Omega-3 Fish Oils and Atrial Fibrillation: My Story

by Michael Mooney

I was diagnosed with atrial fibrillation (afib) when I was 25 years old in 1978. I’d had an atrial fibrillation where I fell face forward on a plate just after finishing desert during dinner at a friend’s house, my heart beating with odd rhythms at hyperspeed. Then I had another one walking up a hill with a friend, who caught me as I started to fall.

The doctor I saw gave me a prescription for Digoxin, which helps regulate heart beat. I was anti-drug – trying to be “all natural,” so I quit taking it after a couple months and started taking a bunch of dietary supplements that I’d read could help reduce afib events. I managed to handle afibs fairly well, taking the supplements and if an afib started I did yogic deep-breathing and could pop out of it in a few minutes most of the time. Sometimes it took longer.

I didn’t have another uncontrollable afib event for almost twenty years, but when it returned it lasted for two days and nothing would stop it. In frustration I went to my acupuncturist and he stopped the afib doing acupuncture on me for about an hour. He sent me home with a Chinese remedy, burnt licorice tea. If I had an afib the burnt licorice tea would stop it in about ten minutes most of the time, but it wasn’t perfect.

This went on for about another five years. I had some control of the afibs but they still happened ten or fifteen times a year. Usually, one would happen if I lifted weight and started lifting vigorously too quickly, without enough warm-up. The best way to handle it seemed to be to have to drink a bunch of water, lay flat and do yogic deep breathing. Not fun.

Sometime in the late 1990’s I saw a medical journal study that indicated that fish oil could reduce the potential for afibs. I’d been taking 3 fish oil capsules a day, thinking that was a good amount, but the study made me think I needed more to help reduce afibs.

After a little experimentation, over a period of about six months I found that if I took four fish oil capsules (Jarrow Brand MaxDHA) twice a day I had no afibs. I did fine, having no afibs, for about two years, but suddenly I had an afib.

What was wrong?

After assessing things I realized that I had been skipping taking my evening fish oil dose for about two weeks – since I’m don’t really like taking all the nutrient pills I take, so I was only taking four capsules a day, not enough it seemed.
So, the all-important concept of dosing came into play. I needed a total of eight capsules a day to stop my afib. Taking only four capsules for two weeks equaled the return of afibs. (Today in 2010, I take five capsules twice a day, which has reduced the number of afibs to basically none.)

I am asked by others if this will work for them. Good question. There are several studies at this point that strongly suggest that people who experience afib will experience a reduction or cessation of afibs if they get enough omega-3 fish oil fats. (By the way, flax oil doesn’t work.)

However, there are caveats, so no one can say that it is universally true that this will work for everyone. One study showed that people who had pace-makers had more afibs when they took fish oil, so it does not work the same for everyone.

Therefore, it is wise to consult a knowledgeable doctor about this, also being aware that most American doctors have little to no understanding of the value of fish oils in cardiovascular health, so keep that in mind, too.

The other thing about fish oils is that they are good for many things in health, including brain health, and cancer risk reduction, so if you take them and they stop your afib, they will also be improving your chances of better long-term health, in general.

Below are reprints of abstracts of some studies pointing at using omega-3 fish oil fats to reduce the potential for afibs. You might print them out and show them to your doctor for their opinion.

Wishing you good health!
Michael Mooney

Omega-3 polyunsaturated Fatty Acid supplementation reduced atrial fibrillation recurrence after pulmonary vein antrum isolation.

Abstract
OBJECTIVE: To assess if patients treated with omega-3(n-3) polyunsaturated fatty acids (PUFAS) had lower procedural failure rates compared to an untreated population.
METHODS
AND RESULTS: From January 2004 to 2007, 1500 PVAI patients underwent catheter ablation. Two hundred and eighty five (19%) patients were treated with PUFAs. These patients were matched in a nested case controlled analysis. After matching, there were 129 patients in the PUFA group and 129 in the control group. Thirty-five (27.1%) patients in the study group had early recurrence vs. 57 (44.1%) in the control group p-value< 0.0001. Twenty-nine (23.2%) patients in the PUFA group vs. 41 (31.7%) in the non-PUFA group had procedural failure (p-
value < 0.003). There were no significant differences in complications in the PUFA and non-PUFA groups. CONCLUSION: Patients treated with PUFAs had lower incidences of early recurrence and procedural failure compared to an untreated population.


