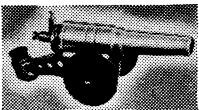


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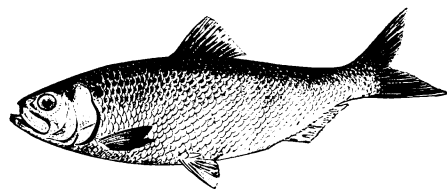
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it possible for lamprey eels, also originally from the Atlantic Ocean, to find their way into the upper Lakes where they multiplied rapidly while feeding

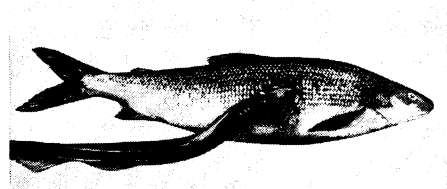


Interior

Alewife: smelly nuisance on shore.

on the juicy lake trout and whitefish they found there.

As the eels grew fat and ever more numerous, the populations of trout



Interior

Lamprey and dinner—a whitefish.

and other desirable fish rapidly dropped to near zero.

This, then, paved the way for the alewives. With the alewife-eating trout out of the way, the little—nine inches at most—alewives had essentially free run of the Lakes. Even the voracious lampreys found them too small for a satisfying meal and so left them alone to multiply.

Today, Greenwood estimates, there are several billion pounds of alewives in Lake Michigan alone. They have become the basis of a booming fishing industry which sells them for pet food and fish meal to processing plants that have set up along the Lake.

They have also become an annual nuisance, he notes, when they die after spawning. The city of Milwaukee has, for example, had to hire a commercial fisherman to do nothing but clean up the dead alewives from its lakefront.

This year's unusually large numbers of dead alewives simply attests to the especially successful hatch of alewife eggs observed by limnologists three years ago . . . the length of a typical alewife's life-span.

Besides its dual role as nuisance and profit-making industrial fish, the alewife is now slated for a third role—that of helping to restore some semblance of the natural ecological balance of fish in the Great Lakes.

The State of Michigan, Greenwood says, has been stocking Lakes Superior

and Michigan with lake trout and Boho salmon from the Pacific Coast. These fish, it is hoped, will eat the plentiful alewives and grow to provide fun and food for sport and commercial fishermen.

Success of the salmon and trout-raising program rests on the previous success of an anti-lamprey program. The eels, which would regard a salmon or trout as 24 hours worth of food, have been successfully controlled in Lake Superior and are on their way out in Lake Michigan, according to Greenwood.

Their demise has been largely due to use of a chemical lampricide placed in the streams where the eels lay their eggs. The chemical, discovered in 1957, kills the lampreys but not fish. Electric weirs have also been used to keep the eels from their spawning grounds. The chemical is now slated for use in the streams around Lake Huron, to rid the upper Lakes of lampreys.

## THALIDOMIDE

### A Clue to the Mechanism

Until German scientists reported in 1961 that 150 deformed infants—lacking developed arms and legs—had been born to women taking thalidomide during early pregnancy, the sedative was not a suspected teratogen. And because the details of its behavior in the body were poorly understood—as is the case with many drugs—scientists could not have predicted its interference with normal embryonic growth.

And so, with the hope of coming up with a measure for predicting which other drugs might also produce gross abnormalities in unborn infants, investigators set to work to clarify the mechanism of thalidomide's behavior at the molecular level. Their goal was to find out exactly how thalidomide works chemically; how its molecules combine with or interfere with chemical activities in living organisms.

Thalidomide is made up of three molecular rings. And, the design or structure of its third ring resembles the molecular design of a substance called glutamic acid—a naturally occurring amino acid present in all completed proteins. Because of this resemblance, some researchers concluded that thalidomide interferes with the metabolism of glutamate—a salt of glutamic acid implicated in the manufacture of nucleic acids. Presumably thalidomide caused deformities by blocking the synthesis of certain of these acids in cells which would eventually develop into limbs.

As a result of this conclusion, suspicion was cast on the safety of other compounds containing molecular structures similar to glutamate. A variety of

such drugs—otherwise unrelated—have been called into question.

However, new work indicates that the theory that thalidomide acts through glutamic acid won't stand up. Scientists at the National Institutes of Health, Bethesda, Md., now propose another explanation for thalidomide's behavior, and they suggest that, on the basis of their theory, it should be possible to spot other drugs that may cause deformed infants.

**In studies with rabbits** and rats, Drs. Herbert Schumacher, David A. Blake and James R. Gillette studied the toxicity of an analogue of thalidomide—a similar compound, but one without the third of its three rings. "Because this third ring is missing, this other compound cannot be metabolically changed to a glutamate derivative," Dr. Gillette says, "but the new drug is just as toxic to embryos as thalidomide itself."

The investigators suggest that thalidomide acts either directly on the fetus by invading its cells and irreversibly altering cell components such as DNA

and RNA which are directly involved in the transmission of genetic information. Or, it may act on some kind of "organizer" substances which theoretically determine why specific cells become primordial for certain organs—in this case, limbs.

