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THE HARD FACTS AND NOT SO SIMPLE TRUTH ABOUT DIETARY FATS

OPŠTE-PRIHVAĆENE ČINJENICE I NE TAKO JEDNOSTAVNA ISTINA O MASTIMA U LJUDSKOJ ISHRANI

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Apstrakt
Brojna epidemiološka istrazivanja urađena od pocetka 60-tih godina prošlog veka indikovala su povezanost načina ishrane, posebno unosa zasićenih masti, sa rizikom nastanka kardiovaskularnih oboljenja. Iako veliki broj istraživača u svojim studijama ukazuje na povezanost načina ishrane i razvoja bolesti, veoma je mali broj studija koje su zaista bazirane na validnim modelima, tako da naučni dokazi istraživanja ne potvrđuju misljenja i hipoteze autora. Kako su tokom zadnjih decenija kardiovaskularna oboljenja postala jedan od vodećih uzroka smrtnosti u industijalizovanim zemljama, od izuzentne je važnosti da buduća epidemiloška i klinička istraživanja budu dizajnirana nepristrasno u odnosu na već opšte-prihvaćena mšljenja kako bi dala što objektivnije i statistički validne rezultate. Samo objektivna analiza svih potencijalno promenljivih faktora rizika, kao što su: ukupan unos kalorija, fizička aktivnost, različiti načini ishrane i stres, dovešće do boljeg razumevanja uzroka, a samim tim i do moguće prevencije i boljeg tretmana kardiovaskularne bolesti.

According to the American Heart Association, Coronary Heart Disease (CHD) is the largest major cause of death among North Americans. CHD, by definition, is a narrowing of the small blood vessels that supply blood and oxygen to the heart. It is a well accepted opinion that CHD is caused by atherosclerosis: a build-up of fatty material and plaque inside the coronary arteries. The ”Diet-Heart” hypothesis, presented initially in the early 1960’s, was largely based on the ecological studies associating increased consumption of dietary fat with the increased levels of cholesterol in the blood; and consequently, with the increased risk for developing CHD (1-3). These researchers suggested that there was a strong positive correlation between high dietary fat intake and mortality rates related to CHD. Over the next few decades, the low-fat-food trend became predominant in the medical community and was extensively popularized by the media.

The first step to unravelling the truth about fats in relation to CHD is to understand fats themselves. Fats are classified into four major groups: saturated, monounsaturated, polyunsaturated and man-made hydrogenated/trans, fats. Saturated fats are solid at room temperature and do not oxidize at high temperatures. They can be found in animal fats, tropical oils and are manufactured by the human body from carbohydrates. Monounsaturated fats are liquid at room temperature and are relatively stable at high temperatures. Olive oil and some nuts are rich with these fats. Also, human bodies are able to make monounsaturated fats from saturated fats. Polyunsaturated fats (omega-6 and omega-3) are liquid at room temperature. These fats are most commonly found in cottonseed, corn, sunflower, and soybean oil. These oils are unstable, oxidize easily and should never be heated or used in cooking. Human bodies cannot make these fats and therefore they are called ”essential fatty acids” (4). All types of fats, especially saturated fats have important roles in various metabolic and physiological processes, especially in preserving the integrity of cell membranes. However, when these fats are exposed to extreme heat, like broiling or frying, the fats and cholesterol become susceptible to conformational oxidation and may contribute to heart disease (5). It is essential to understand that of all the fatty acids consumed by the body, polyunsaturated oils are most easily turned into harmful fats by food processing, especially during hydrogenation in order to create trans-fats. Despite this, the food industry is mostly based on using hydrogenated polyunsaturated oils.

The primary advocate of the ”Diet-Heart” hypothesis was Dr. Ancel Keys in the early 1960’s. He compared the total amount of fat consumed to the mortality rates across seven countries and published his research as the ”Seven Countries Study” (1,2). He was among the first to suggest that increased consumption of dietary fats might be the main

