Fish consumption and risk of coronary heart disease. What does the evidence show?

The concept that fish intake may reduce the risk of coronary heart disease apparently originated from reports on the small population of non-acculturated Eskimos in arctic Greenland where a high consumption of marine animals (e.g. seal, fish) was observed\(^1\text{-}^3\). It was claimed that coronary rates were low, but available data were, and remained, limited and tenuous. Also, it has been observed that inhabitants of the Japanese island of Okinawa have low coronary heart disease mortality rates, and they too consume high amounts of fish\(^3\). These observations prompted epidemiological investigations — the first from the Netherlands — on the relationship of fish consumption to coronary heart disease and myocardial infarction.

The most recent results are from the Chicago Western Electric Study\(^5\). This study involved 1822 men employed at the Hawthorne Works of the Western Electric Company in Chicago, Illinois, U.S.A. At entry, in 1958, participants were ages 40–55, free of cardiovascular disease, 68% blue collar workers and 47% Catholic. Information on food consumption was collected by two extensive diet histories, administered in 1958 and 1959, including a dietary questionnaire on amounts of fish and other foods usually consumed. Consumption of fish, categorized into four strata (0, 1–17, 18–34, and \(\geq 35\) g day\(^{-}\))\(^{-}\), was significantly and independently associated in a graded manner with lower 30-year risk of myocardial infarction, particularly non-sudden myocardial infarction death. Multivariate relative risks (95% confidence intervals) of death from coronary heart disease, all myocardial infarction (sudden and non-sudden), and non-sudden myocardial infarction were, respectively, 0-62 (0-40–0-94), 0-56 (0-33–0-93) and 0-33 (0-12–0-91) for men consuming 35+ grams of fish per day compared to non-consumers. This relationship prevailed across the 30 years of the study, and after adjustment for multiple potential confounders, demographic, biomedical, and dietary.

Design, methods, and results of other studies on fish intake and coronary heart disease-myocardial infarction are summarized in reviews by Shekelle and Stamler\(^6\) and Nordoy\(^7\). In brief, findings from four prospective population studies (Zutphen, Rotterdam, Sweden, U.S. physicians), two case-control studies (northern Italy and Seattle), and one secondary prevention trial (Diet and Reinfarction Trial, Wales, U.K.) also showed a significant inverse relationship between fish intake and risk of coronary heart disease or myocardial infarction mortality. However, other studies (Norway, Hawaii, and U.S. Health Professionals Study) have reported no association. This apparent inconsistency in results may hypothetically be due to: (1) different methods of dietary assessment and measurement of fish intake; (2) different distributions of reported fish intake, such that in some cohorts (e.g. Norway and Hawaii) few or no people ingested little or no fish, hence there was no fully suitable reference group; (3) different mixes of type of fish eaten (e.g. fatty and lean fish); (4) different study sites and times with populations' general dietary content differing (e.g. levels of intake of cholesterol, saturated fats, antioxidants, fibre) possibly influencing the relationship of fish to coronary heart disease risk; for example, Western Electric and Dutch cohorts vs Norwegian, Hawaiian Japanese-American, U.S. health professionals cohorts, the latter studied in the 1980s and 1990s, after widespread awareness of general dietary approaches to coronary heart disease prevention and of the idea that fish may 'protect' against coronary heart disease; (5) the possibility of bias due to influence of this later recent awareness, i.e. people more coronary-prone differentially becoming greater fish eaters in the 1980s, with possible inversion of the fish–coronary heart disease relationship from 'cause-effect' to 'effect-cause' (e.g. Health Professionals Study finding on fish and CABG); (6) different durations of follow-up, ranging from 4 years to decades (e.g. findings in Physicians' Health Study with 4- and with 12-year follow-up); (7) different coronary heart disease end-points, with only a few studies reporting on fish and fatal myocardial infarction, and only two prospective studies (Western Electric Study and Physicians Health Study\(^8\)) on fish and suddenness of coronary heart disease death; (8) interpretation of findings (e.g. in the Health Professionals Study, are data on coronary heart disease death more soundly interpreted as indicating no relationship or an inverse relationship of fish intake to this end-point?); (9) chance, i.e. random variation across studies in results.

Data from the epidemiological studies with positive results indicate that ingestion of small amounts of fish, including mainly lean (non-fatty) fish, is sufficient to produce the association with risk of coronary heart disease or myocardial infarction. It
seems unlikely that decades-long intake of small amounts of fish protect, if fish is indeed aetiologically protective, via the very small amounts of omega-3 long-chain polyunsaturated fatty acids so ingested, for example, via known influences on plasma lipids or thrombogenic factors. Such effects apparently require much larger amounts of these fatty acids. Could protection, then, if real, relate to influences of these fatty acids on cell membranes? Or could components of fish protein, for example, particular mixes of amino acids, be involved?

The resolution of this issue has important implications for public health and nutritional recommendations. Thus, further studies — observational and interventional, particularly trials — are needed to resolve whether there is an aetiologically significant protection against coronary heart disease or myocardial infarction afforded by regular ingestion of modest amounts of fish.

In the meantime, merely adding fish to a nutritionally adverse diet will not grant a population immunity from epidemic coronary heart disease. However, including fish as part of a diet that is moderate in total fat (<30% of calories), low in saturated fat (<10% of calories), low in dietary cholesterol (<250 mg. day⁻¹), low in salt, and (for drinkers) low in alcohol and contains ample amounts of fresh fruits, vegetables, whole grains, and legumes is sound dietary advice for prevention of heart disease. With this comprehensive approach, fish in some forms — deep fat fried, highly salted — should be avoided.

Also, the putative heart protective benefits of fish consumption should not be used to support the use of fish oil pills or other supplements. Sound information is not available to support population use of fish oil capsules providing n-3 fatty acids in much larger amounts than fish itself, and there is evidence of possible harm. To achieve further decline in coronary rates and end the epidemic, the population should be encouraged to focus less on pills and more on improved life styles (eating and exercise habits, non-smoking, avoidance of excess alcohol intake).

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References