We stress that it is important to view the possible hazard of aflatoxin from the perspective of the many everyday possible hazards of life and with the knowledge that there are a great many uncertainties in the use of animal bioassay data in extrapolation to humans. As we discussed at length, the promotional aspects of cancer are also critical, and it is likely that the hazard from aflatoxin will be much lower in the absence of some toxicity in the liver such as from hepatitis virus, alcoholic cirrhosis, or the maximum tolerated dose in rodents. Since the HERP values for synthetic pollutants, including pesticides, are usually an order of magnitude less than that from aflatoxin, concern over them should be even less.

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Response: We generally agree both with Stoloff's letter and the response of Ames *et al.* However, we *were* aware that the reliability of the connection between human cancers and exposure to aflatoxin B1 has been called into question by the realization that a more important risk factor is infection with hepatitis B virus, which inevitably confounds the data. Nonetheless, we believe that the certainty for human carcinogenesis is high, although not absolute; it is certainly superior to the evidence for cancers caused by dioxin. The 20 parts-per-billion action level for aflatoxin in peanut butter may indeed have been set at a detection limit (although we do not like this practice). However, as Stoloff himself points out, it has *not* been reduced, although a modest, in our view inadequate, proposal to reduce it to 15 ppb was made in 1977 long after more sensitive detection equipment was available. The proposal was abandoned.

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Erratum: In table 1 of the article "Changes in the distribution of American family incomes, 1947 to 1984" by Frank Levy (22 May, p. 923), the first quintile (%) for 1949 was inadvertently omitted. It should have been 4.5.