Dietary cholesterol, once thought to play a primary role in determining plasma cholesterol levels, is now understood to have a negligible effect on blood lipids in most healthy people. On average, increasing one’s intake of dietary cholesterol by 100 mg/day results in a 2.3 mg/dl increase in total cholesterol, a 1.9 mg/dl increase in LDL-cholesterol, and a 0.4 mg/dl increase in HDL cholesterol. Because HDL cholesterol increases with dietary cholesterol intake, the LDL:HDL ratio remains virtually unchanged.

Many factors—including genetic inheritance, body weight, and baseline blood lipids—influence the extent to which plasma cholesterol levels respond to dietary changes. Thus, the relationship between diet and blood lipids is highly individual. In general, research demonstrates that dietary saturated fat and trans-fat have the greatest impact on the lipid profile. Saturated fat intake increases total- and LDL-cholesterol levels. Intake of trans-fat appears particularly harmful since it raises LDL-cholesterol while lowering HDL-cholesterol levels. Data from meta-analyses conducted by Clarke et al. (n=80 studies) and Howell et al. (n=224 studies) indicate that while saturated fat intake raises serum cholesterol levels substantially, dietary cholesterol has only a limited effect. While both analyses showed that a 50 mg change in dietary cholesterol was associated with a 1 mg/dl change in both plasma total and LDL cholesterol levels, they indicated that replacing just 1% of calories from saturated fats with polyunsaturated fats would be expected to decrease plasma total and LDL-cholesterol levels by 3.0 mg/dl and 2.2 mg/dl, respectively. Therefore, limiting dietary saturated fat and trans-fat appears more effective in improving lipid profiles than reducing dietary cholesterol.

Physiological responses to changes in dietary cholesterol are heavily influenced by genetic predisposition. For example, individuals with certain genetic variants of apolipoprotein E tend to have a heightened sensitivity to dietary cholesterol (meaning that their blood cholesterol levels are prone to rise more than average in response to cholesterol in the diet. An estimated 15-20 percent of the population falls into this category.) Even in these cholesterol-sensitive (hyperresponsive) individuals, the effect of cholesterol intake is minimal. On average, cholesterol-sensitive individuals exhibit a 3.9 mg/dl change in plasma total cholesterol in response to an increase or decrease of 100 mg of cholesterol/day, while those with normal sensitivity experience an average change of 1.4 mg/dl.

Many cholesterol-containing foods are also naturally high in saturated fat. Eggs are one of the rare exceptions. While they are high in cholesterol, eggs contain very little saturated fat (one Large egg contains only 1.5 grams) and are low in calories (only 75 calories per Large egg). Egg consumption has been shown in several clinical trials to have only a minimal effect on blood lipids. In one such trial, when 640 mg of cholesterol from eggs was added to the daily diets of 91 healthy men and women, the additional cholesterol had no effect on blood cholesterol levels or LDL atherogenicity in hyporesponsive individuals. In hyperresponsive participants (those who experienced >2.5 mg/dl increase in plasma cholesterol for every additional 100 mg of dietary cholesterol), as LDL-cholesterol levels rose, the LDL subclass pattern shifted toward a larger, less-atherogenic particle type. These findings led the study authors to conclude that limitations on egg intake may not be necessary for healthy men and premenopausal women, and that egg consumption does not increase the atherogenicity of LDL particles.
Impact of egg intake on plasma lipids cont...

Does dietary cholesterol increase the risk for heart disease?

Does egg intake increase the risk for heart disease?

How many eggs should my patients eat in a week?

Data from the Multiple Risk Factor Intervention Trial (MRFIT; a large, randomized, clinical trial with a cohort of 12553 middle-aged men classified as being at high risk for CHD) indicated that there was no significant association between cholesterol intake or egg consumption and plasma cholesterol levels at baseline. The National Health and Nutrition Examination Surveys (NHANES) III also showed no evidence of a relationship between egg consumption and plasma cholesterol levels in a cohort of men and women. Decades of data from the Framingham Heart Study have consistently indicated that there is no relationship between egg intake and plasma cholesterol levels or CHD risk.

Dietary cholesterol has not been associated with atherosclerosis or CHD risk. In a study of 21,930 male subjects, Pietinen et al. found that dietary cholesterol was not associated with the risk of coronary death. Subjects in the highest quintile of dietary cholesterol intake (768 mg/day) had a CHD risk similar to that of subjects in the lowest quintile (390 mg/day). The results of this study are consistent with those reported by Hu et al., who evaluated CHD risk within a lower range of cholesterol intakes.

A recent review article on the relationship between dietary cholesterol and atherosclerosis concluded that "for the general population, dietary cholesterol makes no significant contribution to atherosclerosis and risk of cardiovascular disease." No study has yet established an independent effect of dietary cholesterol on CHD risk after accounting for confounding dietary factors.

Although epidemiological data examining the relationship between egg intake and CHD incidence are limited, a number of these studies indicate that there is no relationship between egg consumption and CHD risk.

For example, Dawber and colleagues examined data from the Framingham Study and found that CHD risk did not differ between participants in the highest vs. the lowest tertile of estimated egg intake. In addition, an analysis of the relationship between per capita egg consumption and cardiovascular mortality rates in 24 countries (data from the World Health Organization) demonstrated a significant, inverse association between egg consumption and CVD mortality. In fact, the countries with the highest per capita egg consumption (Japan, Mexico, France, and Spain) had the lowest rates of CHD mortality, indicating that egg consumption does not contribute to the development of CHD.

The most comprehensive study to date (and the only study specifically designed to examine the relationship between egg consumption and CHD incidence) was undertaken using data collected in the Nurses’ Health Study (80,082 women) and the Health Professionals Follow-up Study (37,851 men). Hu and colleagues found no correlation between egg intake and risk for CHD. In fact, they found that those who ate one egg per week had the same relative risk for CHD as did those who ate an egg a day. As Hu and colleagues concluded, “These findings suggest that consumption of up to 1 egg per day is unlikely to have substantial overall impact on the risk of CHD or stroke among healthy men and women.”

Although no official recommendation exists for egg intake, research suggests that heart disease risk does not increase in healthy adults consuming up to 7 eggs per week. Because physiologic responses to dietary cholesterol are highly individual, making specific recommendations for egg intake for the general population would be impractical, at best. This question is best addressed by an individual’s physician and/or dietitian, who can take into account individual lifestyle factors such as age, weight, physical activity, gender, family history, and typical eating habits before making recommendations regarding dietary intake.

Citations...

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