Oxidized lipids: A key to heart disease?

New research suggests it's the quality of cholesterol in the diet, not the quantity, that contributes most to one's risk of developing coronary heart disease and atherosclerosis (hardening of the arteries). Conventional wisdom has suggested that diets rich in lipids—a family of biological compounds including cholesterol, fatty acids, phospholipids and triglycerides (fat)—could play a major role in the development of human heart disease. But research presented April 29 in Miami at the American Chemical Society's 189th national meeting now suggests that those lipids might present a serious hazard only if and when they are transformed through oxidation.

Oxidation is any chemical reaction involving the transfer of electrons from one substance to another. Oxidizers' ability to grab electrons away from other substances makes them highly reactive chemically.

Researchers from the University of Illinois in Urbana and elsewhere report that lipids in food are likely to oxidize in a number of situations, including: when cooked meats are stored for an appreciable length of time before eating; when prepared foods—such as dehydrated eggs in cake mixes — are processed at extremely high temperatures; and when foods are cooked in recycled oil that has been heated for a long time.

"All lipids are unstable and will eventually oxidize," explains U. of Illinois food chemist Fred A. Kummerow. Cooking just speeds the process (SN: 2/9/85, p. 88). In one test, Kummerow and his colleagues found that chicken meat began accumulating oxidized lipids within 24 hours of cooking—even though the meat had been refrigerated immediately after cooking.

The researchers also showed that oxidized lipids are toxic to arterial cells. In one in vitro test, both partially oxidized cholesterol and partially oxidized vitamin D (also a lipid) decreased these cells' ability to keep out calcium. Not only will too much calcium kill cells, but calcium deposits also characterize advanced atherosclerotic lesions.

In another test, the Illinois team counted the number of dead cells in smooth muscle from the abdominal aorta of chickens that had been fed diets high in either cholesterol or one of its oxidation products, 7-ketocholesterol. In four- and eight-week trials, roughly 2 percent of these cells were dead in chickens that had been fed a diet containing 1 percent cholesterol—about twice the rate of cell death in chicken fed no such lipid. In chickens that received the 7-ketocholesterol, almost 10 percent of the cells were dead after a four-week trial, and 22 percent in animals fed the diet for eight weeks.

Kummerow also cites work by others showing that low-density lipoproteins, protein-cholesterol agglomerations, would not enter arterial cells—a step believed necessary to deposit the lipids that help form atherosclerotic lesions — until those lipoproteins had been modified by malonaldehyde, an indicator of lipid oxidation.

Paul B. Addis of the department of food science and nutrition at the University of Minnesota in St. Paul and his colleagues report that they have recently developed the first single gas-chromatography test that will allow detection of any lipid oxide that might be present in food. He suggests that screening tests focus on 7-ketocholesterol as a flag of lipid oxidation.

While the researchers offer no formal dietary recommendations at this point, several did suggest that consumers trim the fat from the edges of meat, be aware of processed eggs and other such foods precooked at very high temperatures and store meat—particularly cooked meat—for as short a time as possible before eating.

Cholesterol oxides were implicated in more than just heart disease. Addis notes that they have also been shown to inflame arteries and other tissues. And, he adds, the current view of atherosclerosis suggests that it develops, much like cancer, in a two-step process. Once atherosclerosis is initiated, high blood levels of cholesterol may be able to fuel its continued development, he says. But even here, he cautions against indicting normal dietary cholesterol. Where there may be a link, he says, is between dietary consumption of saturated fats and blood levels of cholesterol.

The body has developed a sophisticated antioxidant defense system. Kummerow and Addis say it's not clear whether lipid oxidants overwhelm that defense in certain individuals or attack via unprotected pathways. —*J. Raloff*

Was it a comet, or merely the solar wind?

For nearly two years, Christopher Russell of the University of California at Los Angeles has been pursuing the possibility that a Venus-orbiting spacecraft in 1982 detected the invisible signs of the passage of an otherwise unknown comet. The case got even stranger last year, when a study of similar events suggested that at least some of them might indeed be due to a known object — but the object, named 2201 Oljato, was one that had previously shown almost no signs of being a comet. Perhaps, it was inferred, Oljato might be an old cometary nucleus that retained only enough of its ices to "outgas" a little in the sun's heat and produce the traces recorded by spacecraft, but not enough to form a more active comet's familiar, fuzzy "coma" and tail.

Now Devri S. Intriligator of the Carmel Research Center in Santa Monica, Calif., has concluded that the "original" event, seen in data from the Pioneer Venus orbiter, was not due to a comet at all, and was simply an example of the well-known variable behavior of the solar wind.

Intriligator notes that around the time of the original event, the orbiter showed a marked increase in the number of helium ions, compared with the number of hydrogen ions, in the interplanetary medium. And helium ions almost surely come from the sun, not from comets. In addition, she reports in the April Geophysical Research Letters, there was a disturbance in the interplanetary magnetic field of just the sort that would be produced by a trailing "filament" of the solar wind blowing past the spacecraft. Furthermore, she says, both the increased helium and the magnetic-field disturbance were also detected by another spacecraft (ISEE-3), which was about 40 percent farther from the sun.

But the case is far from closed. Russell agrees that helium ions are not likely to come from comets, and that the fickle behavior of the solar wind is capable of producing magnetic-field disturbances that will show up at widely separated spacecraft. However, he says, a detailed examination of the magnetic-field data from the Pioneer Venus orbiter shows a "signature" that would be difficult to explain without putting some sort of obstacle in the way—like a comet or a cloud of ionized atoms that have "outgassed" from it. The magnetic-field lines, he says, appear bunched up around the center of the disturbance, becoming more separated off to the sides, as though the field lines borne by the solar wind had run into something and were on their way to wrapping around it. The same "wraparound" effect is what yields the magnetic "tails" of planets such as earth and Jupiter. The solar wind, he says, could have accounted for the helium, and magnetic-field disturbances are far from rare. But no matter what the solar wind was doing at the time, the particular bunching-up or "compression" of the field lines looks too much as though something else was also present — even if no earth-based astronomers saw it.

Still, says Intriligator, compressions do happen in the solar-wind field, triggered, for example, by blobs of escaping solar "plasma" or by the interaction of separate streams of the solar wind.

So the possibility of the unseen comet remains a mystery. More insight may come, however, when the ISEE-3 spacecraft (now renamed ICE) flies through the tail of Comet Giacobini-Zinner this September, and six months later, when several probes pass Comet Halley. —*J. Eberhart*