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Review

Dietary fats and cardiovascular disease: Putting together the pieces of a complicated puzzle

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ABSTRACT

Dietary fatty acids play significant roles in the cause and prevention of cardiovascular disease (CVD). Trans fatty acids from partially hydrogenated vegetable oils have well-established adverse effects and should be eliminated from the human diet. CVD risk can be modestly reduced by decreasing saturated fatty acids (SFA) and replacing it by a combination of polyunsaturated fatty acids (PUFA) and monounsaturated fatty acids (MUFA). Although the ideal type of unsaturated fat for this replacement is unclear, the benefits of PUFA appear strongest. Both n-6 and n-3 PUFA are essential and reduce CVD risk. However, additional research is needed to better define the optimal amounts of both and to discern the patients and/or general population that would benefit from supplemental n-3 fatty acid intake. Furthermore, consumption of animal products, per se, is not necessarily associated with increased CVD risk, whereas nut and olive oil intake is associated with reduced CVD risk. In conclusion, the total matrix of a food is more important than just its fatty acid content in predicting the effect of a food on CVD risk, and a healthy diet should be the cornerstone of CVD prevention.

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1. Introduction

The World Health Organization (WHO) defines cardiovascular diseases (CVD) as a group of disorders of the heart and blood vessels that includes: coronary heart disease (CHD), cerebrovascular disease, peripheral arterial disease, rheumatic heart disease, congenital heart disease, and deep vein thrombosis and pulmonary embolism. The most common acute clinical manifestations of CVD are myocardial infarction and stroke. In the 1960s and early 1970s, CVD mortality had reached record levels in many developed countries [1]. In the USA, in 1963, 805 people per 100,000 died due to CHD, stroke and other diseases of the circulatory system. By 2010, this figure had dropped to less than 236 per 100,000 [2]. Likewise, in the European Union, in 2000, 115 people per 100,000 died due to CHD. By 2010, this figure had dropped to 76 per 100,000 (Fig. 1). However, despite this progress, CVD still represents the major cause of adult morbidity and mortality in most developed and many developing countries [3,4]. Each year CVD causes over 4 million deaths (47% of all deaths) in Europe and over 1.9 million deaths (40% of all deaths) in the European Union (EU). Overall, CVD is estimated to cost the EU economy almost €196 billion a year [5].

In most cases, the underlying cause of CVD is atherosclerosis, a chronic inflammation of the arteries, which develops over decades in response to the biologic effects of underlying risk factors [6,7]. Atherosclerosis is a multi-factorial disease with both genetic and environmental etiology. The primary modifiable risk factors are dyslipidemia (increased low density lipoprotein (LDL) and/or decreased high density lipoprotein (HDL) cholesterol), hypertension and smoking. Nutritional habits, especially dietary fat are implicated in the process of atherosclerosis. Therefore, both the European Society of Cardiology (ESC) and the American Heart Association (AHA) establish clear targets in dietary fat intake in order to prevent CVD [8,9]. The focus of this review is to provide an overview on the complex relationship between dietary fat intake, plasma lipoproteins and cardiovascular morbidity and mortality, especially in light of a significant number of recent meta-analyses, prospective cohort studies, and randomized control trials (RCTs) published in the field over the last few years.

2. Methods

We reviewed the literature for English-language articles published through Dec 2013 by performing searches of Medline, hand-searching of citation lists, and direct author contact. Inclusion criteria were any systematic review or meta-analysis of RCTs or prospective cohort studies in humans evaluating consumption of dietary fats and main food sources with incident CVD risk, as well as individual RCTs or prospective cohorts. Search terms included "trans fatty acid(s)", "saturated fatty acid(s)", "polyunsaturated fatty acid(s)", "monounsaturated fatty acid(s)", "red meat", "processed meat", "milk", "egg", "olive oil", "nut", "lipoproteins", "blood pressure", "cardiovascular disease(s)", "coronary heart disease" and "stroke". We focused on identifying systematic reviews and/or meta-analyses of RCTs and prospective cohorts on CVD endpoints, supplemented by review of additional recent RCTs and prospective cohorts. Considering complementary strengths and limitations of these two research paradigms conclusions were considered more robust when findings were concordant [10,11]. Wherever there were not enough data on disease outcomes we focused on intermediate risk factors. We excluded a priori animal experiments, ecological studies, case reports, and commentaries.

