Validation of dietary patterns assessed with a food-frequency questionnaire

Dear Sir:

In their recent article, Hu et al (1) identified dietary patterns using factor-analyzed data from food-frequency questionnaires (FFQs) and they assessed the validity of these patterns in part by examining their association with patterns identified by using factor-analyzed dietary-record data. Their use of dietary-record data to assess the validity of dietary patterns requires comment. Specifically, although dietary records are an acceptable gold standard for validating FFQs for measuring food or nutrient intake, the same strategy does not provide an equivalent validation of dietary patterns. To the extent that dietary records provide more accurate estimates of food intake, they also allow more precise estimation of factor scores than do FFQs. By conducting a factor analysis to identify dietary patterns, however, Hu et al essentially created two factor–analytically derived scales to measure intakes from Western and prudent dietary patterns, with each food or food group representing one (differentially weighted) item in each scale. Validation of FFQ-based dietary patterns against dietary-record-based patterns with use of scales derived from factor analysis based on the same food items is comparable with validation of a scale against the same scale with individual items measured more accurately. In essence, the validation strategy presumes that the item-level data are valid and uses these data rather than an independent indicator of each food pattern.

The ability to assess the validity of dietary patterns measured by factor analysis is limited by our understanding of what dietary patterns actually represent. Nutritional anthropologists have researched numerous dimensions of intake patterns—how foods are organized into dishes and dishes into meals, which foods are integral to the meal, and even the time, place, and context in which meals are eaten (2). Measuring patterns by using factor-analyzed FFQ responses assumes that patterns can be characterized adequately by food-intake frequencies and their intercorrelations. Although this method may capture enough variation in eating habits to render measurement of other dimensions unnecessary, examining dimensions of dietary patterns other than with the use of food-frequency data may provide valuable additional information in some instances. For example, effects on iron bioavailability of concurrent consumption of meats as absorption enhancers or phytates as absorption inhibitors (3) illustrate the potential importance of considering the organization of foods into meals. Whether scales derived from factor analysis based on food frequencies alone are acceptably valid measures of actual dietary patterns, therefore, remains to be evaluated. Identifying a more appropriate gold standard for validation will require a more complete conception of what the Western and prudent dietary patterns actually are. Indeed, the greater challenge may be to gain a more complete a priori understanding of dietary patterns before trying to measure them, thus raising the possibility of measuring dietary patterns directly rather than relying on ad hoc interpretations of dietary data.

Hu et al’s analysis does provide evidence of food groupings that might have been anticipated a priori. The finding of similar patterns across methods also provides evidence of the reproducibility of their approach. As such, their evaluation showed that FFQs can be a useful and convenient source of dietary data for measuring dietary patterns, even though they were not originally intended for dietary-pattern measurement.

Marilyn Tseng

Population Science Division
Fox Chase Cancer Center
7701 Burholme Avenue
Philadelphia, PA 19111
E-mail: m_tseng@fccc.edu

REFERENCES

Reply to M Tseng

Dear Sir:

We thank Tseng for her interest in our paper on validation studies of dietary patterns assessed with a food-frequency questionnaire (1). We agree that there is no gold standard for assessing dietary patterns. Nevertheless, the consistency of major dietary patterns assessed with food-frequency questionnaires and multiple, weekly dietary records suggests the usefulness of factor-analytic approaches for assessing dietary patterns. More importantly, dietary patterns were reasonably correlated with plasma biochemical measures of cardiovascular disease and nutrient intakes, further suggesting the validity of the method. The ultimate test of validity, however, lies in whether dietary patterns can independently predict disease outcomes. Analyses are underway to examine the relation between major dietary patterns assessed with food-frequency questionnaires and the incidence of cardiovascular disease.

Frank B Hu
Walter C Willett

Department of Nutrition
Harvard School of Public Health
665 Huntington Avenue
Boston, MA 02115

REFERENCE
Dear Sir:

Dreon et al (1) concluded that “There is no apparent lipoprotein benefit of reduction in dietary fat from 20–24% to 10% in men with large LDL particles…”. They also suggest that switching from an average American diet to a very-low-fat diet “in a subset of men who convert to phenotype B, [is] suggestive of an increase in coronary disease risk.” However, Ornish et al (2) observed regression of atherosclerosis in subjects consuming a very-low-fat (VLF) diet even though their serum triacylglycerol concentrations increased and their HDL-cholesterol concentrations decreased (lipoprotein changes often associated with an increase in small dense LDL particles, also known as phenotype B or pattern B). In contrast, Ornish et al (2) observed the progression of atherosclerosis in subjects consuming a more moderate-fat diet, even in those subjects who were taking cholesterol-lowering drugs. Many years ago, Morrison (3) showed a dramatic reduction in both cardiovascular disease and in all-cause mortality in subjects consuming a VLF diet compared with those consuming an average American diet. We know of no comparable clinical trials showing that diets with ≥20–30% of energy as fat lead to regression of atherosclerosis, a reduction in all-cause mortality, or both. Should suggestive evidence from Dreon et al’s short-term trial outweigh evidence from these much longer clinical trials with harder endpoints (eg, overall mortality and cardiovascular disease mortality)?