**Beneficial effects of intravenously administered N-3 fatty acids for the prevention of atrial fibrillation after coronary artery bypass surgery: a prospective randomized study.**

Heidt MC, Vician M, Stracke SK, Stadlbauer T, Grebe MT, Boening A, Vogt PR, Erdogan A.

**Abstract**

**BACKGROUND:** Atrial fibrillation (AF) is a common complication after coronary artery bypass grafting operation (CABG). Experimental data have shown antiarrhythmic effects of n-3 polyunsaturated fatty acids (PUFA) on myocardial cells. Orally administered PUFA could significantly reduce the rate of postoperative AF. We assessed the efficacy of PUFA for the prevention of AF after CABG. PUFA were given intravenously to prevent variation in bioavailability. **METHODS AND RESULTS:** 52 patients were randomized to the interventional group, 50 served as controls. In the control group free fatty acids (100 mg soya oil/kg body weight/day) were infused via perfusion pump, starting on admission to hospital and ending at discharge from intensive care. In the interventional group PUFA were given at a dosage of 100 mg fish oil/kg body weight/day. Primary end point was the postoperative development of AF, documented by surface ECG. Secondary end point was the length of stay in the ICU. The demographic, clinical and surgical characteristics of the patients in the two groups were similar. Postoperative AF occurred in 15 patients (30.6%) in the control and in 9 (17.3%) in the PUFA group (P < 0.05). After CABG, the PUFA patients had to be treated in the ICU for a shorter time than the control patients. No adverse effects were observed. **CONCLUSIONS:** Perioperative intravenous infusion of PUFA reduces the incidence of AF after CABG and leads to a shorter stay in the ICU and in hospital. Our data suggest that perioperative intravenous infusion of PUFA should be recommended for patients undergoing CABG.


**Omega-3 polyunsaturated fatty acids inhibit transient outward and ultra-rapid delayed rectifier K+currents and Na+current in human atrial myocytes.**

Li GR, Sun HY, Zhang XH, Cheng LC, Chiu SW, Tse HF, Lau CP.

**Abstract**

**AIMS:** The omega-3 (n-3) polyunsaturated fatty acids (omega-3 PUFAs) eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) from fish oil were recently reported to have an anti-atrial fibrillation effect in humans; however, the ionic mechanisms of this effect are not fully understood. The present study was designed to determine the effects of EPA and DHA on transient outward and ultra-rapid delayed rectifier potassium currents (I(to) and I(Kur)) and the voltage-gated sodium current (I(Na)) in human atrial myocytes. **METHODS AND RESULTS:** A whole-cell patch voltage clamp technique was employed to record I(to) and I(Kur), and I(Na) in human atrial myocytes. It was found that EPA and DHA inhibited I(to) in a concentration-dependent manner (IC(50): 6.2 microM for EPA; 4.1 microM for DHA) and positively shifted voltage-dependent activation of the current. In addition, I(Kur) was suppressed by 1-50 microM EPA (IC(50): 17.5 microM) and DHA (IC(50): 4.3 microM). Moreover, EPA and DHA reduced I(Na) in human atrial myocytes in a concentration-dependent manner (IC(50): 10.8 microM for EPA; 41.2 microM for DHA) and negatively shifted the potential of I(Na) availability. The I(Na) block by EPA or DHA was use-independent. **CONCLUSION:** The present study demonstrates...
for the first time that EPA and DHA inhibit human atrial I(to), I(Kur), and I(Na) in a concentration-dependent manner; these effects may contribute, at least in part, to the anti-atrial fibrillation of omega-3 PUFAs in humans.