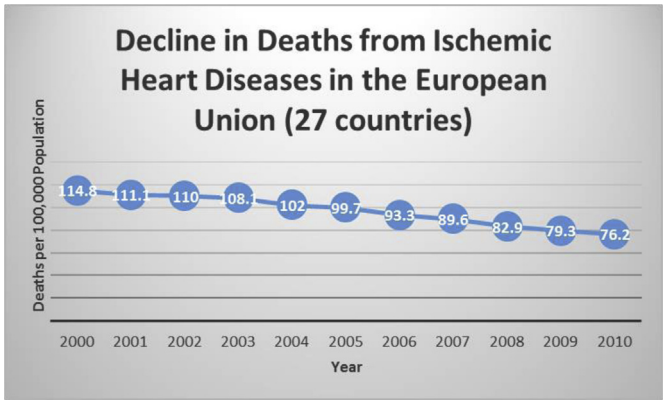

Fig. 1. Standardized death rate by 100,000 inhabitants due to ischemic heart diseases in the European Union (27 countries). Data are derived from Eurostat (Dec 2013) and are subject to change. (http://epp.eurostat.ec.europa.eu/tgm/table.do?tab=table&init=1&language=en&pcode=tps00119&plugin=1).

2.1. The traditional diet-heart paradigm

The diet-heart hypothesis refers to the link between dietary fat intake, blood cholesterol (as the mediating factor) and risk of CVD. Although it stems from animal experimentation undertaken a century ago by Nikolai Anitschkow [12], the Seven Countries Study, led by Ancel Keys, is credited as one of the earliest human epidemiological studies to support this link in humans [13]. The strong correlations between dietary saturated fatty acid (SFA) intake, plasma cholesterol and CVD mortality were sufficient to persuade health professionals that high intakes of SFA, through their effects on plasma cholesterol, were responsible for the high levels of cardiovascular mortality of the 1960s and 1970s across western societies [14,15]. Since then, major advances in clinical and nutritional science have established additional dietary fats (not only saturated fats) and multiple mechanistic pathways (thrombosis, hypertension, insulin resistance, inflammation, endothelial function and arrhythmia) linking diet to CVD [16].

The diet-heart hypothesis has shaped dietary guidelines towards total dietary fat reduction (with an emphasis on saturated fat) without further specifying the replacement nutrient. This historical emphasis has resulted in decreased SFA consumption in the US and many other nations, but with concomitant increases in (mainly refined) carbohydrates; this approach has done little to slow down the increasing rates of obesity and type 2 diabetes [17]. Indeed, a low-(saturated) fat approach, replaced principally by carbohydrates, has not been effective in reducing CHD, stroke, or CVD incidence in a large RCT of women [18]. Nowadays, it is widely recognized that higher-fat diets can be beneficial if healthy fats (polyunsaturated fatty acids (PUFA), monounsaturated fatty acids (MUFA)) are consumed, whereas high-carbohydrate diets (particularly those with high glycemic load) might contribute to CHD [19–21], especially amongst women, overweight, and obese individuals [22–24].

2.2. Impact of dietary fats on blood lipids and lipoproteins

Keys and Hegsted in the 1950s and 1960s, respectively, were the first to demonstrate the quantitative relationship between plasma cholesterol and the amount and type of fat in the diet [25–28]. Although exhibiting minor quantitative differences, they both arrived at the same basic conclusions: dietary cholesterol has a modest plasma cholesterol-raising effect; dietary SFA have potent plasma cholesterol-raising effects; dietary PUFA have a plasma cholesterol-lowering effect; and the cholesterol raising effect of dietary SFA is more potent than the lowering effect of PUFA. Reducing dietary SFA intake has remained the cornerstone of public health nutrition policy for reducing CVD risk ever since [8,9].

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