Fresh surface waters decline in Black Sea

A shallow freshwater layer atop the otherwise salty Black Sea supports fisheries vital to four nations. However, new observations suggest this oxygen-rich zone has thinned by about 30 percent in the past 13 years. Scientists disagree whether the process could threaten fish and whether dams around the Black Sea may have withheld enough fresh water to contribute to the change, discovered during five research cruises this spring and summer.

The expeditions, organized by U.S. and Turkish scientists and involving researchers from seven countries, offered Westerners their first opportunity since 1975 to sample the Black Sea's water and sediments. The sediment samples are scheduled to arrive in the United States by early November aboard the returning ship *R/V Knorr.*

Shipboard scientists found that the oxygen-rich layer, which in 1975 extended as far down from the surface as about 150 meters, now reaches no deeper than about 110 meters. They also observed that hydrogen sulfide no longer exists at the bottom of the oxygen-rich zone or directly beneath it. Instead, a band of water as thick as 40 meters and containing virtually no oxygen or hydrogen sulfide now spreads throughout the sea beneath the freshwater zone.

Scientists attribute the thinning of the surface layer, and perhaps its separation from the hydrogen sulfide region, to a change in the balance between saline and fresh water entering the 2-kilometer-deep sea. Salty Mediterranean water flows into the sea through the Bosporus strait, while fresh water enters it from Turkish and Eastern European rivers, many of them dammed.

Whether dams, nature or a combination of both has altered the water balance remains an unknown variable that could determine the fate of Bulgarian, Rumanian, Soviet and Turkish fisheries. "If the change is due to the damming of rivers, we may not have seen the end result," says expedition organizer James W. Murray of the University of Washington in Seattle. But Bernward J. Hay of the Woods Hole (Mass.) Oceanographic Institution doubts dams could account for the drop in freshwater volume. Hay, who led one of the research cruises, says normal variations in climate and the sea's biological productivity could account for the change in water balance.

New clues to the cause could come from the sediment cores. Scientists hope the sediments — deposited at different times in the past — will indicate when oxygen depletion killed off marine life at various depths. Such evidence could help them determine if the recent decrease corresponds to a trend that predates dam construction. However, only long-term monitoring of the balance between the Black Sea's saline and fresh water can clarify the future of its fisheries, Murray says. — *C. Knox*

Dioxin migrates out of milk cartons

Throughout the industrialized world, people are accumulating a low-level body burden of toxic dioxins and furans, including the highly toxic TCDD (SN:7/13/85, p.26). A Canadian government scientist has found one potential source in the food supply: cardboard cartons used to package cow's milk.

John J. Ryan, an environmental chemist with Health and Welfare Canada in Ottawa, examined milk samples as part of a large study to identify dietary sources of dioxin. While all samples showed low levels of highly chlorinated dioxins — those having 5 to 8 chlorine atoms per molecule — only a few contained the 4-chlorinated dioxin, TCDD, and a somewhat less toxic 4-chlorinated furan, TCDF. Quantities of both were low — 0.04 parts per trillion (ppt) TCDD and 1.0 ppt TCDF — and appeared only in milk from plastic-coated cardboard cartons. Milk packaged in plastic or glass showed none.

Ryan noticed a similarity between the TCDD:TCDF ratio in his milk and a TCDD:TCDF ratio reported a year ago by U.S. Environmental Protection Agency scientists studying pulp-paper mills. The EPA study showed that mills using chlorine to bleach their wood pulp create dioxins and furans that can end up in the paper. When Ryan analyzed his cardboard milk cartons, he found TCDD and TCDF. He presented preliminary findings at an August symposium in Umeå, Sweden.

If all milk were contaminated with the levels Ryan found, "there'd be an immediate concern," says dioxin toxicologist Stephen Safe at Texas A&M University in College Station. As it is, he says, this constitutes a problem and should be eliminated. Michael Farrar, vice president for Environment and Health with the New York City-based American Paper Institute, says, "We had been trying to do [paper-dioxin] migration work, but had trouble" measuring the trace levels involved. About two weeks ago, one of the institute's researchers met with Ryan to discuss his analytical techniques and findings. On the basis of that meeting, Farrar says, the institute now expects to begin its own study of the potential problem. — *J. Raloff*

Platelets enter into Alzheimer's disease

Several years ago, scientists found that Alzheimer's patients with excess fluid in the surface membranes of their blood platelets develop symptoms earlier — in their mid-60s and early 70s — and suffer more debilitating effects than Alzheimer's patients without the platelet abnormality. The researchers, at the University of Pittsburgh, also found increased platelet-membrane fluid in about half the parents and siblings of Alzheimer's patients with the biological defect (SN: 11/7/87, p.301).

The same group now reports in the October ARCHIVES OF GENERAL PSYCHIATRY that parents and siblings of Alzheimer's patients with elevated platelet-membrane fluidity who develop symptoms of Alzheimer's disease do so significantly earlier — again in their 60s and early 70s — than relatives of patients with normal platelet-membrane fluidity.

The average age at which the first Alzheimer's symptoms appeared for relatives of patients with increased platelet-membrane fluidity was more than five years younger than for relatives of patients without the potential biological marker.

The finding further supports the notion that there is more than one form of Alzheimer's disease, write psychiatrist Gary W. Small and biomathematician David A. Greenberg of the University of California, Los Angeles, in an accompanying editorial. They say it is also consistent with the hypothesis that multiple genes, possibly including one or more coding for platelet-membrane fluidity, increase a person's risk for Alzheimer's disease.

A biological marker may have little or nothing to do with the causes of a disease, Small and Greenberg caution, although platelet abnormalities could indicate Alzheimer's is a metabolic disease extending well beyond the brain.

"Costly and time-consuming" prospective studies of relatives of Alzheimer's patients are necessary to learn which participants with elevated platelet-membrane fluidity will develop the disease, add the Los Angeles researchers. The strength of the biological measure as a diagnostic tool is still unclear.

The Pittsburgh study, directed by psychiatrist George S. Zubenko, concentrated on 421 parents and siblings of 43 Alzheimer's patients and of 47 healthy controls. Family members and others who knew each participant well answered a standardized questionnaire designed to distinguish between symptoms of Alzheimer's disease and other disorders, such as stroke, alcoholism and Parkinson's disease. — *B. Bower*

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