Omega-3 fatty acid: a role in the management of cardiac arrhythmias?
Cheng JW, Santoni F.

Abstract
OBJECTIVE: The objective of this study was to review and evaluate published evidence on the use of omega-3 fatty acid in the prevention and treatment of atrial and ventricular arrhythmias. Postulated mechanisms of the antiarrhythmic effects of omega-3 fatty acid are discussed. DATA SOURCES: Peer-reviewed articles/abstracts published in English language were identified from MEDLINE and Current Content databases (both 1966 to May 15, 2008) using the search terms fish oil, omega-3 fatty acid, sudden death, ventricular arrhythmia, and atrial fibrillation. Citations from available articles were also reviewed for additional references. Abstracts presented at recent professional meetings are also reviewed. STUDY SELECTION AND DATA EXTRACTION: Observational studies and interventional clinical studies published on omega-3 fatty acid or fish consumption and atrial or ventricular arrhythmias and sudden cardiac death are selected. The design and results of the studies are evaluated. DATA SYNTHESIS: Several mechanisms have been postulated to explain the antiarrhythmic effect of omega-3 fatty acid. It is believed that omega-3 fatty acid has an indirect effect on the autonomic nervous system, inhibits the fast, voltage-dependent sodium and L-type calcium channels, restores a favorable omega-6 fatty acid/omega-3 fatty acid balance, and exerts anti-inflammatory effects. While the majority of observational evidence demonstrated that increased consumption of omega-3 fatty acid was associated with reduction in risk of sudden cardiac death, in ventricular arrhythmia, there was evidence suggesting that omega-3 fatty acid in patients experiencing nonischemic ventricular arrhythmia may be proarrhythmic. Other studies demonstrated a neutral effect. In terms of management of atrial fibrillation, short-term small-scale studies demonstrated that the use of omega-3 fatty acid preoperatively may reduce the incidence of postoperative atrial fibrillation. However, such observations were not consistent with those reported from retrospective cohorts. CONCLUSIONS: Additional studies are needed to evaluate the effect of omega-3 fatty acid before it can be routinely recommended for the management of arrhythmia.


Omega-3 Fatty Acid supplementation for the prevention of arrhythmias.
Chung MK.

Abstract
Fish oil, or omega-3 (n-3) polyunsaturated fatty acid (PUFA), supplements have been purported to produce potential health benefits. One of the strongest supported effects of n-3 PUFAs may be their potential benefits in reducing the risk of sudden cardiac death. This article reviews clinical and mechanistic studies that may explain the effects of these agents on ischemic arrhythmias, sudden death, and atrial fibrillation.


The role of fish oil in arrhythmia prevention.
Anand RG, Alkadri M, Lavie CJ, Milani RV.

Abstract
Numerous epidemiological studies, case-control series, and randomized trials have demonstrated the ability of fish oil to reduce major cardiovascular events, particularly sudden cardiac death and all-cause mortality. We discuss the potential benefits of fish oil therapy to improve overall autonomic tone and potentially reduce the risk of major ventricular and atrial arrhythmias. Specifically, this review focuses on how fish oil therapy has performed in 3 primary prevention trials in patients with implantable cardioverter defibrillators, reviews the effects that fish oil has on the autonomic nervous system, focuses on the use of fish oil as a novel therapy for atrial fibrillation, and revisits other beneficial properties of fish oil (ie, ability to lower serum triglycerides, anti-inflammatory effects, and possible improvements in arterial pressure/diastolic function). We also discuss the safety profile of fish oil, including effects on bleeding time and bleeding complications as well as provide commentary regarding fish oil supplementation in light of increasing contaminants contained in fish. In summary, any patient with documented coronary heart disease and those with risk factors for sudden cardiac death, such as left ventricular dysfunction, left ventricular hypertrophy, prior myocardial infarction, or high-grade ventricular dysrhythmias, should consider fish oil supplementation. The American Heart Association recommends four 3-ounce servings of oily fish weekly. For those who cannot eat fish or do not have access to fish, as well as those who would prefer not to eat fish regularly, capsules of fish oil are readily available in various concentrations. At the present time, we recommend doses of eicosapentanoic acid and docosahexanoic acid in the combined range of 800 to 1000 mg/day for primary and secondary prevention of cardiovascular disease.


