 718 days (range, 60-815 days). The baseline demographics of patients assigned to fish oil and those randomized to placebo were well balanced (TABLE 1). There were no significant differences in serious adverse events in patients assigned to fish oil compared with those assigned to placebo, with the possible exception of an excess of hospitalizations for neurologic events in patients assigned to placebo (TABLE 2). Seventeen patients assigned to fish oil and 26 assigned to placebo stopped...
study medication prior to the end of the trial because of adverse effects or unrelated severe illness.

**Red Blood Cell and Plasma Omega-3 PUFA Levels**

At baseline, there was no difference in plasma or red blood cell membrane plasma omega-3 fatty acids (DHA plus EPA), expressed as a percentage of total fatty acids, between patients assigned to placebo and fish oil. Baseline plasma fatty acid levels were 1.9% in patients assigned to fish oil, rising significantly to 4.4% by 1 month, with no significant change thereafter through 24 months. Red blood cell membrane levels in patients assigned to fish oil rose significantly, from 4.7% at baseline to 6.8% at 1 month, and continued to increase to 8.3% at 3 months, with no significant change thereafter. Patients assigned to placebo had no significant change in plasma or red blood cell omega-3 fatty acid levels over 24 months of follow-up. Plasma and red blood cell membrane omega-3 fatty acid levels in patients assigned to fish oil were higher than levels in patients assigned to placebo at all follow-up time points (P < .001). There was no difference over time or between groups in the plasma or red blood cell membrane levels of the 2 primary components of the placebo, oleic acid and palmitic acid.

**Time to First Episode of VT or VF**

During follow-up, patients received ICD therapy for a total of 45 VF episodes and 901 VT episodes. Other episodes of ICD therapy included 47 for atrial fibrillation, 124 for supraventricular tachycardia, 2 for oversensing, and 31 that could not be classified. The results of the analysis of the primary end point, time to first episode of ICD therapy for VT/VF after randomization, are shown in Figure 2. At 6, 12, and 24 months after randomization, respectively (Figure 2), compared with 37% (6%), 43% (6%), and 65% (6%) among those assigned to placebo (P = .007).

Regardless of ejection fraction, the group assigned to fish oil tended to have a shorter time to first episode of ICD therapy for VT/VF than those assigned to placebo (Figure 3). Figure 4 shows the hazard ratios and confidence intervals (CIs) for the analysis of time to first VT/VF episode for each of the prespecified subgroups and for the study population as a whole. Given the predictive value of presenting arrhythmia on the primary end point, a post hoc analysis of time to first VT and first VF episode was performed. There was a trend toward an increased risk of VT in patients assigned to fish oil but no apparent effect on the risk of VF (Figure 5). In multivariate analysis, an ejection fraction less than 40% (hazard ratio, 1.7; 95% CI, 1.1-2.5) and VT as the qualifying arrhythmia (hazard ratio, 2.0; 95% CI, 1.3-3.1) were the independent predictors of time to ICD therapy for VT/VF. When treatment assignment was added to this model, the

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**Table 2. Adverse Events**

<table>
<thead>
<tr>
<th>Adverse Events</th>
<th>Placebo (n = 100)</th>
<th>Fish Oil (n = 100)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>10</td>
<td>4</td>
<td>.16</td>
</tr>
<tr>
<td>Cardiac</td>
<td>5</td>
<td>2</td>
<td>.44</td>
</tr>
<tr>
<td>Sudden cardiac</td>
<td>0</td>
<td>2</td>
<td>.47</td>
</tr>
<tr>
<td>Hospitalization for</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina</td>
<td>7</td>
<td>10</td>
<td>.61</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>16</td>
<td>21</td>
<td>.53</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>12</td>
<td>14</td>
<td>.83</td>
</tr>
<tr>
<td>Neurologic</td>
<td>6</td>
<td>0</td>
<td>.04</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>4</td>
<td>7</td>
<td>.54</td>
</tr>
<tr>
<td>Coronary revascularization</td>
<td>4</td>
<td>2</td>
<td>.68</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>3</td>
<td>1</td>
<td>.61</td>
</tr>
<tr>
<td>Cancer</td>
<td>4</td>
<td>3</td>
<td>&gt; .99</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>12</td>
<td>11</td>
<td>&gt; .99</td>
</tr>
</tbody>
</table>

**Figure 2. Survival Curves for Time to First Episode of ICD Therapy for VT or VF in All Patients and in VT Patients by Fish Oil vs Placebo Group**

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fish oil group had a hazard ratio of 1.4 (95% CI, 0.96-2.0).