**In either case**, new evidence contradicts the view that thalidomide produces abnormalities by interfering with glutamate. Its mechanism of action is more direct.

The NIH team suggests the major mechanism for teratogenesis by chemically unstable drugs such as thalidomide is through a process called acylation. In this process, a drug crosses the fetal barrier by dissolving in the lipids or fats in the barrier membrane and, because of its chemical instability, is able to react directly with genetic material in the fetal cells.

What this means, though other mechanisms may still be found, is that careful studies for possible teratogenic effects should be made on any drug that turns out to be both lipid soluble and chemically unstable.

#### AMA CONVENTION

## Cholesterol Study Sparks FDA Policy Review

Nondairy shortening and vegetable oil makers are fond of the words "polyunsaturated fats," and of using them to fight the dairy and animal food industries in the battle for cholesterol-conscious customers. They would like as well to list the proportions of kinds of fats in their products, but the Food and Drug Administration banned that 16 months ago. FDA said it would imply an unproved health claim which might cause people to start trying to treat themselves for real or imagined heart ailments.

Last week, however, results were released of a study that is already causing FDA Commissioner James L. Goddard to reconsider his agency's stand, and to wonder whether a food's saturated fat level isn't information that consumers—particularly heart patients—ought to have. His decision was expected to be announced by the end of this week.

**The cause of the FDA's** possible about-face was not even a major effort. Though it took more than two years, cost \$3.5 million and involved 2,400 subjects, the team of medical investigators who ran the study is adamant that it is only preliminary. In fact the doctors won't be happy with anything short of a study of up to 68,000 subjects, which could cost \$50 million.

**The preliminary study**, which was carried out only to see if a larger one could feasibly be made, was reported to the American Medical Association

meeting in Atlantic City last week by a team headed by Dr. E. Cowles Andrus of Johns Hopkins University. Ironically, he was standing in for study head Dr. Irvine H. Page of the Cleveland Clinic Foundation, who was recovering from a heart attack suffered the week before.

The investigation's subjects, all males between 45 and 54, included residents of Baltimore, Boston, Chicago, Minneapolis-St. Paul and Oakland, Calif., as well as institutionalized men from the Faribault, Minn., State School and Hospital. Some of the subjects lived on a normal American diet, while the rest received a custom-made diet of foods with controlled fat content. The study was double-blind, in which none of the subjects knew which of them were on which diet.

The key result reported was a 13.6 percent reduction in serum cholesterol in the group on the experimental diet. This diet's major feature was a reduction in foods containing saturated fats and cholesterol, and an increase in intake of polyunsaturated fats. An absolute reduction in fats of all kinds was not involved.

**Cholesterol is one**—but not the only—body lipid thought to be associated with heart disease. It apparently collects on the walls of the blood vessels, thickening and stiffening them while creating strain on the heart and circulatory system.

The feasibility study was never in-

tended to answer the question of whether low-fat diets would prevent heart attacks, Dr. Andrus emphasized. The study was too small. In recommending the larger study he pointed out that there is "overwhelming evidence that the incidence of coronary disease is strongly associated with serum cholesterol level and that this can be safely lowered by modification of the usual American diet in ways that are acceptable to large numbers of people."

#### HEALTH PHYSICS

## TV Radiation Assessed

X-rays emitted from color TV sets are no longer an easily dismissable problem.

Defective sets contribute to the total genetic radiation dose of the population, as well as causing possible cornea damage, such as cataracts, to viewers.

**Children crawling** under the badly designed console could be over-exposed to X-rays, since the harmful beam is directed downward. However, any *floor strong enough to support a color TV set would have sufficient matter to absorb all the radiation, so there would be no hazard to anyone in a basement beneath it.*

Dr. Harold Stewart, director of the X-ray exposure laboratory of the Public Health Service's National Center for Radiological Health warned, at a Conference on Radiological Health in Washington last week, of the possibility of slight mutations to persons exposed to a direct beam for a certain number of hours, though he was unable to be more precise.

Dr. Stewart found no significant X-ray emission from color TV sets tested in late 1966. Recent inspection of color TV's, however, showed radiation from some shunt voltage regulators, which direct the high voltage necessary to center the picture tube's electron beam.

General Electric Co., which produced the color TV assemblies in question is now exchanging the regulator tubes causing the trouble with another, non-harmful type. The danger of excessive radiation was first found by GE health physicists in routine tests of occupational hazards.

Some of the defective regulator tubes emitted as much as eight roentgens an hour in a downward and slightly forward direction, Dr. Stewart said. The National Council on Radiation Protection has set a limit of one-half a milliroentgen per hour at two inches.

Although only GE color TV sets with a K-C chassis and 6EF4 or 6LC6 regulator tubes have so far been declared as defective, the Public Health Service is testing 20 models of color TV from various manufacturers.