Letters

Synthetic Elicitors

We would like to draw attention to an error in Thomas H. Maugh II's article "Exploring plant resistance to insects" (Research News, 14 May, p. 722) pertaining to a description of our work on synthetic elicitors. He reports that both aryl cluster glycosides and fluorinated carbohydrates stimulate production of phytoalexins in soybeans about ten times as effectively as naturally occurring elicitors. This is incorrect; both series of compounds were determined to be inactive by the soybean cotyledon bioassay.

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Mycotoxin Weapons

In Eliot Marshall's article "The Soviet elephant grass theory" (News and Comment, 2 July, p. 32), Paul Nelson is quoted as saying that he "has never come across any reference to a toxin-producing *Fusarium* in Vietnam, Laos, or Kampuchea." In fact, no one has systematically surveyed the area for toxigenic fusaria. To our knowledge, the four trichothecene-positive samples collected from sites of "yellow rain" attacks were not examined for the presence of toxin-producing fungi.

In Marshall's article "Yellow rain: Filling in the gaps" (News and Comment, 2 July, p. 31), the value of T2 toxin as a weapon is questioned because of high cost: "Researchers must pay $7000 or more for a gram of the most potent variety of T2." This point confuses the issue. First, there is only one "variety" of T2 toxin. Second, the $7000 per gram price is extrapolated from 10-milligram analytical standards. Moreover, even the Department of State maintains that crude extracts are being used. It is our view that such crude extracts would be simple and inexpensive to produce and could be manufactured by Southeast Asian countries. The processes involved are described in the open scientific literature [for example, see (*1*)].

Readers of Marshall's articles might conclude that high toxin-producing strains of *Fusarium* are unavailable. In fact, highly toxic strains are available with virtually no security checks from dozens of laboratories and culture collections.

The evidence, to date, for Soviet complicity in the production of mycotoxin weapons rests on a listing (*2*) of Soviet scientists involved in mycotoxin research and the existence of fermentation facilities in the Soviet Union. Clearly, by this standard of circumstantial evidence, we must also be occupied with such diabolical pursuits. Why then is our government so eager to condemn our Soviet colleagues?

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References

1. H. R. Burmeister, *Appl. Microbiol.* **21**, 739 (1971).
2. *Report on Yellow Rain* (Department of State, Washington, D.C., 22 March 1982).

An unanswered question in the article by Eliot Marshall describing the use of mycotoxins in Kampuchea is why T2 toxin was present in blood samples from people 2 weeks or more after exposure. I suggest that this is best explained by a sequestering of the chemical in certain organs, particularly the kidney, rather than by binding to serum albumin, as was proposed. Many drugs have a high affinity for albumin, including, for example, clofibrate, warfarin, and most of the aspirin-like compounds; yet all of these are eliminated quite rapidly from the body (*1*). Other chemicals, such as gentamicin and ochratoxin A, have quite different binding affinities toward albumin; yet both are concentrated to a high degree in the proximal tubules of the kidney and are eliminated very slowly from the body over a period of time. Thus, the binding and accumulation of trichothecenes in the kidney, followed by a slow elimination phase, would account for the appearance of measurable quantities found in victims weeks after exposure.

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References

1. A. G. Gilman, L. Goodman, A. Gilman, *The Pharmacological Basis of Therapeutics* (Macmillan, New York, ed. 6, 1980).

The article by Eliot Marshall "Yellow rain: Filling in the gaps" is a fairly accurate account of the feelings of various U.S. *Fusarium* and trichothecene specialists concerning this topic. Most believe that, indeed, (i) *Fusarium* toxins have been found in Southeast Asia; (ii) they are not of natural origin; (iii) they could explain a majority of the signs and symptoms described, although reservation exists as to the rapidity of the onset of hemorrhaging; and (iv) T2 and HT2 have been found in blood and urine of victims, but it is difficult to understand why residue was found in victims 18 days after exposure.

Our laboratory has been involved in the analysis of various *Fusarium* toxins since 1963 and draws on this experience in coping with the analysis of trichothecenes in various substrates. Since 1970 we have developed and used a combination of gas chromatography and mass spectroscopy in the detection and quantitation of these mycotoxins. Our present mass spectral analytical approach includes selected ion monitoring in both the electron impact and positive chemical ionization mode. Our resolution is done on 15- and 30-meter capillary columns, so that retention times are very precise. In all selected ion monitoring analyses, we insist on a minimum of a base peak and a molecular ion with the correct ratio of intensity before a positive identification is made. When possible, we obtain a full mass spectral scan to support our conclusion. My assistant Robert Pawlosky (mass spectrometer operator) and I have a high degree of confidence in our results and hence take a positive view in the questions we ask, namely, What organs are involved in binding the T2 residue? Can T2 and metabolites in blood and urine be used as a diagnostic tool by a veterinary diagnostician? And what are the effects on metabolism of T2 when the liver and kidney are overwhelmed with a toxic dose?

I believe Marshall's article should have stressed more poignantly that T2