**Omega-3 fatty acid supplementation reduces one-year risk of atrial fibrillation in patients hospitalized with myocardial infarction.**


**Abstract**

**PURPOSE:** Current strategies for avoiding atrial fibrillation (AF) are of limited value. We aim to assess the relationship between omega-3 fatty acids (n-3 PUFA) and AF occurrence in post-myocardial infarction (MI) patients. **METHODS:** A population study, linking hospital discharge records, prescription databases, and vital statistics, was conducted and included all consecutive patients with MI (ICD-9: 410) in six Italian local health authorities over a 3-year period. A propensity score (PS)-based, 5-to-1, greedy 1:1 matching algorithm was used to check consistency of results. Sensitivity analysis was performed to assess the robustness of findings. **RESULTS:** N-3 PUFA reduced the relative risk of the hospitalization for AF [hazard ratio (HR) 0.19, 95% CI 0.07-0.51] and was associated with a further and complementary reduction in all-cause mortality (HR 0.15, 95% CI 0.05-0.46). PS-based matched analysis and sensitivity analysis confirmed the main results. **CONCLUSION:** n-3 PUFA reduced both all-cause mortality and incidence of 1-year AF in patients hospitalized with MI.


**Omega-3 fatty acids: antiarrhythmic, proarrhythmic or both?**

von Schacky C.

**Abstract**

**PURPOSE OF REVIEW:** Recent publications seem to indicate no or an untoward effect of the marine omega-3 fatty acids eicosapentanoic acid and docosahexaenoic acid on cardiac rhythm. This review puts these developments into perspective. **RECENT FINDINGS:** In-vitro or in animal models, little pro-arrhythmic effect, but many antiarrhythmic mechanisms of omega-3
fatty acids have been documented. In intervention studies in humans, eicosapentaenoic acid plus docosahexaenoic acid suppressed new atrial fibrillation in patients undergoing coronary bypass grafting. More importantly, in systematic reviews, it has been demonstrated that eicosapentaenoic acid plus docosahexaenoic acid reduce sudden cardiac death by 50%. In a recently published intervention study with eicosapentaenoic acid in patients at high cardiovascular risk in Japan, sudden cardiac death was rare. Sudden cardiac death is even rarer in the general population of Japan: it occurs 20 times less frequently than in the general population in Europe, e.g. Germany. In Japan, levels of eicosapentaenoic acid plus docosahexaenoic acid are high (omega-3 index estimated around 11%), whereas in Germany levels of eicosapentaenoic acid plus docosahexaenoic acid acids are low (omega-3 index around 4%). These and other data strengthen the concept that a low omega-3 index is a risk factor for sudden cardiac death, as a tool to assess the status of a person in terms of eicosapentaenoic acid plus docosahexaenoic acid, and as a means to monitor therapy with eicosapentaenoic acid plus docosahexaenoic acid. SUMMARY: Concerns about pro-arrhythmic effects of eicosapentaenoic acid plus docosahexaenoic acid are largely theoretical. The evidence in favour of an antiarrhythmic effect is overwhelming, especially, when factoring in the omega-3 index.


Omega-3 polyunsaturated fatty acids prevent atrial fibrillation associated with heart failure but not atrial tachycardia remodeling.


