Letters to the Editor

Eggs and heart disease risk: perpetuating the misperception

Dear Sir:

Although the report by Weggemans et al (1) confirms the findings from many recent reported analyses that dietary cholesterol has a small effect not only on plasma total and LDL cholesterol but also on plasma HDL cholesterol (2–4), their interpretation of the findings contradicts more than a decade of epidemiologic studies showing that dietary cholesterol is not a contributor to heart disease risk. The reviews by Howell et al (2), Clarke et al (3), McNamara (4), and Weggemans et al indicate that a 100-mg/d change in dietary cholesterol increases plasma total cholesterol concentrations by 0.06 mmol/L (2.3 mg/dL), LDL cholesterol by 0.05 mmol/L (1.9 mg/dL), and HDL cholesterol by 0.01 mmol/L (0.4 mg/dL). However, Weggemans et al conclude from their analysis that dietary cholesterol increases the ratio of total to HDL cholesterol and that adding an egg a day to the diet increases heart disease risk by 2%. This conclusion is difficult to accept given that a decade of epidemiologic studies indicate that eggs and dietary cholesterol are not significant factors in heart disease risk (5, 6).

The problem is that although dietary cholesterol–mediated changes in total, LDL-, and HDL-cholesterol concentrations are constant relative to the dose, changes in the ratio of LDL to HDL cholesterol are a function of the actual value of each variable. For example, patient X with LDL and HDL concentrations of 3.1 mmol/L (120 mg/dL) and 1.0 mmol/L (40 mg/dL), respectively, has an LDL-HDL ratio of 3.00. Theoretically, adding an egg a day to this patient’s diet would increase LDL and HDL concentrations to 3.2 mmol/L (123.8 mg/dL) and 1.1 mmol/L (40.8 mg/dL), respectively, resulting in a ratio of total to HDL cholesterol of 3.03, similar to the ratio of 0.04 predicted by Weggemans et al. In contrast, patient Y with LDL and HDL concentrations of 4.1 mmol/L (160 mg/dL) and 1.0 mmol/L (40 mg/dL), respectively, has a ratio of 4.00. Adding an egg a day to this patient’s diet would increase the LDL concentration to 4.2 mmol/L (163.8 mg/dL), the HDL concentration to 1.1 mmol/L (40.8 mg/dL), and the ratio of LDL to HDL cholesterol by 0.01–0.04, not the predicted change of 0.04. Thus, an individual with a very low risk of myocardial infarction could, theoretically, increase their risk by 1.5%, whereas an individual with a high risk could increase their risk by 0.5%. These values represent maximal estimates because, as shown by Weggemans et al, the response of plasma LDL cholesterol to dietary cholesterol was attenuated when the ratio of polyunsaturated to saturated fatty acids (P:S) in the background diet was >0.7 [an increase of 0.04 mmol/L (1.4 mg/dL) with a diet low in saturated fat compared with an increase of 0.06 mmol/L (2.2 mg/dL) with a diet high in saturated fat with each additional increase of 100 mg cholesterol/d], whereas the response of plasma HDL cholesterol is unchanged. Under these conditions there would be no measurable change in the ratio of LDL to HDL cholesterol in our 2 hypothetical patients after adding one egg a day to their diets (ratio: 3.00–3.01 in patient X and 3.99–4.00 in patient Y).

More importantly, I am surprised at the unfortunate interpretation of Weggeman et al’s finding that egg restriction can reduce the risk of heart disease. Statistical significance and biological importance must be viewed as distinct concepts. The assertion that eating an egg a day increases the risk of heart disease by 2% (but only in those with low ratios of LDL to HDL cholesterol and a low dietary P:S) needs to be put in perspective relative to other risk factors: 1) the 72% increase in risk associated with a body mass index (in kg/m²) of 25–28.9 relative to a body mass index < 23 (7), 2) the 42% decrease in risk associated with the replacement of 5% of energy from saturated fat with 5% from unsaturated fat (8), and 3) the 51% decrease in risk associated with 1.5 h of vigorous walking 1 d/wk (9).