There is little doubt that pattern B is associated with an increased risk of atherosclerosis in people who eat a moderate- to high-fat diet. But what is the evidence that a change in LDL status from pattern A to pattern B as a result of restricting dietary fat promotes atherosclerosis?

A low HDL-cholesterol concentration is associated with an increased risk of ischemic heart disease (IHD), and restriction of dietary fat generally leads to a drop in HDL cholesterol. However, in countries where VLF diets are the norm, the incidence of IHD is much lower than in the United States, despite significantly lower HDL-cholesterol concentrations. In hamsters, it was shown that reverse cholesterol transport is not impaired by fat restriction despite a nearly 50% reduction in HDL (4). Perhaps there are other metabolic changes associated with the change to pattern B when fat is restricted that reduce the risk of atherogenesis when a VLF diet is consumed? For example, Parks et al (5) showed a significant reduction in the susceptibility of LDL to oxidation in subjects consuming a diet containing 10% of energy as fat (10%-fat diet) even though there was little change in LDL particle size.

In addition, it would be incorrect to generalize from Dreon et al’s results and conclude that all VLF diets inevitably lead to an increased number of small, dense LDL particles (pattern B). Indeed, at the Pritikin Center we found that the LDL status of 6 of 22 subjects actually changed from pattern B to pattern A (a predominance of large LDL particles) while consuming a VLF diet, which is the exact opposite of the trend observed by Dreon et al (6). There are several possible factors that may have contributed to the opposite trends observed during the 2 diets, even though both provided ≈10% of energy as fat. One reason we saw a trend away from pattern B at the Pritikin Center is exercise.

Exercise tends to raise HDLs and lower triacylglycerols and thus may also reduce the predominance of small, dense LDL particles. However, there are 3 possible differences between Dreon et al’s 10%-fat diet and the 10%-fat diet we used or in the way the diets were fed that might explain the opposite trends observed.

1) Dreon et al’s higher-fat diet actually had 50% more fiber than their 10%-fat diet, but our 10%-fat diet had much more fiber than either of Dreon et al’s diets. Because dietary fiber improves blood lipids (7) and most high-fat foods are low in fiber, it seems odd that the higher-fat diet of Dreon et al would contain more fiber than their 10%-fat diet. The unusually low-fiber content of Dreon et al’s VLF diet would also have biased their results against the VLF diet.

2) Those who advocate a VLF diet to treat and prevent IHD generally recommend a high fiber intake and a reduction in dietary cholesterol and animal protein. However, Dreon et al’s VLF diet not only had less fiber but also had at least as much cholesterol (and presumably animal protein) as their more moderate-fat diet. Again, these differences would tend to reduce the efficacy of Dreon et al’s VLF diet for treating dyslipidemia compared with the kind of VLF diets typically advocated for the treatment and prevention of IHD.

3) Finally, those who advocate VLF diets also generally recommend that they be consumed ad libitum. As Dreon et al noted, “…the tendency for ad libitum consumption of low-fat diets to promote weight loss need[s] to be considered.” Why was it not considered in their experimental design? This is important because extra energy and an increased body weight increase the size of small, dense LDL particles (8). Indeed, Dreon et al’s data showed that their subjects actually consumed 14% more energy with the VLF diet than with their usual diet. Would not the extra 1548 kJ/d (370 kcal/d) provided by Dreon et al’s VLF diet than by their moderate-fat diet have caused more adverse effects on blood lipids and again biased the results against the VLF diet? It has been shown that the presumably adverse metabolic effects on blood lipids associated with VLF diets compared with higher-fat diets largely disappear when both diets are fed ad libitum rather than isocalorically (9). Few people adhere long term to diets with a prescribed energy level that differs significantly from what their appetite demands (10), which makes the results of short-term studies with a controlled energy intake of limited clinical value.

If the point of Dreon et al’s study is that a VLF, energy-dense, low-fiber diet consisting largely of refined sugars and white flour is of questionable value for many, if not most, normolipidemic individuals, we agree. However, if Dreon et al believe that their data show that a more vegetarian, high-fiber, VLF diet is likely to increase atherogenesis and IHD in normolipidemic individuals relative to an average American diet, we disagree. The peculiar nature of the VLF diet used in Dreon et al’s study coupled with the fact that it provided a higher energy intake than the subjects’ usual diet make it inappropriate to suggest or imply that all VLF diets promote pattern B, an increased risk of IHD, or both.