Abstract
BACKGROUND: There is epidemiological evidence that omega-3 polyunsaturated fatty acids (PUFAs) reduce the risk of atrial fibrillation (AF), but clinical data are conflicting. The present study assessed the effects of PUFA on AF in experimental models. METHODS AND RESULTS: We studied the effects of oral PUFA supplements in 2 experimental AF paradigms: electrical remodeling induced by atrial tachypacing (400 bpm for 1 week) and congestive heart failure-associated structural remodeling induced by ventricular tachypacing (240 bpm for 2 weeks). PUFA pretreatment did not directly change atrial effective refractory period (128 +/- 6 [mean +/- SEM] versus 127 +/- 2 ms; all effective refractory periods at 300-ms cycle lengths) or burst pacing-induced AF duration (5 +/- 4 versus 34 +/- 18 seconds). Atrial tachypacing dogs had shorter refractory periods (73 +/- 6 ms) and greater AF duration (1128 +/- 412 seconds). PUFAs suppressed ventricular tachypacing-induced increases in AF duration (952 +/- 221 versus 318 +/- 249 seconds; P<0.05) and attenuated congestive heart failure-related atrial fibrosis (from 19.2 +/- 1.1% to 5.8 +/- 1.0%; P<0.001) and conduction abnormalities. PUFAs also attenuated ventricular tachypacing-induced hemodynamic dysfunction (eg, left ventricular end-diastolic and left atrial pressure from 12.2 +/- 0.5 and 11.4 +/- 0.6 mm Hg, respectively, to 6.4 +/- 0.5 and 7.0 +/- 0.8 mm Hg; P<0.01) and phosphorylation of mitogen-activated protein kinases (extracellular-signal related and P38 kinase). CONCLUSIONS: PUFAs suppress congestive heart failure-induced atrial structural remodeling and AF promotion but do not affect atrial tachycardia-induced electrical remodeling. The beneficial effects of PUFAs on structural remodeling, possibly related to prevention of mitogen-activated protein kinase activation, may contribute to their clinical anti-AF potential.


Anti-arrhythmic properties of N-3 poly-unsaturated fatty acids (n-3 PUFA).
Lombardi F, Terranova P.

Abstract
Omega-3 fatty acids (Poly-Unsaturated Fatty Acids or PUFA n-3) have been initially found to reduce plasma levels of triglycerides and to increase levels of high-density lipoprotein in patients with marked hypertriglyceridemia. However, in both bench research studies and clinical trials, omega-3 fatty acid intake has recently been associated with an anti-arrhythmic efficacy. At experimental level, n-3 PUFA administration produces several actions on ionic channels regulating transmembrane action potential. At clinical level, the most significant finding was the reduction in the incidence of sudden death in survivors of MI in the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI)-Prevention trial and the subsequent recommendation for administration of fish oil as part of the post-infarction regimen in European guidelines. More recently, Omega-3 fatty acids administration has been associated with a lower incidence of atrial fibrillation in patients who underwent cardiac surgery. Contrasting results have been instead reported in patients with implantable cardioverter defibrillators. This article reviews in detail the basic and clinical research studies of fish oil as an anti-arrhythmic entity, the types of arrhythmias that have been beneficially affected by fish oil administration, and the presumed and known mechanisms by which the beneficial actions are exerted.


New antiarrhythmic treatment of atrial fibrillation.
Naccarelli GV, Wolbrette DL, Samii S, Banchs JE, Penny-Peterson E, Gonzalez MD.

Abstract
Antiarrhythmic pharmaceutical development for the treatment of atrial fibrillation (AF) is moving in several directions. The efficacy of existing drugs, such as carvedilol, for rate control and, possibly, suppression of AF, is more appreciated. Efforts are being made to modify existing agents, such as amiodarone, in an attempt to ameliorate safety and adverse effect concerns. This has resulted in promising data from the deiodinated amiodarone analog, dronedarone, and further work with celivarone and ATI-2042. In an attempt to minimize ventricular proarrhythmia, atrial selective drugs, such as intravenous vernakalant, have demonstrated efficacy in terminating AF in addition to promising data in suppression recurrences when used orally. Several other atrial selective drugs are being developed by multiple manufacturers. Other novel therapeutic mechanisms, such as drugs that enhance GAP junction conduction, are being developed to achieve more effective drug therapy than is offered by existing compounds. Finally, nonantiarrhythmic drugs, such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, high-mobility group coenzyme A enzyme inhibitors and omega-3 fatty acids/fish oil, appear to have a role in suppressing AF in certain patient subtypes. Future studies will clarify the role of these drugs in treating AF.


n-3 (omega-3) polyunsaturated fatty acids prevent acute atrial electrophysiological remodeling.
da Cunha DN, Hamlin RL, Billman GE, Carnes CA.

