Consumers Thwarted in Fight on Fat?

Concerned by the link between heart disease and high levels of cholesterol in the blood, U.S. consumers have made healthy strides in trying to purge cholesterol and unnecessary fat from their diet. But those attempts have been seriously hampered, not only by a lack of good nutritional information and labeling, but also by counterproductive government regulations, according to a National Research Council (NRC) report released this week. The unintended result, the report says, is that while many consumers have decreased consumption of visible fat, they simultaneously have increased consumption of invisible fats — those in prepared foods (like salad dressings), in baked goods, in fast foods and in partially prepared convenience foods.

As a result, consumers over the past 20 years have shaved fat's share in the U.S. diet by only about 4 percent, according to Scott M. Grundy, director of the Center for Human Nutrition at the University of Texas Health Science Center in Dallas and one of the report's authors. That still leaves them consuming too much fat — about 6 percent more than is recommended by the American Heart Association (AHA) and other groups (SN: 3/26/88, p.203). Perhaps more importantly, data in the report indicate that 90 percent of adult women and 96 percent of children eat more saturated fats — the type posing the greatest cardiovascular risk — than the AHA recommends. Though comparable data on men are not yet available, the report says they are expected to follow a similar trend.

According to the NRC panel authoring the report, the "real solution" to lowering dietary consumption of fat and cholesterol should not be avoiding red meat or trimming the fat off the meat — two common approaches — but instead producing leaner animals and providing better nutritional information for consumers. The panel recommends changing several federal regulations to help achieve this.

For example, current meat-grading policies actually "encourage producers to overfatten their cattle," says panel chairman David L. Call, dean of Cornell University's College of Agriculture and Life Sciences. Originally, this system was based on the principle that meat well marbled with intramuscular fat is juicier and more tender. But, Call says, "recent research, some of it published in this report for the first time, has demonstrated that differences in palatability between lean and fatty cuts of beef are less than one might expect." He says this argues for the need for a new grading scheme.

Panel member Gary Smith, a Texas A&M meat scientist, points to another problem: government regulations prohibiting the grading of carcasses that are trimmed of fat immediately after slaughter. If regulations required that meat producers be paid only for lean muscle, he says, less fat would reach the consumer and producers would have an incentive to grow leaner animals.

Today, low-sodium cheese can't be labeled as "cheese" or low-fat mayonnaise as "mayonnaise" because of what panel member Timothy Hammonds views as antiquated Food and Drug Administration (FDA) regulations. Hammonds, senior vice-president of the Washington, D.C.-based Food Marketing Institute, says that because such restrictions tend to confuse rather than inform a health-conscious consumer, they should be revised. The panel also recommends changing what it sees as a counterproductive FDA rule that prohibits labeling of foods as low-sodium or low-fat unless all other nutrient information is included.

Finally, the report notes that during the past 20 years there has been a steady increase of fat in U.S. food supplies. But data now show that the increase is due almost entirely to certain vegetable fats — many of them (including palm and coconut oils) as highly saturated as animal fats.

This, along with the panel's citing of new research showing that not all saturated fats elevate cholesterol, pleased Eric Hentges, assistant research director for the National Live Stock and Meat Board in Chicago. In fact, Hentges says, the beef industry endorses both the report and its recommendations.

— J. Raloff

Tissue transplant boosts memory in rats

Memory problems caused by long-term alcohol use in rats can be mostly reversed with transplants of brain tissue rich in a neurotransmitter already implicated in the memory deficits of Alzheimer's disease, according to a report in the March 30 NATURE.

Similar transplants eventually may help humans whose memory is muddled from years of heavy drinking or as a result of Alzheimer's disease, say Thomas Arendt and his colleagues of the Institute of Psychiatry in London, England. Although it is unclear if the rats provide a model for any specific type of memory disturbance in alcoholics, "the rats' memory problems were produced in much the same way as human alcoholics develop severe memory problems," says neuropsychologist and study participant Jeffrey A. Gray.

The investigators provided 78 male rats with an alcohol solution as the only fluid in an otherwise normal diet for 28 weeks. Another 16 rats drank only water with their food during the same period. Four weeks after the alcohol-treated rats returned to water, they made many more errors on memory tests in which they had to remember which four arms of an eight-spoked maze contained sugar pellets.

But about nine weeks after receiving transplants of fetal rat brain tissue to two brain areas — the outer layer of the frontal part of the brain and an inner structure concerned with memory function known as the hippocampus — 12 alcohol-treated rats performed nearly as well as water-only rats on maze trials. The fetal tissue contained large amounts of acetylcholine, a chemical that helps transmit information from one brain cell to another.

"The transplants must act on general memory rehearsal or retrieval mechanisms in the brain," says Gray, since the animals recovered the ability to use visual, tactile and spatial orientation cues, and avoided both unbaited arms and those from which a sugar pellet had already been taken.

Alcohol-treated rats given tissue transplants with acetylcholine to only one of the brain areas showed minimal memory recovery. Rats that received tissue with the neurotransmitter noradrenaline, but not acetylcholine, in one or both brain areas did not improve on maze trials.

Acetylcholine and its chemical precursors have been implicated in the memory loss associated with diseases of aging, such as Alzheimer's disease, says Gray, but the clinical effectiveness of drugs that alter acetylcholine levels has not been clearly demonstrated.

The persistent memory deficits of alcohol-treated animals mimic the irreversible memory loss seen in chronic alcoholics who develop Korsakoff's psychosis, says Gray. The rats' brains, however, do not show damage typical of the Korsakoff brain. "This evidence questions whether the brain damage observed in humans with Korsakoff's psychosis is related to their memory problems," notes Gray. *— B. Bower*