Fish oil slows some developing cancers

While scientists know little about what causes pancreatic cancer, they have a strong hunch that high-fat diets are a major risk factor for this disease, the fifth-leading cancer killer in the United States. New animal data forcefully support that hunch and suggest that adding a significant amount of fish oil to the diet can slow critical stages in the development of this and other cancers.

Several years ago, researchers from Cornell University in Ithaca, N.Y., and Dartmouth Medical School in Hanover, N.H., induced the development of precancerous tumor nodules by injecting two-week-old rats with azaserine — a potent pancreatic carcinogen. After four months on diets containing 20 percent corn oil (by weight), the rats showed a proliferation of growing precancerous lesions. Other rats on diets containing 20 percent menhaden (fish) oil developed only about one-third as many lesions.

Though the fat level in these diets was high—about 45 percent of the calories—it was only 18 percent higher than the level consumed by the average U.S. adult. By lowering the fat in the rats' diets after tumors had begun to develop, the researchers slowed the growth of the tumors, says T. Colin Campbell of Cornell, a nutritional biochemist and coauthor of the study.

In their newest study, described in the June 7 JOURNAL OF THE NATIONAL CANCER INSTITUTE, the same researchers showed that rats started on 20 percent fish oil but switched to corn oil midway through the experiment were hardly better off at the end of four months than those who ate 20 percent corn oil throughout the study. In contrast, rats started on corn oil but switched to fish oil two months later reaped virtually the same benefits in reduced precancer development as those dining on fish oil only. Campbell says these data raise an important question: Would similar benefits result if fish oil were given after the lesions had developed into true cancers?

Recent biochemical data suggest the answer is yes. Working with two types of cancers, human fibrosarcoma and a mouse melanoma, "we are showing that using different [dietary fats], you can affect the progression of a cancer," says Reuven Reich, a biochemist with the National Institute of Dental Research in Bethesda, Md.

The body converts linoleic acid — an essential fatty acid that makes up 60 percent of corn oil — into arachidonic acid. Fish oils contain scant linoleic or arachidonic acid but are rich in eicosapentanoic acid. The only difference between arachidonic and eicosapentanoic acid, explains Reich, is that the former has four double bonds and the latter has five. In fact, the same enzymes metabolize both. However, he and his colleagues have recently shown that, given a choice between the two fatty acids, enzymes in mammalian cells preferentially metabolize the eicosapentanoic acid in fish oils.

In the April 28 BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, Reich and his colleagues present data showing why that's apparently beneficial. Arachidonic acid's metabolites are at least 100 times more biologically active than those of eicosapentanoic acid, they report, and they have demonstrated in mice that this activity relates to the metabolites' ability to foster metastasis — the spawning of new tumors far from the initial cancer.

Arachidonic acid's metabolites probably promote metastasis, Reich's data suggest, by suppressing the body's natural killer cells or by promoting the activity of cancer-cell enzymes that can cut through the connective tissue that would otherwise confine a malignancy (SN: 4/15/89, p.228).

"There's no doubt about it; something about fish oil puts it in a separate category from the average oil," says Leonard Cohen at the American Health Foundation in Valhalla, N.Y. He says that's why he and other cancer researchers are increasingly being drawn to it. However, his data also indicate that "you have to have a hefty amount in the diet before you see an [anticancer] effect." — *J. Raloff*

Looking for Lyme in the nervous system

Physicians have suspected Lyme disease as the culprit behind an assortment of central nervous system (CNS) ailments. Some of these illnesses, such as meningitis, are known to result from infection with the Lyme-causing bacterium, but others may be coincidental. Now, for the first time in North America, researchers have demonstrated that analysis of spinal fluid can provide "a fairly straightforward way to tell whether someone's CNS symptoms are due to Lyme infection," says study leader John J. Halperin, a neurologist at the State University of New York at Stony Brook.

The study, involving 85 patients with antibodies to the bacterium in their blood, offers the first direct evidence that active Lyme-causing bacteria in the central nervous system can trigger a brain disorder, or encephalopathy, that results in the cognitive and memory deficits observed in many Lyme patients. The researchers found that many study subjects diagnosed with encephalopathy harbored the antibodies in their spinal fluid as well as in their blood. These patients' Lyme disease symptoms improved substantially after antibiotic treatment, the team reports in the June NEUROLOGY.

The new results also strongly suggest that Lyme disease does not cause multiple sclerosis, a finding corroborated by Patricia K. Coyle, also at Stony Brook, in a separate study described in the same issue. And they hint that Lyme disease does not cause psychiatric ailments such as depression and psychosis. The study is the first to test spinal fluid in groups of patients with five different classes of nervous system abnormalities to determine which might stem from Lyme disease.

"There's this notion that Lyme [disease] can cause everything under the sun. I'm trying to establish that there are very specific patterns to what Lyme produces," Halperin told SCIENCE NEWS.

Spinal taps are seldom used to diagnose Lyme disease in North America, and Halperin contends that most labs analyzing spinal fluid for Lyme-related antibodies do not use the optimal dilution. U.S. physicians typically look for a positive result from the somewhat unreliable blood-antibody test, coupled with the presence of telltale symptoms of Lyme disease (SN: 3/25/89, p.184), to infer that the Lyme-causing bacterium led to a CNS disorder, Halperin says.

Halperin's method enables physicians to distinguish between people whose spinal fluid antibodies originated in the bloodstream and those who are producing the antibodies within the central nervous system. This should result in more appropriate treatment, because people with the active CNS infection should receive antibiotics intravenously rather than orally, says Michael F. Finkel of the Western Wisconsin Lyme Disease Center in Eau Claire.

Halperin and his co-workers did spinal taps on 53 patients and tested various patients with brain magnetic resonance imaging and "evoked potentials," a measure of electrical activity in stimulated sensory nerve cells. They found abnormal evoked potentials in the multiple sclerosis patients only. Magnetic resonance images were abnormal in five of six multiple sclerosis patients and in seven of 17 encephalopathy patients but were normal in all others, they report.

The spinal tap was the only method yielding an abnormal result in a significant number of the patients with CNS disorders, Halperin says. The team found that 12 of 18 patients with encephalopathy, meningitis or a focal CNS disease (localized in one brain area) had Lyme-related antibodies in their spinal fluid. In contrast, none of the patients with either multiple sclerosis or a psychiatric illness, and only two of 24 patients with peripheral nervous system ailments, showed spinal fluid antibodies. — *I. Wickelgren*