Abstract
BACKGROUND AND PURPOSE: Recent reports suggest that n-3 (omega-3) polyunsaturated fatty acids (PUFAs) may reduce atrial fibrillation (AF). Reduction of the atrial effective refractory period (ERP) is believed to be an important early remodeling event that favors the development and perpetuation of AF. We hypothesized that n-3 PUFAs would attenuate early atrial electrophysiogical remodeling in a canine model of acute atrial tachypacing. EXPERIMENTAL
APPROACH: Adult dogs of either sex received n-3 PUFAs (n=6), n-6 PUFAs (n=6), or saline (n=6) infused over 1 h. After a stable ERP was established, treatment was initiated concurrently with 6 h of rapid atrial pacing (400 b.p.m.). Serial right atrial ERPs were measured during rapid atrial pacing, and induction of atrial tachyarrhythmias was attempted at the conclusion of each study. KEY RESULTS: There was no change in P wave duration or in the PQ, QRS, QT or QTc intervals in any of the treatment groups. N-3 PUFA treatment significantly reduced the shortening of atrial ERP, compared to both control groups (P<0.05). In separate experiments, the same n-3 PUFA infusion was given to dogs remaining in normal sinus rhythm. During sinus rhythm, n-3 PUFA infusion did not alter any electrocardiogram (ECG) parameter or the atrial ERP. CONCLUSIONS AND IMPLICATIONS: We conclude that acute n-3 PUFA treatment prevents acute atrial electrophysiological remodeling during high rate activity, which may minimize the self-perpetuation of AF.


Antiarrhythmic effects of omega-3 fatty acids.

Reiffel JA, McDonald A.

Abstract
Fish oil, and omega-3 fatty acids in particular, have been found to reduce plasma levels of triglycerides and increase levels of high-density lipoprotein in patients with marked hypertriglyceridemia, and a pharmaceutical-grade preparation has recently received approval from the US Food and Drug Administration to market for this purpose. However, in both bench research studies and clinical trials, evidence for clinically significant antiarrhythmic properties has also been detected in association with omega-3 fatty acid intake. Arguably the most significant finding in this data set was the reduction in the incidence of sudden death in survivors of myocardial infarction in the Gruppo Italiano per lo Studio della Sopravvivenza nell’Infarto Miocardico (GISSI)-Prevenzione trial and the subsequent recommendation for administration of fish oil as part of the postinfarction regimen in Europe. This article reviews in detail the basic and clinical research studies of fish oil as an antiarrhythmic entity, the forms of preparation and/or administration that appear to possess these properties and those that do not, the types of arrhythmias (ventricular ectopy and atrial fibrillation as well as ventricular tachyarrhythmias) that have been beneficially affected by fish oil administration, and the presumed and known mechanisms by which the beneficial actions are exerted.

Michael’s Comment: The following study provided details that show how high dose fish oil supplementation can reduce atrial fibrillation.

Heart Rhythm 2010 Dec 10. [Epub ahead of print]

Effects of Chronic Omega-3 Polyunsaturated Fatty Acid Supplementation on Human Atrial Electrophysiology.

Kumar S, et al.

ABSTRACT

BACKGROUND: Omega-3 polyunsaturated fatty acids in fish oils may have anti-fibrillatory effects. Their mechanism of action in humans is poorly understood.
OBJECTIVE: To investigate the effects of chronic fish oil supplementation on human atrial electrophysiology.

