Behavior

Dealing with a distant disaster

Emotional aftershocks of the massive earthquake that devastated Soviet Armenia in December 1988 struck Armenian teenagers in Los Angeles during the first week after the disaster, according to a report in the July BULLETIN OF THE MENNINGER CLINIC.

Interviews with Armenian-American adolescents, conducted by Viken V. Yacoubian, a graduate psychology student at the University of Southern California in Los Angeles, and the late psychiatrist Frederick J. Hacker, uncovered few of the post-traumatic stress reactions of people directly exposed to a disaster. The Los Angeles teenagers did, however, express considerable guilt and remorse over having lived while others died; they strongly identified with the victims and reported a great deal of rage linked to the catastrophe.

At first, many of the teenagers — students at a private Armenian school where Yacoubian is a teacher and counselor — rushed into hectic disaster-relief activities. They said they could tolerate their grief only by constantly doing something. Students exerted strong pressure on one another to make great sacrifices for the cause. Doubters who questioned the usefulness of specific relief activities "were quickly silenced and severely attacked," the researchers say. Most of the students expressed strong resentment toward teachers and parents who wanted them to maintain regular school hours.

A number of teenagers experienced what the researchers call "participation envy," an envious resentment at being excluded from what was perceived as a unique experience rallying Armenians throughout the world. These students often developed fantasies of being magically transported to Armenia to assist survivors directly.

On the other hand, the researchers note, the students did not express religious doubt or anger at God in the week following the earthquake. They attended more church services and prayer meetings, the investigators say, pointing to a renewed link between national Armenian and religious Christian loyalty.

Yacoubian and Hacker interviewed two groups of students ranging in age from 15 to 18 years. One session, with 25 students, occurred five days after the disaster. A second session, with 20 students, took place eight days after the earthquake.

Elderly suicides rise in 1980s

Recently released federal statistics indicate the suicide rate among people 65 years of age and older increased by 25 percent between 1981 and 1986 in the United States. The elderly suicide rate has long been higher than the rate for other age groups but had been steadily dropping for nearly 50 years, a trend apparently undergoing reversal in the 1980s.

The six-year increase brought the suicide rate for those 65 and older to 21.6 per 100,000 people, according to data compiled by the National Center for Health Statistics in Hyattsville, Md. During the same period, the overall national suicide rate climbed 5 percent, from 12.1 per 100,000 to 12.8 per 100,000.

The 1986 suicide rate of those 15 to 24 was 13.1 per 100,000, also a 5 percent increase. The suicide rate of those 25 to 44 was 15.5, and those 45 to 64 had a rate of 16.7; both groups showed a slight decrease in suicides. Youngsters aged 5 to 14 had a suicide rate of 0.8 per 100,000.

The Hyattsville center gathers its statistics from state records of death certificates listing suicide as the cause of death. These figures are thought to underestimate the actual number of suicides. Although the federal report offers no explanations for the surge in elderly suicides, some researchers have already noted a growing tendency among the elderly to choose "rational suicide" over the infirmities of old age (SN: 12/3/88, p.366).

Biomedicine

Patrick Young reports from Bar Harbor, Maine, at the annual Short Course in Medical and Experimental Mammalian Genetics

Four steps to lymphoma

Working with genetically engineered mice, Charles L. Sidman of the Jackson Laboratory in Bar Harbor has discovered four specific steps in the development of B-cell lymphoma — a cancer of the immune system — that occur in mice after activation of a cancer-triggering gene, or oncogene. And in a flip of the usual pattern of finding animal models that mimic human diseases, Sidman has identified a human condition that appears to mimic the mouse malignancy and has joined with physicians to investigate it. "Guided by what we've been learning in the mouse, we're trying to study successive changes in humans that mirror this [mouse] disease," he says.

Sidman uses a strain of transgenic mice that always develop B-lymphocyte tumors. His studies reveal that the mice first experience a proliferation of B-cells, and then the proliferation goes away. In step three, the proliferation returns. In step four, the B-cells transform from normal cells to cancerous ones.

But sometimes step four can precede step three. "That's a striking observation," Sidman says. "The early concept was that cells proliferated wildly and that's the cancer. But we can get cells that can create a cancer [in other mice] before the return of proliferation. The return of proliferation may not be synonymous with the ability of cells to cause a cancerous tumor."

These findings suggest new points where physicians might one day intervene to prevent cancer. Sidman suggests the four-step process applies to human leukemias and lymphomas and may apply to all cancers. He is now trying to decipher the biological mechanisms that regulate the various steps in mice.

Some people develop a condition called benign monoclonal gammopathy, marked by a proliferation of a specific set of B-cells. Each year, about 6 percent of these people go on to develop malignant myeloma, which will strike about 11,600 people in the United States this year. Sidman and a research team at the Maine Cytometry Research Institute in Portland plan to test and follow the B-lymphocyte changes in several dozen patients with benign monoclonal gammopathy. "This gives us a situation in which to explore the events and genes that make a difference," he says.

Modifying Mendel one more time

Even as geneticists adjust to the heretical concept of genomic imprinting (SN: 5/20/89, p.312), they confront yet another discovery that challenges classical Mendelian genetics.

The genomic imprinting concept holds that while two genes may contain identical DNA sequences, how a gene behaves may depend on whether the mother or the father provided it. Recently, researchers reported another startling finding — two cystic fibrosis patients who inherited both copies of chromosome 7 from their mothers. "That's not supposed to happen," says Judith G. Hall of University Hospital in Vancouver, British Columbia, reviewing this and other genetic anomalies at the Bar Harbor meeting. Researchers from the Baylor College of Medicine in Houston and Jerusalem's Hadassah Hospital originally described the finding last fall.

Human cells contain 23 chromosome pairs. Classical genetics holds that one chromosome in each pair comes from the mother, the other from the father. The two cystic fibrosis cases represent the first reported examples of uniparental disomy — the inheritance of both chromosomes in a pair from the same parent. Cystic fibrosis requires inheriting two defective genes, normally one from each parent. In the two uniparental disomy cases, the mother contributed both defective genes.

"What you're doing is producing a recessive disease with one parent as the carrier," Hall says. "The question — because this work is so new — is how common is it? It's not going to be common, but it is for real."