James J Kenney

Pritikin Longevity Center
2811 Wilshire Boulevard, Suite 410
Santa Monica, CA 90403

1 Dreon et al (1) concluded that “There is no apparent lipoprotein benefit of reduction in dietary fat from 20–24% to 10% in men with large LDL particles…” They also suggest that switching from an average American diet to a very-low-fat diet “in a subset of men who convert to phenotype B, [is] suggestive of an increase in coronary disease risk.” However, Ornish et al (2) observed regression of atherosclerosis in subjects consuming a very-low-fat (VLF) diet even though their serum triacylglycerol concentrations increased and their HDL-cholesterol concentrations decreased (lipoprotein changes often associated with an increase in small dense LDL particles, also known as phenotype B or pattern B). In contrast, Ornish et al (2) observed the progression of atherosclerosis in subjects consuming a more moderate-fat diet, even in those subjects who were taking cholesterol-lowering drugs. Many years ago, Morrison (3) showed a dramatic reduction in both cardiovascular disease and in all-cause mortality in subjects consuming a VLF diet compared with those consuming an average American diet. We know of no comparable clinical trials showing that diets with ≥20–30% of energy as fat lead to regression of atherosclerosis, a reduction in all-cause mortality, or both. Should suggestive evidence from Dreon et al’s short-term trial outweigh evidence from these much longer clinical trials with harder endpoints (eg, overall mortality and cardiovascular disease mortality)?

There is little doubt that pattern B is associated with an increased risk of atherosclerosis in people who eat a moderate- to high-fat diet. But what is the evidence that a change in LDL status from pattern A to pattern B as a result of restricting dietary fat promotes atherosclerosis?

A low HDL-cholesterol concentration is associated with an increased risk of ischemic heart disease (IHD), and restriction of dietary fat generally leads to a drop in HDL cholesterol. However, in countries where VLF diets are the norm, the incidence of IHD is much lower than in the United States, despite significantly lower HDL-cholesterol concentrations. In hamsters, it was shown that reverse cholesterol transport is not impaired by fat restriction despite a nearly 50% reduction in HDL (4). Perhaps there are other metabolic changes associated with the change to pattern B when fat is restricted that reduce the risk of atherogenesis when a VLF diet is consumed? For example, Parks et al (5) showed a significant reduction in the susceptibility of LDL to oxidation in subjects consuming a diet containing 10% of energy as fat (10%-fat diet) even though there was little change in LDL particle size.

In addition, it would be incorrect to generalize from Dreon et al’s results and conclude that all VLF diets inevitably lead to an increased number of small, dense LDL particles (pattern B). Indeed, at the Pritikin Center we found that the LDL status of 6 of 22 subjects actually changed from pattern B to pattern A (a predominance of large LDL particles) while consuming a VLF diet, which is the exact opposite of the trend observed by Dreon et al (6). There are several possible factors that may have contributed to the opposite trends observed during the 2 diets, even though both provided ≈10% of energy as fat. One reason we saw a trend away from pattern B at the Pritikin Center is exercise.

Exercise tends to raise HDLs and lower triacylglycerols and thus may also reduce the predominance of small, dense LDL particles. However, there are 3 possible differences between Dreon et al’s 10%-fat diet and the 10%-fat diet we used or in the way the diets were fed that might explain the opposite trends observed.

1) Dreon et al’s higher-fat diet actually had 50% more fiber than their 10%-fat diet, but our 10%-fat diet had much more fiber than either of Dreon et al’s diets. Because dietary fiber improves blood lipids (7) and most high-fat foods are low in fiber, it seems odd that the higher-fat diet of Dreon et al would contain more fiber than their 10%-fat diet. The unusually low-fiber content of Dreon et al’s VLF diet would also have biased their results against the VLF diet.

2) Those who advocate a VLF diet to treat and prevent IHD generally recommend a high fiber intake and a reduction in dietary cholesterol and animal protein. However, Dreon et al’s VLF diet not only had less fiber but also had at least as much cholesterol (and presumably animal protein) as their more moderate-fat diet. Again, these differences would tend to reduce the efficacy of Dreon et al’s VLF diet for treating dyslipidemia compared with the kind of VLF diets typically advocated for the treatment and prevention of IHD.

3) Finally, those who advocate VLF diets also generally recommend that they be consumed ad libitum. As Dreon et al noted, “…the tendency for ad libitum consumption of low-fat diets to promote weight loss need[s] to be considered.” Why was it not considered in their experimental design? This is important because extra energy and an increased body weight increase the size of small, dense LDL particles (8). Indeed, Dreon et al’s data showed that their subjects actually consumed 14% more energy with the VLF diet than with their usual diet. Would not the extra 1548 kJ/d (370 kcal/d) provided by Dreon et al’s VLF diet than by their moderate-fat diet have caused more adverse effects on blood lipids and again biased the results against the VLF diet? It has been shown that the presumably adverse metabolic effects on blood lipids associated with VLF diets compared with higher-fat diets largely disappear when both diets are fed ad libitum rather than isocalorically (9). Few people adhere long term to diets with a prescribed energy level that differs significantly from what their appetite demands (10), which makes the results of short-term studies with a controlled energy intake of limited clinical value.

If the point of Dreon et al’s study is that a VLF, energy-dense, low-fiber diet consisting largely of refined sugars and white flour is of questionable value for many, if not most, normolipidemic individuals, we agree. However, if Dreon et al believe that their data show that a more vegetarian, high-fiber, VLF diet is likely to increase atherogenesis and IHD in normolipidemic individuals relative to an average American diet, we disagree. The peculiar nature of the VLF diet used in Dreon et al’s study coupled with the fact that it provided a higher energy intake than the subjects’ usual diet make it inappropriate to suggest or imply that all VLF diets promote pattern B, an increased risk of IHD, or both.