Secondary End Points
An actuarial analysis of the time to recurrent episodes of VT/VF (ie, time to each day on which an arrhythmia occurred) showed a significant increase in the incidence of days with episodes of ICD therapy for VT/VF in patients assigned to fish oil (P<.001). Patients assigned to fish oil had ICD therapy for VT/VF on a mean of 3.5 (SE, 0.6) days compared with 2.2 (0.5) days for patients assigned to placebo over the 2-year study.

To investigate whether compliance may have affected the results, analyses were performed of time to first episode of VT/VF using plasma or red blood cell membrane omega-3 PUFA levels as time-dependent covariates. These analyses showed no significant association between (P>.20) omega-3 PUFA levels and time to first episode of VT/VF. During follow-up, class I or class III antiarrhythmic medications were initiated for 12% (3%), 22% (4%), and 29% (5%) of patients assigned to fish oil at 6, 12, and 24 months, respectively, compared with 13% (3%), 15% (4%), and 25% (5%) in patients assigned to placebo (P=.45). There was no difference in the results of electrophysiologic testing between patients assigned to fish oil vs placebo (TABLE 3).

COMMENT
In this study, fish oil supplementation in patients with an ICD and a history of VT or VF did not prevent episodes of VT or VF. It is unlikely that the lack of demonstrable beneficial effect was due to inadequate power given the overall trend toward an increased risk of VT/VF in the fish oil group. In fact, the trend toward a higher incidence of VT/VF overall with fish oil supplementation, the significant increase in VT/VF occurrence in patients assigned to fish oil (P<.001) all suggest that fish oil may be proarrhythmic in this population.

The finding that fish oil did not reduce the risk of VT or VF and may be proarrhythmic in this population is unexpected given evidence suggesting that omega-3 PUFAs reduce cardiovascular mortality via an antiarrhythmic effect. Long-term feeding studies in rats have shown that a diet high in omega-3 PUFAs significantly reduced risk of VF during acute ischemia compared with control animals. In an ischemic VF model in dogs, acute infusion of free omega-3 PUFAs reduced the risk of ischemic VF by 75%. In support of the concept that omega-3 PUFAs have direct antiarrhythmic effects are the findings that omega-3 PUFAs change the spontaneous beating rate of cultured myocardial cells, prevent and terminate drug-induced arrhythmias in cultured myocardial cells, and can bind to and inactivate myocardial sodium channels, a class I antiarrhythmic effect. There is also evidence for antiarrhythmic effects of omega-3 PUFAs in humans. A retrospective analysis of the Physician’s Health Study revealed that eating at least 1 fish meal per week was associated with a reduced relative risk of sudden cardiac death after correction for known risk factors. There was no association between fish intake and
the risk of myocardial infarction. When blood levels of omega-3 PUFAs were studied in a nested case-control analysis in this same population, patients in the highest quartile of omega-3 PUFA levels (6%-10% of whole blood fatty acid) had the lowest relative risk of sudden cardiac death after correction for known risk factors. Similarly, Sisovcick et al showed that cardiac arrest cases had significantly lower intake of fish and lower levels of red blood cell omega-3 PUFAs than controls.

Four prospective randomized trials have shown that supplementation with fish oil or other measures to increase the intake of omega-3 PUFAs is associated with a decreased risk of sudden death without a consistent change in risk of myocardial infarction. The largest of these studies was the GISSI-Prevenzione trial, in which 11,323 patients who had experienced a myocardial infarction in the prior 3 months were randomized to receive 1 g of fish oil or placebo daily in an open-label study. Total mortality and sudden death mortality were significantly reduced in patients assigned to fish oil, with significant differences in mortality apparent within 4 months. There was no observed difference in the rate of myocardial infarction suggesting that the reduced mortality was due to an antiarrhythmic effect of fish oil. As a result of this evidence, the American Heart Association has recommended 2 fatty fish meals per week for the general population and 1 g of EPA/DHA per day for patients with coronary artery disease. Similarly, the US Food and Drug Administration has authorized a qualified health claim of a reduction in the risk of coronary artery disease for food containing EPA and DHA.

Despite enthusiasm for the beneficial effects of omega-3 PUFAs, ours is not the first study to suggest a potentially proarrhythmic effect of fish oil. Burr et al randomized 3114 men with coronary artery disease to a programmed intervention to either not change their intake of fish or to eat 2 portions of fatty fish per week (patients who could not follow the increased fish diet were given 3 g/d of fish oil as an alternative). In this unblinded study, the risk of sudden death was higher among the 1572 patients randomized to fish or fish oil (hazard ratio, 1.54; 95% CI, 1.06-2.23), with a more prominent effect in the subset of 462 patients given fish oil (hazard ratio, 1.84; 95% CI, 1.11-3.05). It is possible that in that unblinded study, overall compliance with cardiovascular health recommendations was poor because patients consuming a fish diet or fish oil thought they were protected. However, this could not have occurred in our double-blind study.