reason behind CHD and he recommended that consumption of fats should be significantly reduced in everyday diet. A few years later, other researchers published work demonstrating and supporting the same postulations (3-6). Over the next few decades a number of epidemiological studies addressed the association between total dietary fat intake and risk of CHD in an effort to validate the ”Diet-Heart” hypothesis. The multi-year British study, The U.S. Multiple Risk Factor Intervention Trial, (7) and The Lipid Research Clinics Coronary Primary Prevention Trial (8) compared eating habits, with the risk of developing CHD and mortality rates of more than 350,000 thousand men, over a period of few decades. Interestingly enough, instead of confirming the hypothesis, these studies demonstrated that those groups of patients consuming low-fat/low-cholesterol diets had only a marginal and insignificant reduction in incidence of CHD. Most importantly, those patients on a low-fat diet had a significant increase in overall mortality from cancer, brain haemorrhages, suicide and violent death (9, 10). Another significant study was The Framingham Heart Study, which lasted 40 years and involved over 6,000 people from the town of Framingham, Massachusetts. After completion of this study, it was acknowledged that the results were indicative, yet inconclusive in supporting the ”Diet-Heart” hypothesis (11).

Regardless of the lack of unbiased, statistically valid, epidemiological data, both the medical community and the popular press, interpreted and promoted these studies as a confirmation that the consumption of saturated fats was a primary risk factor of CHD. During the 70s, fat-phobia was fully enforced upon the public and fats were identified as the main cause of obesity, CHD and stroke. Also, contrary to the inconclusive and conflicting studies and trials, main stream scientists, continued to report that the more one lowers fat and cholesterol in one’s diet, the more one reduces the risk of heart disease (12). In 1984, at the National Institute of Health (NIH) Consensus Conference on Lowering Blood Cholesterol to Prevent Heart Disease, the medical community recognized cholesterol as a major cause of heart disease and called for incorporating lipid management into standard clinical practice (13). Once in place and afterwards, being aggressively reinforced by the establishment, the ”Diet-Heart” hypothesis significantly impacted the main-stream media, the food industry, medical research, and most importantly preventive health-care. Consequently, the ”low-fat”, and more so the ”zero-fat” diet, reached pandemic proportions and began impacting the life-styles of millions across the globe.

In the 1980s the NIH considered performing a definitive, double-blind, placebo-controlled, randomized clinical trial to demonstrate whether low-fat diets actually prolong life. However, this trial would be very expensive due to the thousands of individuals who would have to be monitored over many decades to provide statistically significant results. Instead of performing the trial, in the early 1990s, the NIH made a nation-wide recommendation that everyone should significantly restrict fat intake. This shows that the decisions on lowering plasma cholesterol through a low-fat diet, was done without direct valid experimental proof. Furthermore, the U.S. Surgeon General’s Office, in 1988 published its 700-page landmark ”Report on Nutrition and Health,” identifying fat as the single most harmful factor of the American diet. However, eleven years later the same report was revoked without any explanation to the public.

Things went from bad to worse when the pharmaceutical industry caught up with this trend and began developing and testing various cholesterol-lowering drugs. Many clinical trials were conducted, testing the efficacy of cholesterol-lowering drugs on individuals with clinical CHD and even on healthy participants; however, no single one could be considered conclusive. The results from the LRC-CPPT study showed that the probability of dying from a heart attack during the seven-plus years of the study was reduced from 8.6% in the placebo group to 7.0%; meaning that the probability only dropped from 2.0% to 1.6 %”(14).

More recently, an independent international group of eminent researches (14-17), completed comprehensive reviews of the major epidemiological past ”Diet-Heart” studies. These researchers were surprised to find that none of the major studies were able to confirm the association between dietary fat intake and increased blood cholesterol levels, or a relationship between increased blood cholesterol levels and risk of developing CHD. Moreover, the reviewers pointed out many serious flaws with the methodology, data misinterpretations and contradictory conclusions. Positive correlation between these factors had been found in four studies, while six studies found no such association (15, 16). Furthermore, cross-sectional and comparative post-mortem studies on Masai (18) and Indian (19) people have shown that high fat and cholesterol intake has no impact on CHD and mortality. The Honolulu Heart Program demonstrated that the more saturated fat, cholesterol and calories one ate, the lower his/her blood cholesterol (20). Also, case-control studies, cohort studies and randomized clinical trials (21-23) were not able to find epidemiologic evidence that link a high intake of cholesterol with CHD risk.

