ies to the relative risk that smokers have of dying from heart disease. But the diet-cancer risks are inconsistent—some studies show them and others do not. The smoking–heart disease relation is far better established. Of course, if there were good evidence of a relative risk of 2 in the cancer studies, it would be of great importance.

Hennekens says he did not describe the carotene hypothesis as "soft." He did, however, call it "immature."

Although Willett may well believe the vitamin A hypothesis should be actively pursued, he was not misquoted. The quote comes from our lunchtime conversation, not from his remarks at the meeting.—GINA KOLATA

Contamination of Arizona Corn

In his article of 5 August 1983 (News and Comment, p. 526), Eliot Marshall discusses the assertions of Chester Mirocha regarding the effect of corn contaminated with *Fusarium moniliforme* that was used as feed for chickens in Arizona. This moldy corn caused the death of many chickens, which led to serious financial losses for chicken farmers and feed suppliers.

Mirocha is quoted as affirming that *F. moniliforme* does not produce trichothecenes and that corn samples from Arizona contaminated by this fungus did not contain trichothecenes. These affirmations have been challenged by a number of scientists.

There are many published reports (*1*) that *F. moniliforme* produces some trichothecenes, mainly vomitoxin and diacetoxyscirpenol, in addition to zearalenone, moniliformin, and some other compounds (antibiotics, gibberellin, complex hormone substances from *Gibberella fujikuroi*—the perfect stage of *F. moniliforme*; and other related gibberellin fractions, including gibberellic acid). *Fusarium moniliforme* is a field fungus common in soil and many crops, including maize, oats, millet, sorghum, barley, and wheat.

Mirocha is quoted as saying that there is "no significance" in the fact that Arizona corn feed contained extremely high counts of *F. moniliforme* spores. He also is quoted as saying that "Nobody has demonstrated a correlation between spore counts and toxicity of cereal grains."

During my investigative work on alimentary toxic aleukia, I have consistently found a correlation between spore counts and toxicity of cereal grains. Cultures of *F. poae, F. sporotrichioides*, and other fungi have been shown to be most toxic during the period of abundant sporulation (*2*).

Toxic strains of *F. moniliforme* and their varieties, isolated from various substrates in Israel, have been characterized morphologically by abundant production of spores, and those strains that did not produce abundant spores have not been toxic. It is logical to propose that high spore counts of *F. moniliforme* in Arizona are also correlated with an increase in toxicity of corn and thus are a significant factor that should be taken into account.

Fusarium moniliforme is common in Israel and has been found in light, medium, and heavy soils and loess. It has been isolated in Israel from the following agricultural plants: maize, sorghum, onion, cucumber, pepper, cotton, groundnuts, and various fruits, such as bananas, grapefruits, Valencia oranges, avocados, and mangoes (*3*). In all, 133 isolates of *F. moniliforme* from Israel, in addition to 13 isolates from other countries, have been examined for phytotoxic and other toxic properties (*4*). In Israel, *F. moniliforme* is the most prevalent fungus. It has been found mainly on maize kernels, and our evidence indicates that toxic metabolites produced by this fungus are a major cause of disease. Crude extracts from *F. moniliforme* have caused moderate to strong inflammatory reactions, edema, and necrosis when applied to rabbit skin.

The role that *F. moniliforme* plays in the potential mycotoxicoses of farm animals consuming agricultural commodities that have been produced in semitropical and tropical countries has not been determined. However, it is suspected that mycotoxicoses do occur in these regions. Most research on fusariotoxin outbreaks have centered on crops produced in the temperate zone. Considerably less is known about mycotoxins other than aflatoxin in semitropical and tropical climates.

A comprehensive survey of *F. moniliforme* and its varieties, which belong to the Liseola section, would provide valuable information about their role in mycotoxicoses.