Another consideration is whether our placebo, olive oil, could have been antiarrhythmic. This explanation seems unlikely given that animal studies have shown that olive oil does not have antiarrhythmic properties. Furthermore, there was no significant difference between the groups over time in the red blood cell and plasma levels of the main constituents of the olive oil placebo, oleic acid and palmitic acid. In contrast, red blood cell and plasma omega-3 PUFA levels, which have been shown to correlate well with myocardial omega-3 PUFA levels, were significantly elevated when first tested after 1 month of therapy, suggesting that an adverse effect of fish oil was responsible for the prominent divergence of the event curves observed over the first 90 days of therapy.

The discordance between our results and the antiarrhythmic properties of omega-3 fatty acids in other studies may lie in the fact that experimental...
models used ischemic VF as an end point and the cohort and clinical trials showing benefit used sudden death as an end point, whereas in this study we used ICD therapy for VT or VF as the primary end point. The end point of ICD therapy for VT or VF certainly addresses the influence of fish oil on the risk of ventricular arrhythmias but may not be an ideal surrogate for the risk of sudden death. In addition, the prior clinical studies were performed in patients with recent myocardial infarction and relatively well-preserved ventricular function, in whom ischemic VF might be expected to be the primary cause of sudden death. In contrast, the patients in our study were substantially different in that they had not had a recent myocardial infarction, on average had significantly reduced left ventricular function, and, perhaps most important, had a history of sustained ventricular arrhythmia.

A hypothesis suggested by Leaf et al supports the concept that fish oil may have its most profound antiarrhythmic effects in the setting of acute ischemia and VF. They point out that by shifting the voltage required to change sodium channels from inactive to activatable, fish oil could effectively inactivate sodium channels in partially depolarized ischemic myocardium, thereby preventing rapid spontaneous depolarizations and reducing the risk of VF. Although the majority of patients in our study had coronary artery disease, they all had experienced episodes of sustained VT or VF outside of the setting of acute myocardial infarction. The mechanism of arrhythmia in such patients, especially VT patients in whom we observed the most clearly adverse response to fish oil, is unlikely to be ischemic but, instead, in the majority was probably myocardial scar-based reentry. Although sodium channel blockade of the sort produced by omega-3 PUFAs may be antiarrhythmic in the setting of acute ischemia, sodium channel blockade has been shown to be proarrhythmic in patients with premature ventricular contractions after myocardial infarction.27 Our study has several limitations. Although we designed the study to have a 92% chance of detecting a 33% reduction in event rate, the total event rate in the placebo group and the difference between placebo and fish oil were less than predicted. As a result, post hoc analysis revealed that the study had only 70% power to detect a 33% reduction in event rate. This finding suggests that the lack of a demonstration of statistically significant proarrhythmia in the primary end point may be a function of inadequate power. However, we cannot rule out the possibility that the observed differences are due to chance or are the result of an imbalance in an unmeasured variable that affects the risk of VT/VF.

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12. Antiarrhythmics Versus Implantable Defibrillators (AVID) Investigators. A comparison of antiar-

Author Contributions: Drs Raitt, Morris, and MacNulty had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Raitt, Connor, Morris, Halperin, Chugh, Gerhard, Kraemer, Marchant, McAnulty. Acquisition of data: Raitt, Connor, Morris, Kron, Halperin, Chugh, McClelland, Cook, MacMurdy, Swenson, Connor, Gerhard, Oseran, Marchant, Calhoun, Shidner, McAnulty. Analysis and interpretation of data: Raitt, Connor, Morris, Halperin, Chugh, MacMurdy, Connor, Kraemer, Marchant, McAnulty. Drafting of the manuscript: Raitt, Morris, Kron, Cook, Kraemer, McAnulty.

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CONCLUSIONS

This study was undertaken to better understand the previously observed reduction in sudden death mortality after myocardial infarction associated with fish oil supplementation. The fact that we were not able to demonstrate an antiarrhythmic effect of fish oil does not call into question the potential benefits of fish oil or dietary fish intake in patients who have had a myocardial infarction.22 Instead, our results suggest that the mechanism of benefit, if due to antiarrhythmic properties, may not be due to the suppression of reentrant VT or VF. The lack of benefit and the suggestion that fish oil supplementation may increase the risk of VT or VF in some patients with ICDs can reasonably be interpreted as evidence that the routine use of fish oil supplementation in patients with ICDs and recurrent ventricular arrhythmias should be avoided.


An old literary truth: What we write pleases us, otherwise we surely wouldn’t have written it.
—Johann Wolfgang von Goethe (1749-1832)