in LDL cholesterol compared with the AAD. Similar to earlier studies, the low-fat Step II diet was found to raise triacylglycerol compared with the higher-fat diets. HDL cholesterol differed only marginally and insignificantly between diets. The authors concluded that “a high-MUFA, cholesterol-lowering diet may be preferable to a low-fat diet” in prevention of coronary heart disease. The same opinion was expressed by other authors (2).

We would like to warn people against considering high-fat monoene diets to be the most healthy diets. There are several reasons that high-fat diets—even those based on monoene fats—should be avoided in modern societies in which physical activity and energy requirements are low. First, any high-fat diet increases the likelihood of developing obesity, as do most other highly energy-dense diets (3). Even the olive oil–consuming Greeks have become more obese during the past decades because of their increasingly sedentary lifestyle (4, 5).

Second, in the recent study by Kris-Etherton et al and in most earlier trials comparing diets with various fat contents, energy intake was fixed and constant (ie, isoenergetic conditions were maintained) to keep body weight constant. This design is inappropriate because spontaneous energy intake would normally differ if diets with different fat contents were eaten ad libitum (6). It was shown very elegantly in a meta-analysis by Kris-Etherton’s own group that fat-reduced diets cause a dose-dependent decrease in energy intake and body weight (7). The spontaneous weight loss that would be expected with a low-fat Step II diet was thus inhibited by the design used in the recent study (1). Accordingly, the blood lipid response was importantly biased. It is well known that weight loss causes triacylglycerol to decline and that concomitant increases in HDL cholesterol are often seen (8). In addition, earlier long-term trials of healthy and hyperlipemic persons showed that diets comparable with the Step II diet had no adverse effects on triacylglycerol if eaten ad libitum (9, 10).

Third, not only blood lipids but also several other cardiovascular risk factors are influenced by diet and therefore need to be considered in the overall evaluation of the health effect of a diet. We and other researchers showed that low-fat, high-fiber diets may affect blood coagulation and fibrinolysis strongly in an antithrombotic manner (11, 12). The effects on the hemostatic system seem to rely heavily on the carbohydrate quality of the diet, ie, the fiber content and the glycemic index (13). Therefore, it is unfortunate that Kris-Etherton et al did not report anything about these aspects of their experimental diets.

Fourth, there is strong epidemiologic evidence that high intakes of fruit and vegetables are associated with less coronary heart disease and cancer morbidity. High-fat diets prevent high intakes of fruit and vegetables because of the low energy ceiling of modern sedentary societies. Remember that the Greeks of the 1950s and 1960s were very physically active fishermen and farmers and that their high-fat, olive oil–based diets still allowed consumption of a large amount of bread, vegetables, and fruit (5, 14).

For these 4 reasons (more could be added), we believe it is incorrect to consider high-fat monoene diets the most healthy choice for sedentary people. We can take good care of our body weight, our blood lipids, our hemostatic system, and our need for trace elements and unknown nonnutrients present in foods only if we allow plenty of our energy to be supplied from foods with low fat contents. Where to set the fat limit is a matter of discussion, but the epidemic of obesity tells us that we still eat more fat than is appropriate. We consider a population average fat intake of ≈30% of total energy intake to be a wise recommendation.

Peter Marckmann
Arne Astrup

Research Department of Human Nutrition
Royal Veterinary and Agricultural University
Rolighedsvej 30
DK-1958 Frederiksberg
Denmark
E-mail: pma@kvl.dk

REFERENCES

Reply to P Marckmann

Dear Sir:

Numerous studies, including one we published recently (1), have shown beneficial effects of a weight-maintenance, high–monounsaturated fatty acid (MUFA), blood cholesterol-lowering...
diet compared with a high-carbohydrate, low-fat diet on important cardiovascular disease (CVD) risk factors, notably triacylglycerol, HDL-cholesterol, and plasma glucose and insulin concentrations (2, 3). It is clear that elevated triacylglycerol and glucose and insulin concentrations increase the risk of CVD, as does a low HDL-cholesterol concentration. Lowering plasma triacylglycerol, glucose, and insulin and increasing HDL cholesterol decrease the risk of CVD. Although not measured in our recent study, a weight-maintenance, high-carbohydrate, low-fat diet has also been shown to increase fibrinogen concentrations, whereas a high-MUFA, blood cholesterol–lowering diet does not (4). An elevated fibrinogen concentration was shown to increase the risk of CVD. Thus, the evidence is convincing that a weight-maintenance, high-MUFA, blood cholesterol–lowering diet beneficially affects CVD risk.

As noted by Markmann and Astrup, body weight is an important factor that must be considered in contemporary dietary recommendations. The key questions are 1) What is the best diet for weight loss and weight maintenance? and 2) Will a higher-fat diet promote weight gain?

In our recent meta-analysis (5), we observed a linear relation between decreasing percentage of energy from fat and a decrease in body weight. Although this finding suggests that higher-fat diets promote weight gain, 3 important limitations of this study must be noted. First, a higher-fat weight-loss diet was not tested, so one can question whether weight loss might be less, the same, or maybe even greater because of better adherence to the higher-fat (ie, high MUFA), energy-reduced diet. It is generally accepted that “a calorie is a calorie” regardless of whether the energy is derived from fat, carbohydrate, or protein. Thus, weight loss with any energy-reduced diet is due to the reduction in energy intake relative to expenditure. What is not clear is whether macronutrient-manipulated weight-loss diets have any effect on adherence and, hence, on long-term weight loss and weight maintenance. Second, the weight loss was small despite a large reduction in energy intake from fat (ie, 2.6-kg weight loss associated with an 8–percentage point decrease in percentage of energy from fat). A smaller decrease in fat, consistent with current intake recommendations, would be expected to result in less weight loss (only ≈1.3 kg for a 4–percentage point decrease in fat intake). Finally, in long-term studies over a period of 2–4 y, only small changes in body weight were shown (≈0.5-kg weight loss).