ABRAHAM Z. JOFFE
Department of Botany,
Hebrew University of Jerusalem,
Jerusalem 91904, Israel

References

1. U. L. Diener, *Proc. Am. Phytopath. Soc.* **3**, 126 (1978); C. M. Hesseltine, in *Proceedings of the Third International Biodegradation Symposium*, J. M. Sharpley and A. M. Kaplan, Eds. (Applied Science, London, 1978), pp. 607–623; J. Robb, K. S. Kirkpatrick, M. Norval, *Vet. Rec.* (23 October 1982), p. 389; C. I. Szathmary, in *Trichothecenes: Chemical, Biological and Toxicological Aspects*, Y. Ueno, Ed. (Elsevier, Amsterdam, 1983), pp. 221–228; R. F. Vesonder, A. Ciegler, A. H. Jensen, *Appl. Microbiol.* **26**, 1008 (1973); R. F. Vesonder, A. Ciegler, H. R. Burmeister, A. H. Jensen, *Appl. Environ. Microbiol.* **38**, 344 (1979).
2. A. Z. Joffe, *Mycopathol. Mycol. Appl.* **16**, 201 (1962); in *Microbial Toxins*, S. Kaolis, A. Ciegler, S. J. Ajl, Eds. (Academic Press, New York, 1971), pp. 139–189; in *Mycotoxins*, I. F. H. Perchase, Ed. (Elsevier, Amsterdam, 1974), pp. 229–262; in *CRC Handbook of Foodborne Disease of Biological Origin*, M. Rechcígl, Jr., Ed. (CRC Press, Boca Raton, Fla., 1983), pp. 353–495.
3. A. Z. Joffe, *Plant Dis. Rep.* **46**, 31 (1962); *Mycologia* **55**, 271 (1963); *Soil Sci.* **95**, 353 (1963); *Mycopathol. Mycol. Appl.* **32**, 209 (1967); *ibid.* **39**, 255 (1969); *Plant Dis. Rep.* **56**, 963 (1972); *Plant Soil* **38**, 339 (1973); _____ and Nadel-Schiffmann, *Fruits* **22**, 97 (1967); *ibid.* **27**, 117 (1972); A. Z. Joffe, Y. Yaffe, J. Palti, *Soil Sci.* **104**, 263 (1967); A. Z. Joffe, J. Palti, R. Arbel-Sherman, *Mycopathol. Mycol. Appl.* **50**, 85 (1973).
4. A. Z. Joffe, in *Trichothecenes: Chemical, Biological and Toxicological Aspects*, Y. Ueno, Ed. (Elsevier, Amsterdam, 1983), pp. 95–111.

Gene Therapy Researchers

I would like to correct two statements made in the article "Gene therapy method shows promise" by Gina Kolata (Research News, 30 Mar., p. 1376).

Our laboratory did not develop the β-thalassemic mouse. This mutant was developed by Susan Lewis, Chemistry and Life Sciences Group, Research Triangle Institute, Research Triangle Park, North Carolina, under contract with the National Institute of Environmental Health Sciences (NIEHS) as part of the National Toxicology Program. The development was part of a long-term program to obtain medically and biologically significant mouse mutants. Our laboratory joined with Lewis as well as with Loren Skow and Frank Johnson at NIEHS and Ray Popp at the Oak Ridge National Laboratory to study the molecular defect in the β-thalassemic mouse. These mice are available from Lewis.

Eli Gilboa is at Princeton rather than Yale.

W. FRENCH ANDERSON
National Heart, Lung, and Blood Institute, Bethesda, Maryland 20205

Erratum. In the article "NSF plans help with big computer problems" (News and Comment, 24 Feb., p. 797) by John Walsh, a reference to Kenneth G. Wilson's work was incorrectly printed. The third sentence in the second full paragraph of the third column should have begun, "Kenneth G. Wilson of Cornell, who won the 1982 Nobel Prize for his theoretical work on phase transitions. . . ."

Erratum. On page 1426 of the report "Association of parvoviruses with rheumatoid arthritis of humans" by R. W. Simpson *et al.* (30 Mar., p. 1425), a portion of the text beginning on line 18 of the first full paragraph in column 3 was incorrectly printed. It should have read, "We were unable to detect 24-nm particles in brains of normal mice from our colony or mice intracerebrally inoculated with extracts of synovial cells from patients with noninflammatory degenerative joint disease (DJD). More conclusive evidence for the identity of RA-1 as a parvovirus comes from our recent success in demonstrating that CsCl gradient fractions containing the infectious 24-nm particles can be extracted for a single-stranded DNA which is approximately 4.5 kilobases in size."