The fact that no studies in the past decade have reported a significant relation between either egg consumption or dietary cholesterol intakes and heart disease risk (5) is consistent with the view that the hypothesis that dietary cholesterol is a risk factor for heart disease should be dismissed. A small, statistically significant increase in the ratio of total to HDL cholesterol has little biological importance concerning heart disease risk when considered relative to those dietary and lifestyle factors that do in fact contribute to heart disease risk. Concerning the suggestion by Weggeman et al that eggs make no important contributions to the diet, I refer them to a recent supplement of the Journal of the American College of Nutrition (10) in which the merits of egg consumption are documented. In an evaluation of the relation between dietary cholesterol and the risk of heart disease, it is crucial not only have accurate estimates of risk but also a practical perspective of what a risk estimate represents.

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REFERENCES


Reply to DJ McNamara

Dear Sir:

McNamara suggests that the effect of dietary cholesterol on blood lipids is constant whatever the initial concentration. As a result, the relative effect of dietary cholesterol on total cholesterol would be lower and that on HDL would be higher in subjects with higher total cholesterol and lower HDL concentrations. This suggestion is contradicted by studies showing that the response of total cholesterol to dietary cholesterol and fats is greater in subjects with higher baseline concentrations of cholesterol (1, 2). However, this finding does not exclude the possibility that the elevated response to dietary cholesterol in subjects with high baseline cholesterol concentrations is mainly due to HDL. We therefore plotted the effect of the consumption of an extra 200 mg dietary cholesterol/d (equivalent to the cholesterol in one egg) on the ratio of total to HDL cholesterol as a function of the mean baseline cholesterol concentration in 17 studies (3). There was a tendency for the change in the ratio of total to HDL cholesterol to be lower when baseline cholesterol concentrations were higher (Figure 1): the slope of the change in the ratio as a function of baseline cholesterol was −0.046 per mmol/L (95% CI: −0.082, −0.009 mmol/L). The negative slope was caused by 2 groups of subjects with baseline total cholesterol concentrations >6 mmol/L (232 mg/dL); both groups were from the same study (4). In the 22 other groups of subjects, the effect of dietary cholesterol on the ratio was the same irrespective of the baseline total cholesterol concentration. Thus, the adverse effect of dietary cholesterol on the ratio of total to HDL cholesterol might be limited to subjects with baseline serum cholesterol concentrations <6 mmol/L. However, this finding needs to be verified in subjects with baseline cholesterol concentrations >6 mmol/L because the finding is based on the results of only one study (4).

McNamara also states that “a decade of epidemiologic studies indicates that eggs and dietary cholesterol are not significant factors in heart disease risk” and cites the results of 2 prospective cohort studies (5) to support this statement. However, he ignores the findings of 2 earlier prospective cohort studies that found a significant association between dietary cholesterol and the risk of coronary heart disease (6, 7). These observations, together with data showing that dietary cholesterol causes atherosclerosis and coronary heart disease in animals, including primates (8), should give us pause before deciding that dietary cholesterol does not affect heart disease risk in humans. Extrapolations from animal models to humans are often unreliable, but the data are too consistent and strong to be dismissed totally.

We agree that the effect of eating eggs on coronary heart disease risk, calculated from the ratio of total to HDL cholesterol, is small compared with the risk associated with being overweight. However, dietary cholesterol might increase the risk of coronary heart disease through intermediates other than serum cholesterol concentration alone. This suggestion is supported by the findings of Stampler and Shekelle (8), who analyzed the association of cholesterol intake with the risk of coronary heart disease in 4 prospective studies. Stampler and Shekelle concluded that there was an independent additional increase in risk associated with a higher dietary cholesterol intake over and above the greater risk induced by the resultant increase in serum cholesterol. Be that as it may, many people do not find it a major hardship to cut back on egg intake, whereas most people find it impossible to lose weight permanently. We fully support advice to lose weight, to replace saturated fats with unsaturated fats in the diet, and to increase exercise levels, but most people have exhausted the easy ways to achieve these goals. Meanwhile, there are small steps—dietary and other—that people can take to improve their chances of staying healthy; together, these steps may lead to an appreciable effect. Moderate egg consumption is one of these steps.

Finally, we did not say that “eggs make no important contributions to the diet.” Eggs are a useful, low-cost source of protein and certain vitamins. What we said was that “in view of the relatively small contribution of eggs to the intake of nutrients that may be beneficial in prevent-

FIGURE 1. The effect of a 200-mg/d increase in dietary cholesterol on the ratio of total cholesterol to HDL cholesterol. The 17 studies provided 24 data points. Each point is the mean of 9–70 subjects.