Over a few decades, pro ”Diet-Heart” hypothesis supporters had tried to prove that by lowering fat and cholesterol levels, the development of CHD could be prevented. Although North Americans’ average fat and cholesterol levels intake have decreased over the last six decades, compared to European countries (24), the occurrence of heart disease remained unchanged. At the same time, the incidence of obesity and diabetes has increased considerably. Interestingly enough, obesity epidemics occurred at the same time as Americans adopted the low-fat diet. It has been well documented that, obesity and diabetes increase the risk for CHD, which could explain why heart disease incidence does not decrease in individuals with either of these two conditions regardless of if they followed a low-fat diet or not (25).

The U.S. Department of Agriculture’s dietary guidelines are presented to the public as a Food Guide Pyramid, which recommends that fats and oils be eaten ”sparingly” favouring grains, over fats. During the past 30 years, over thousands of low-fat food products have been offered to consumers who by avoiding fat, ate less animal proteins and more carbohydrates, most of them from processed food instead from fresh sources. A low-fat diet is by definition, is a high-carbohydrate diet. When fat is removed from food products, various carbohydrates and flavourings must be added to the product to retain its texture and taste. Unfortunately, high carbohydrate diets can raise triglyceride levels, and create a condition known as ”insulin resistance,”

which is associated with a several-fold increase in heart disease risk, even in those patients having normal cholesterol levels (26). In view of this information, animal fat and cholesterol seem to be unlikely culprits for CHD and in order to prevent coronary events, it is important to treat modifiable cardiovascular risk factors. There are a number of factors in modern diets, including total caloric intake, excess consumption of vegetable oils, trans-fats, and processed carbohydrates in the form of sugar and white flour (27-32) which can contribute to CHD. Also, contributing factors could be a sedentary lifestyle, chronic exposure to stress, and deficiencies in fat-soluble vitamins and minerals due to low intake of butter, fish, shellfish, eggs, and organ meats (33-35).

For hundreds of thousands of years our ancestors mainly consumed unprocessed foods like meat, fish, vegetables, fruits, and nuts. They consumed more protein, fat and fibres than we do these days, and their diet was almost starch-free. It is important to recognize that the human body can live healthily without starch, but not without fat. Without consuming fat we would become severely ill in a matter of weeks. The human body has a number of hormones and enzymes to metabolize fat, but only insulin to deal with starches and sugars. When we have normal fat consumption, insulin levels usually remain low in healthy individuals. This suggests that we are designed to metabolize fat slowly and efficiently, and burn it completely. Our ancestors were not obese and most likely they did not have diabetes. The consumption of sugars and starches in North America skyrocketed after WWII and five years ago the United States Centers for Disease Control (USCDC) reported that 32% of American adults over the age of 20 are obese, while 66.5% are overweight (26). Before the 1920s, coronary heart disease was rare in North America, however over the past few decades CHD became the number one killer.

CONCLUSION

Despite 50 years of research, there is still no solid evidence that a low-fat diet prevents CHD and prolongs life. A considerable number of studies contest the hypothesis that dietary fat and high cholesterol play a major role in the causation of atherosclerosis and CHD. Also, a meticulous review of the published reports related to the performed epidemiological studies show that the results are either insignificant, or contradictory in determining the association. The truth is that human bodies need fats. It is particularly important that infants and growing children consume enough fats along with fat-soluble vitamins and minerals. However, the right fats and oils like organic butter, extra virgin olive oil, and expeller-expressed flax should be consumed (36-41). There are many possible causes of CHD, but the emphasis being placed on the "Diet-Heart" hypothesis probably prevents their exploration by restricting funding and intellectual resources. In addition, it is of utmost importance that the new research and discoveries in this field should not be altered or misinterpreted so as to support the old hypotheses and ideas. Breaking this cycle may offer a great opportunity for new unbiased approaches to research in this field and will help to ensure a meaningful contribution to science and more importantly, to mankind.

Abstract

During the past six decades, multiple epidemiological studies were performed with their results indicating a strong positive association between dietary intake of saturated fats and risk of coronary heart disease (CHD). Even though a wealth of literature links dietary fat intake and CHD, the lack of strength of the presented research and the flaws in their methodologies provide only inconclusive evidence that has led to inaccurate and highly debatable conclusions. As CHD remains the number one cause of death in adults in the industrialized world, it is of utmost importance that high-quality, randomized and controlled trials are performed on this topic. Future epidemiological and clinical evaluations of dietary and life style patterns, which are performed in an objective and unbiased manner, should provide valid associations and a better understanding of the modifiable risk factors of CHD.

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