Yellow rain tale: The plot thickens

Canada offers one of the latest developments in the "yellow rain" military mystery of whether Soviet-supplied, internationally outlawed chemical weapons have been used in Southeast Asia. Recognizing the need for an objective look at the U.S. State Department claims that Communist forces have used a fungal-poison weapon, dubbed "yellow rain," in Laos and Kampuchea, the Canadian government called on H. Bruno Schiefer, a veterinary pathologist at the University of Saskatchewan in Saskatoon. In February, Schiefer spent two weeks at refugee camps close to the Thailand-Kampuchea and Thailand-Laos borders, interviewing presumed victims of chemical attacks and collecting plant and soil samples. In a recently released report based on his trip, Schiefer concludes that reported symptoms of alleged attacks cannot be attributed to poisoning due to levels of fungal toxins that could occur naturally in the area or to other naturally occurring diseases. While such a statement far from resolves the issue, it is significant in that it appears to support the U.S. contention that modified fungal toxins are being sprayed in Southeast Asia.

Another development in the yellow rain case is a recently released Soviet rebuttal of U.S. allegations. Since last fall, the State Department has produced various pieces of evidence—including results of analyses of blood samples taken from presumed victims of chemical attack — that suggest the existence of a Soviet-supplied warfare agent based on T2 and other poisons produced in nature by certain species of the fungus *Fusarium* (SN: 10/17/81, p. 250; 11/21/81, p. 327; 2/20/82, p. 122; 4/3/82, p. 230; 5/22/82, p. 343). In the recent rebuttal — prepared by officials of the USSR Academy of Sciences, the USSR Ministry of Health and other Soviet organizations — the Soviet government acknowledges cases of mycotoxin poisoning in Southeast Asia but charges that "the military leaders of the United States are the true guilty parties."

First, the Soviet explanation goes, U.S. troops repeatedly sprayed herbicides on the forests of Vietnam in the 1960s. When the resulting dead wood was ignited by napalm, soil temperature rose to 120°F, killing the microflora and microfauna. These "sterilized" areas then were seeded (from the air) with elephant grass — a plant more herbaceous than tree-like. Next, toxin-producing *Fusaria* — "which live selectively as parasites on herbaceous plants and whose natural [microfungi] enemies ... had been annihilated" — flourished. Finally, winds from the Gulf of Siam carried *Fusarium* spores from Vietnam to certain provinces of the adjacent Kampuchea and Laos. "These facts reveal the hidden truth of who [is] really responsible for the mycotoxicoses in Southeast Asia," the Soviet report concludes.

But James Leonard of the State Department's Bureau of Politico-Military Affairs says such an accusation is "preposterous." The Soviet document is filled with "extravagant conjectures" that have "no scientific basis," says Leonard, who has an educational background in plant pathology. "If the theory had any foundation whatsoever," he told SCIENCE NEWS, "you'd find people suffering [from mycotoxin poisoning] over vast stretches [of Southeast Asia] — not just in isolated areas of military activity." Says Leonard, the State Department now is preparing a detailed critique of the Soviet document.

In addition, the State Department is investigating some of the possibilities suggested in the Schiefer report. For example, the Canadian researcher notes that the reported rapid onset of skin rashes, difficulty in breathing and hemorrhaging after alleged chemical attacks is more consistent with illnesses caused by mycotoxins more complex than the T2 and others that already have been implicated. Of course, Schiefer reports, it is possible that these simpler mycotoxins could act quickly if they were attached to a chemical carrier — such as dimethylsulfoxide (DMSO) — that would facilitate their entrance into the body. "We're actively looking at the possibility of chemical carriers or other agents in yellow rain," Leonard says. Schiefer's study, he says, "is very helpful."

— L. Garmon

Radiation: When less is not better

Understanding how radiation interacts with body tissue is a complex business at best. There are so many variables to consider, such as type of radiation, its energy level, the rate at which it is delivered and the total dose absorbed. Now researchers have encountered what appears to be a paradox involving a particularly potent form of radiation to which nuclear-power-plant workers may be exposed: The ability of "fission" neutrons to transform normal cells into a cancerous state appears to grow stronger as the amount of radiation delivered — per unit time — is reduced.

In the research, Mortimer Elkind (now at Colorado State University), Colin Hill and colleagues at Argonne National Laboratory exposed mouse-embryo cells to fission neutrons from the JANUS reactor — a unique source of "clean" neutron fluxes for biological studies. Neutrons with a mean energy of 0.85 million electron-volts were delivered at very low rates (0.43 rad/minute and 0.086 rad/min.) and at rates as much as 200 times higher. (A rad is a unit of absorbed dose for ionizing radiation.) Total doses ranged from 10 to 454 rads.

What the researchers found was that for equivalent neutron doses, those delivered at the very low rate turned out to be significantly better at transforming normal mouse cells into tumorous cells. At the 10-to-20-rad end of the spectrum, the lower dose rates were 10 times more effective in transforming cells than the higher dose rates (10.3-38.5 rads/min.), according to a report in the July 1 NATURE. And Elkind told SCIENCE NEWS that new data for total doses as low as 2.5 rads, accumulated since the first report was sent to press, "confirm a smooth dose dependence between 0 and 10 rads when we irradiate at a reduced dose rate." The remarkable differential between high and low dose rates tended to disappear as the total dose reached 100 to 150 rads.

Interestingly, regardless of how the dose was "packaged," the cell lethality of a given dose never varied.

In the past, concern over radiation exposures tended to use total accumulated dose as a focus for concern. Less worrisome, especially when the total was relatively small, was how the dose had been packaged — in one acute dose, several small packets or a lifetime of mini doses. Based on research involving gamma-rays, however, conventional wisdom suggested that the smaller the individual dose, the better, for when sublethal exposures were parceled out over time, the body sometimes exhibited "repair" of radiation damage between exposures. Therefore, particularly with regard to their tumor-inducing potential, two 1-rad exposures of gamma radiation were considered preferable to a single 2-rad dose.

Neutrons were another story. Regardless of how the total dose had been packaged, it appeared repair did not occur. As a result, doses were considered additive in effect, explains Douglas Grahn, a radiation geneticist at Argonne National Laboratory, whose work is unconnected with Elkind's. But the implication of the work by Elkind's team now contradicts this: A 2-rad exposure to fission neutrons would not be equivalent to — but actually preferable to — two 1-rad exposures.

Hill says his team's revised explanation for what seems to be happening is that a neutron-damaged cell indeed repairs its own damage, but in an "error-prone" fashion. And it's the faulty repair that induces tumor transformation. However, if a neutron dose is delivered too quickly for the cell to attempt repair, its transformation risk will be reduced.

"Although cancer induction in man involves many factors besides those at the cellular level," writes the Argonne team in NATURE, "the implication from our findings is that the risk of cancer induction due to work-related exposure to neutrons in the nuclear power industry may be greater than previously thought."

—J. Raloff