Targeting fat reduction as the sole means of affecting the global epidemic of overweight and obesity is not justified. Obesity is a complex problem—its causes are not fully understood. Although it is clear that an energy imbalance is the root cause, there is no compelling evidence that this is due to changes in fat intake. A case in point relates to the ongoing increase in the incidence of overweight and obesity in the United States that is concurrent with little or no change in absolute intake of fat and a decrease in percentage of energy from fat (6). It is clear that an increase in energy intake in conjunction with a more sedentary lifestyle (ie, less physical activity) accounts, in part, for the fattening of Americans. Further evidence comes from Sweden, where a small increase in overweight occurred despite virtually no change in the diet (8, 9). In addition, there is ample evidence from other countries that there is no consistent association between increasing overweight and obesity and fat intake (7, 8).

Energy balance and, if needed, weight loss in individuals who are following any diet is dependent on energy intake irrespective of the macronutrient profile. In the context of a high-MUFA, blood cholesterol–lowering diet, it is clear that this diet has beneficial effects on CVD risk factors in weight-stable individuals. Although it has not been tested as a weight-loss diet compared with a high-carbohydrate, low-fat diet in free-living subjects, such a diet could be planned that would have a low energy density (ie, high in fruit and vegetables) to provide bulk and promote satiety. The key issue for controlling energy intake may not be the macronutrient profile of the diet but rather the energy density, because the fat content and energy density of foods are not always perfectly correlated. As argued by Rolls and Bell (9), energy density is more closely related to factors such as the water and fiber contents of foods. In that study and others, subjects ate less when consuming foods of low energy density compared with foods of high energy density, regardless of fat content. Thus, focusing solely on the fat content of foods when designing weight-loss diets may limit the effectiveness of the diets if the effects of energy density are not considered.

We advocate a reexamination of the effects of the macronutrient content of the diet on risk of CVD. While acknowledging that the ultimate test of higher-fat diets will be in free-living individuals consuming foods ad libitum, we believe that by promoting the addition of fruit, vegetables, whole grains, and legumes to these diets, it may be possible to achieve long-term success in terms of both weight control and CVD risk reduction.

Penny M Kris-Etherton
Christine L Pelkman
Guixiang Zhao
Thomas A Pearson
Ying Wan
Terry D Etherton

The Pennsylvania State University
Nutrition Department
S-126 Henderson Building
University Park, PA 16802
E-mail: pmk3@psu.edu

REFERENCES
All cereals may not be equal

Dear Sir:

I wish to comment on the article by Liu et al in your September 1999 issue (1), which found an inverse relation between the prevalence of coronary heart disease and whole-grain consumption in a large prospective study of nurses. In 1988, I carried out a statistical study (2) in which the consumption of ~60 food items in 21 countries belonging to the Organization of Economic Cooperation and Development (OECD) was correlated with mortality from coronary disease. My results, regarding cereal consumption, were as follows in Table 1:

As far as correlation with coronary disease is concerned, all cereals appear to be different. A negative correlation was found only with rice consumption, and even this could be due to an indirect linkage. Coronary mortality in eastern Asia could be low on account of low fat and milk consumption but give the appearance of a negative correlation with high rice consumption. Similarly, the high positive correlation with oats consumption could be indirect. Appreciable quantities of oats are consumed only in a few countries of northern Europe. The true correlation could be between coronary disease mortality and environmental temperature. The world leaders in coronary disease mortality are Russia and its neighbors, which all have cold climates. In warmer countries, coronary mortality tends to be lower, and very low in the tropics. In my opinion, the connection between coronary disease and grain consumption must be treated with caution.

Stephen Seely
3 Truro Drive
Sale, Cheshire M33 5DF
United Kingdom

REFERENCES

Reply to S Seely

Dear Sir:

We appreciate Seely’s proposition that all cereals may not be equal in affecting risk of coronary heart disease mortality. We would like to emphasize 2 main points. First, in a prospective cohort study, we reported an inverse relation between whole-grain consumption and the incidence, not prevalence, of coronary heart disease (1). The difference between prevalence and incidence is subtle but important. Prevalence includes people with coronary heart disease at baseline who may have changed their diet after disease diagnosis. Incidence studies can provide a direct assessment of the association between diet and disease that would not be confounded by factors modified after disease diagnosis (2). Second, the study by Seely can at best suggest a hypothesis to be explored because of important limitations inherent in any such international correlation or ecologic study, including the crude assessment of diet, lack of information on individual diets, and the lack of adjustment for confounding factors including genetic predisposition, environment, and lifestyle practices (3). Seely noted only a few of the many differences between these countries. Large prospective cohort studies with detailed dietary assessment, long-term follow-up, and careful control of multiple confounding factors are better suited to examine diet-disease associations.

Simin Liu
JoAnn E Manson
Walter C Willett
Brigham and Women’s Hospital
Division of Preventive Medicine
900 Commonwealth Avenue East
Boston, MA 02215-1204

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