Why cutting fats may harm the heart

Since dietary fats can play a role in boosting blood-cholesterol concentrations and heart-disease risk, does it make sense to short-circuit the process by dramatically paring fats from the diet?

For most men, at least, the answer may prove an emphatic no, according to new research conducted at Lawrence Berkeley (Calif.) National Laboratory (LBNL).

In most people, genetic programming directs the body to respond to drops in total dietary fat by repackaging fatty particles called lipids, the study shows. In general, these changes tend to increase risk of cardiovascular disease, notes Ronald M. Krauss, who led the research.

In a pair of earlier studies, his team directed 238 healthy men to spend periods of 3 to 6 weeks eating each of two diets: one deriving at least 40 percent of its calories from fats, and the other with just 20 to 24 percent fat.

While on the high-fat diet, one-quarter of the volunteers exhibited a worrisome blood- lipid profile. Their cholesterol-shuttling, low-density lipoproteins (LDLs)—the so-called bad lipoproteins—were unusually small and dense. Such small LDLs have been linked to an especially high risk of heart disease (SN: 9/21/96, p. 182).

Even more troubling, when the men with normal-size LDLs ate the low-fat diet, a third of them began making the small, dense LDLs. With this change came an increase in blood concentrations of triglycerides, another heart-disease high-risk factor, and a drop in the cholesterol exiting their blood via high-density lipoproteins (HDLs), the good lipoproteins.

In the most recent work, the LBNL researchers selected 38 men who had proven resistant to lipid changes in the previous studies and put them on an even lower-fat diet. For 10 days, they ate menus deriving just 10 percent of their calories from fat. Once again, a third began making the especially small LDLs, Krauss's group reports in the March AMERICAN JOURNAL OF CLINICAL NUTRITION.

These men also increased their triglycerides and decreased HDLs—especially HDL_{2B}, which is considered especially beneficial.

In contrast to the earlier two studies, the men whose lipid profiles changed in this test also exhibited an increase in blood concentrations of intermediate-density lipoproteins (IDLs). Though most physicians don't measure IDLs, studies that assayed them generally found high levels "to be the indicator most strongly connected to heart-disease risk," Krauss notes. "It trumps all other standard [lipid] measurements in predicting risk."

Overall, the LBNL data indicate that at least two-thirds of men carry genes for these lipid disturbances, though in most cases the effects only show up on a low-fat diet. Krauss suspects that the benefits of exercise and low calorie intake may compensate for the increased risk imparted by the adverse lipid profile.

The data may underestimate the genetic predisposition for lipid change, Krauss contends, since all the volunteers were of normal weight. With obesity, he says, "there's an even higher prevalence of these lipid disturbances to start with."

In fact, he worries that the overweight couch potato—precisely the person physicians often target for a low-fat diet—may face the greatest risk of expressing these lipid disturbances. Certainly, Krauss says, his data argue strongly against a one-size-fits-all approach to advice on fat consumption.

These data "support what a number of us in the nutrition community have been saying—that low-fat diets are not the way to go [to prevent heart disease]," says Meir J. Stampfer of the Harvard School of Public Health in Boston.

"The nutrition community has pushed this notion that fat equals bad," Stampfer says, "without making distinctions between types of fat." In fact, he explains, because it's saturated fats and *trans* fats (SN: 5/21/94, p. 325) that are bad for the heart, "our message to the public should be to replace them with polyunsaturated and monounsaturated fats" —such as the types in nuts (SN: 11/21/98, p. 328). —*J. Raloff*

DNA data yield new human-origins view

The genetic story of human evolution has taken a surprising turn. Anatomically modern *Homo sapiens* sprang from a population of so-called archaic *H. sapiens* that lived in Africa around 200,000 years ago, a new DNA investigation suggests. That ancestral group then split into African and Asian branches, which interbred to some extent as they evolved into today's humans.

This scenario differs from the two theories of human evolution that have been vying for dominance. Out-of-Africa supporters hold that a single African population of *H. sapiens* emerged between 100,000 and 200,000 years ago and then spread elsewhere. In contrast, multiregional enthusiasts argue that, starting as early as 2 million years ago, human groups in Africa, Europe, and Asia interbred enough to evolve collectively into modern *H. sapiens*.

"Our data fall between the predictions of current human-origins theories," says geneticist Jody Hey of Rutgers University in Piscataway, N.J., who conducted the new study with Rutgers anthropologist Eugene E. Harris. "Researchers may need to rethink their inferences about human evolution."

Harris and Hey examined a nucleotide sequence on the X chromosome of 16 African and 19 non-African men, as well as 2 chimpanzees. The sequence is part of a gene that regulates the chemical breakdown of glucose in the body.

Samples from the African and non-African groups displayed marked genetic differences, the scientists report in the March 16 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. The variation exceeds that typically seen at other chromosomal sites when populations are compared.

One particular nucleotide sequence occurred in all the Asian men and none of their African counterparts. Comparison of the chimp data with the human allowed the researchers to estimate that this mutation, marking the evolutionary split between the groups, arose about 200,000 years ago.

The oldest sequences, which occurred in the African group, originated approximately 1.9 million years ago, Harris and Hey contend.

The X-chromosome sequence varied greatly among the Africans but little among the Asians. This pattern suggests that natural selection affected this particular gene differently in the two groups, according to Hey.

Since modern *H. sapiens* fossils date at most to 130,000 years ago, the X-chromosome findings support the notion that an early form of *H. sapiens* split into separate African populations around 200,000 years ago, Hey says. One group then migrated into Asia, he theorizes, although its members probably didn't trek far into that continent.

A modest amount of interbreeding between African and Asian populations then would have fostered the concurrent evolution of modern human traits, in Hey's view.

"These new DNA data show surprisingly ancient separations in our species," comments anthropologist Henry C. Harpending of the University of Utah in Salt Lake City. The results contradict mitochondrial DNA studies, including some coauthored by Harpending, which place modern humanity's roots in a single African population that greatly expanded its numbers around 50,000 years ago as it spread to other regions.

Suspicions have arisen that natural selection can rapidly reshape mitochondrial DNA structure, casting doubt on the ability of that genetic material to clarify the time and place of modern human origins (SN: 2/6/99, p. 88).

—*B. Bower*