predictions of economic hardship and consumer dissatisfaction that Marshall debunks. In light of the new scientific and economic evidence, for *Science* to continue to take the side of the real "alarmists" over those who urged or took prudent action about Alar is sour grapes, plain and simple.

ADAM M. FINKEL
Center for Risk Management, Resources for the Future, 1616 P Street NW, Washington, DC 20036

REFERENCES AND NOTES

1. B. Toth, *J. Natl. Cancer Inst.* **50**, 181 (1973).
2. E. I. Goldenthal, "Two-year oncogenicity study in mice 'with UDMH' " (Report No. IRDC-399-065, International Research and Development Corporation for Uniroyal Chemical Company, Mattawan, MI, 1990).
3. There is some question whether the 13 mg/kg/day dose caused toxic effects that confounded the carcinogenic response, but even Bruce Ames' colleague Lois Gold apparently accepted the validity of the 7 mg/kg/day dose. Thus, the statement in Marshall's article (attributed to Gold) that "there appeared to be some significant tumors" at 7 mg/kg/day leaps off the printed page; such a statement could lead a reader unfamiliar with the study to believe that there were only three or four animals with cancer rather than 31.
4. An explanation of the rationales for, and influence of, two EPA adjustments that, if omitted, would together bring the 1973 and 1991 estimates closer by a factor of 16 is available from the author.
5. For a 20-kilogram child, the 7.3 mg/kg/day dose corresponds to just under 1 mg/kg/day (if you believe interspecies conversion should be based on body surface area) or about 1.5 mg/kg/day (if you use the proposed EPA–Food and Drug Administration compromise approach of scaling based on body weight to the three-fourths power).

Alar is now gone from the food supply, and public health is better protected. Despite dire industry predictions, neither the quality nor the quantity of apples has diminished in the absence of Alar. The 4 October News & Comment story and the subsequent 1 November editorial on Alar contained several serious errors, creating the impression that Alar posed little health risk. However, the latest studies submitted to the Environmental Protection Agency (EPA) once again demonstrate that Alar's metabolite UDMH (unsymmetrical dimethyl hydrazine) is carcinogenic and that Alar's dietary risk is 26 times EPA's standard of acceptable lifetime cancer risk of 1×10^{-6}. Even with the revised estimate of UDMH's carcinogenic potency derived from the new bioassays, the dietary hazards of Alar exceed EPA's standard of acceptable cancer risk. This conclusion was affirmed in a letter to *Science* (29 Nov., p. 1276) from Victor Kimm, Deputy Assistant Administrator, Office of Pesticide Programs and Toxic Substances at EPA. The most recent cancer studies confirm the results of the earlier bioassays relied upon in the Natural Resources Defense Council's report *Intolerable Risk: Pesticides in Our Children's Food*—that Alar's metabolite UDMH is carcinogenic; there is *no* dispute that UDMH is a probable human carcinogen.

Science has missed the most important lesson of the Alar episode. As long as cancer remains one of the leading causes of disease and death in our society, the prudent course is to reduce and avoid exposure to carcinogens, particularly those that are unnecessary. The removal of Alar without impacts on apple production was an important step toward the goal of decreasing unnecessary and avoidable exposure to carcinogens in the food chain.

LAWRIE MOTT*
Natural Resources Defense Council, 71 Stevenson Street, San Francisco, CA 94105

*Co-signers: Harvey Karp, School of Medicine, University of California, Los Angeles, CA 90405; Frank Mirer, United Auto Workers, Detroit, MI 48214; Herbert L. Needleman, School of Medicine, University of Pittsburgh, Pittsburgh, PA 15213; William J. Nicholson, Mt. Sinai School of Medicine, New York, NY 10029; Brad Sewell, New York, NY 10025; Ellen Silbergeld, School of Medicine, University of Maryland, Baltimore, MD 21201–3613; Robin Whyatt, Garrison, NY 10524.

Response: I believe my editorial and Eliot Marshall's article provide a more balanced approach to the facts than do the two letters above.—DANIEL E. KOSHLAND, JR.

Supermelons?

We folks here in Kentucky are well aware that California produces a lot of fruits and vegetables and we are glad of it, especially in the winter when a hard pink tomato is a real treat. However I garden a bit when I'm not doing biochemistry or reading *Science*, and I was startled to read in the article by Elizabeth Culotta about the "Superbug" (News & Comment, 6 Dec., p. 1445) that Nick Toscano of the University of California, Riverside, found a California field that grows 750,000 melons per acre. I learned as a farm boy that an acre covers 43,560 square feet, so this remarkable field produces about 17 melons per square foot. If these are cantaloupes, with each fruit covering about 1 square foot, I guess they must grow in a stack 17 deep. That sure would be handy for pitching on the truck when picking, but I imagine it makes a tough row to hoe or to spray for those nasty whiteflies. If these unspecified melons are big watermelons the problems must be even worse. Could the weight of these stacks of melons in the Imperial Valley be the cause of California's excessive seismic activity? If so, then the whiteflies may be the only means of preventing the Big One. In spite of these potential problems and Kentucky's proximity to the New Madrid fault, I have just one request: please send me some seeds!

JERALD L. HOFFMAN
Department of Biochemistry, University of Louisville, Lousiville, KY 40292

Response: If Dr. Hoffman gets his melon seeds, he doesn't have to worry about setting off the New Madrid fault. Nick Toscano misinterpreted the growers' figures. That 1-acre field will produce about 500 cartons of cantaloupes—or up to a maximum of about 11,000 melons, depending on how big they are. The 750,000 figure is the total number of melon cartons produced by about 1500 acres in the Imperial Valley. Of course, in the fields with the worst whitefly infestations, the melon yields are zero any way you count them.

—EDS.

The Arginine Fork: Correction

In the paper by B. J. Calnan *et al.* about the interaction of HIV (human immunodeficiency virus) Tat peptides with TAR RNA (Reports, 24 May 1991, p. 1167) (*1*), it was reported that a single arginine in Tat is involved in sequence-specific RNA recognition and that modification of two phosphates at a three-nucleotide bulge in TAR interferes with binding. These phosphates were said to be located between nucleotides A22 and U23 and U23 and C24. However, we have subsequently found that one of the assignments was incorrect and that the two phosphates are actually located one position lower, between nucleotides G21 and A22 and A22 and U23. Both pairs of phosphates, at the junction of the stem and bulge, are consistent with the modeling described in the paper, and the main conclusions are unchanged. We regret any inconvenience this error may have caused.

ALAN D. FRANKEL
JIANSHI TAO
Whitehead Institute for Biomedical Research, Nine Cambridge Center, Cambridge, MA 02142

REFERENCES

1. B. J. Calnan, B. Tidor, S. Biancalana, D. Hudson, A. D. Frankel, *Science* **252**, 1167 (1991).