METHODS: Two groups of patients without clinical AF or structural heart disease and fish intake less 1/week were prospectively recruited into a control group (n=30) and a fish oil group (n=31). The latter were prescribed 6g/day of fish oil for greater than 1 month prior to an electrophysiology (EP) study. The following were compared at time of EP: serum omega-3 levels, right atrial and coronary sinus effective refractory periods (ERPs), inter-atrial, intra-atrial, left atrial and coronary sinus conduction at baseline and the maximal conduction delay with the shortest propagated extra-stimulus, and inducibility of AF (10 inductions/patient).

RESULTS: The following significant differences were noted favoring the fish oil group at time of EP: (i) 2 fold higher total omega-3 levels (P<.001) (ii) lengthening of ERPs by 8-14% at all measured sites and pacing cycle lengths (P<.05) (iii) no effect on baseline inter-atrial, intra-atrial, left atrial and coronary sinus conduction but a significant attenuation of maximal conduction delay (P<.05) (iv) less inducible AF (AF greater than 30 s: 24.2% vs. 7.9%, P<.001) (v) shorter mean duration of induced AF (P=.003) and (vi) prolongation of induced AF cycle length (P<.001).

CONCLUSIONS: Chronic fish oil supplementation in humans prolongs atrial refractoriness and reduces vulnerability to inducible AF. These electrophysiological changes may explain the anti-fibrillatory effect of chronic fish oil ingestion.


Long-term fish consumption is associated with protection against arrhythmia in healthy persons in a Mediterranean region--the ATTICA study.

Chrysohoou, C, et al.

Omega-3 Fats From Fish Oil Reduce Heart Arrhythmias

A study in Greece by Christine Chrysohoou from the University of Athens showed that a diet rich in omega-3 essential fatty acids from fish oils had beneficial effects on heart rhythms that would reduce the potential for fatal abnormal heart rhythms.

The study said that fish consumption could improve the electrical conduction properties of heart cells.

"Long-term consumption of fish is associated with lower QT interval in free-eating people without any evidence of cardiovascular disease. Thus, fish intake seems to provide anti-arrhythmic protection..." said the authors.

The study looked at 3,042 people (1,514 men and 1,528 women) with an average age of 45. Diets were evaluated with a validated food questionnaire. Daily or weekly intake of 156 different foods was recorded, along with alcohol consumption and physical activity.

The study reported that people who ate more than 300 grams of fish per week had significantly lower QT scores (13.6%) than people who did not eat fish. After adjusting for confounding factors such as age, sex, physical activity status, body mass index, smoking habits and intake of
nuts, the reduction in QT associated with high fish consumption rose to 29.2% compared to non-consumers.

Lower QT scores indicate a lower resting heart rate. Other studies have linked a higher resting heart rate to an increased risk of sudden death. Lowering the heart rate is a significant health benefit.


**Effect of dietary omega-3 polyunsaturated fatty acids on the inducibility of ventricular tachycardia in patients with ischemic cardiomyopathy.**

Metcalf RG, Sanders P, et al,

In a placebo-controlled study with 26 patients with coronary artery disease who were being implanted with defibrillators, supplementation with fish oil at 3,000 milligrams per day over a 6 week period of time was found to exert anti-arrhythmic effects.

The effects of fish oil on the inducibility of ventricular tachycardia, and its link to sudden cardiac death, were investigated in this study. Subjects underwent electrophysiologic studies before and after the intervention to determine how much stimulation was required to induce ventricular tachycardia.

Subjects who took fish oil were found to experience significantly less ventricular tachycardia.

In the fish oil group:

1. 42% of subjects had no inducible ventricular tachycardia (compared to 7% in the control group);
2. 42% required more aggressive stimulation to induce ventricular tachycardia (compared to 36% in the control group);
3. 8% required identical stimulation (compared to 36% in the control group);
4. 8% required less stimulation (compared to 21% in the control group).

The authors conclude, "These findings suggest that dietary fish oil can have an antiarrhythmic effect."

**DISCLAIMER:** Always consult your cardiologist before any use of fish oil and show them these studies.

Michael Mooney
www.michaelmooney.net