

Facts Versus Fears (Third Edition)

A Review of the Greatest Unfounded Health Scares of Recent Times

By Adam J. Lieberman (1967-1997), Simona C. Kwon, M.P.H

In a way, this report is a sort of "Greatest Hits" list. But unlike a "Best of . . ." album put out to celebrate a musician's years-long body of good work, this lineup might be called a "Worst of . . ."

Since its founding in 1978 the American Council on Science and Health has been dedicated to separating real, proven health risks — such as cigarettes — from unfounded health "scares" based on questionable, hypothetical, or even nonexistent scientific evidence. This report summarizes some of the most noteworthy scares of the past half century.

In each case we review the charges made against a given product or substance — or even against an entire community. We discuss the basis for the charges, the reactions of the public and the media, and the actual facts as to what risk (if any) ever existed. Where applicable, we give an update on what the latest and most credible scientific studies have to say on each topic. The scares are presented in chronological order, arranged according to the year in which each became a major public issue.

We have chosen these scares because each received great public attention in its day — and each followed its own course to closure in terms of public and regulatory response. (For the same reason we have decided not to discuss certain current scares, such as the furor over breast implants, for which the final chapter has yet to be written.) Some of the scares examined here led to products or substances being banned. In other scares, after an initial panic, consumers shrugged off their fears.

It is interesting to note that these decisions — to ban or to forget — generally depended not on the relative magnitude of the risk but on consumers' understanding of the role the products in question played in their daily lives. In some cases a very small risk was exaggerated, or the risk was not compared with the benefits to be derived from the substance in question. In other cases the available evidence shows no risk to human health, and the people making the charges knew — or should have known — this all along.

Widespread public fears and concerns over matters of health and safety are not new to our era, of course. But what makes these particular scares unique in comparison with the panics of earlier times is that these specifically involved the products of technology, rather than the natural plagues that claimed so many lives in the past; that they were fueled by modern mass media; and that these scares emerged in a period during which Americans have enjoyed better health, an ever-increasing life span, a higher standard of living, and a greater scientific understanding of the true causes of human death and disease.

As you read this report, you will see common themes and patterns emerge in the accounts of the scares:

- the indiscriminate extrapolation of laboratory tests involving rodents fed huge doses of a given substance, with the presumption that if a substance caused cancer in these rodents, it also causes cancer in man;
- ignorance of the basic principle of toxicology, "The dose makes the poison," as consumers fretted over the presence of even a single molecule of a substance that might be hazardous in far larger amounts;
- the acceptance — implicit or explicit — of the "precautionary principle," which states: "where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent degradation." (Bear in mind here that next to no evidence can be considered "lack of scientific certainty."); and
- the fear of "synthetic" chemicals even when some of the same substances exist far more abundantly in nature.

These themes and patterns were all present in the first of our scares, the infamous 1959 "cranberry scare." They continued to pop up in almost every scare of the next three decades, and they reached their zenith with the great Alar scare of 1989.

The response to scares in the post-Alar era has been more muted. This may be due, perhaps, to public "overload" and to growing skepticism in the face of regular front-page health warnings such as the Center for Science in the Public Interest's periodic admonitions against Chinese food, Mexican food, and other popular gustatory diversions.

The purpose of this report is not, of course, merely to reflect on modern society's propensity to fear the unfamiliar. This paper is meant to serve as a cautionary tale of a different kind.

Repeated scares that focus on trivial or nonexistent risks — and the media blitzes and public panics that follow — may actually divert scarce resources away from real, significant public health risks even as they whip up needless anxiety. This report shows just how the American public has been manipulated repeatedly by certain segments of the media, by a handful of scientists outside the scientific mainstream, and by a larger coterie of activists and government regulators, all of whom have frightened the public over hypothetical risks.

This report is intended to make you, the consumer, aware of this continuing pattern. After reading Facts Versus Fears, the next time such an alarm flashes across your TV screen, you might just want to mutter, "Been there; done that" — and switch the channel.

Table of Contents

Introduction

1. The 'Cranberry Scare' of 1959
2. DDT, 1962
3. Cyclamates, 1969
4. DES in Beef, 1972

5. Nitrites, 1972
6. Red Dye Number 2, 1976
7. Saccharin, 1977
8. Hair Dyes, 1977
9. Tris, 1977
10. Love Canal, 1978
11. Three Mile Island, 1979
12. Asbestos in Hair Dryers, 1979
13. 2,4,5-T, 1979
14. Coffee and Pancreatic Cancer, 1981
15. Times Beach, 1982
16. EDB, 1983
17. Alar, 1989
18. Electric Blankets, 1989
19. Video Display Terminals, 1989
20. Benzene in Perrier, 1990
21. Amalgam Dental Fillings, 1990
22. Asbestos in Schools, 1993
23. Cellular Phones, 1993
24. Perchloroethylene in a Harlem School, 1997
25. Not-Quite-Great Unfounded Health Scares

Conclusions

F A C T S V E R S U S F E A R S :

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HEALTH SCARES OF RECENT TIMES**

by

ADAM J. LIEBERMAN (1967–1997)

SIMONA C. KWON, M.P.H.

Prepared for the American Council on Science and Health

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TABLE OF CONTENTS

Introduction	5
1. The ‘Cranberry Scare’ of 1959	6
2. DDT, 1962	7
3. Cyclamates, 1969	11
4. DES in Beef, 1972	13
5. Nitrites, 1972	14
6. Red Dye Number 2, 1976	16
7. Saccharin, 1977	18
8. Hair Dyes, 1977	20
9. Tris, 1977	21
10. Love Canal, 1978	22
11. Three Mile Island, 1979	24
12. Asbestos in Hair Dryers, 1979	27
13. 2,4,5-T, 1979	28
14. Coffee and Pancreatic Cancer, 1981	29
15. Times Beach, 1982	30
16. EDB, 1983	32
17. Alar, 1989	33
18. Electric Blankets, 1989	35
19. Video Display Terminals, 1989	37
20. Benzene in Perrier, 1990	40
21. Amalgam Dental Fillings, 1990	41
22. Asbestos in Schools, 1993	43
23. Cellular Phones, 1993	44
24. Perchloroethylene in a Harlem School, 1997	46
25. Not-Quite-Great Unfounded Health Scares	48
Conclusions	51

Introduction

H. L. Mencken once said that “the whole aim of practical politics is to keep the populace alarmed (and hence clamorous to be led to safety), by menacing it with an endless series of hobgoblins, all of them imaginary.”

Unfounded health scares for instance.

Since its founding in 1978 the American Council on Science and Health has been dedicated to separating real, proven health risks—such as cigarettes—from unfounded health “scares” based on questionable, hypothetical, or even nonexistent scientific evidence. This report summarizes the most noteworthy scares of the past half century.

In each case we review the charges made against a given product or substance—or even against an entire community. We discuss the basis for the charges, the reactions of the public and the media, and the actual facts as to what risk (if any) ever existed. Where applicable, we give an update on what the latest and most credible scientific studies have to say on each topic. The scares are presented in chronological order, arranged according to the year in which each became a major public issue.

We have chosen these scares because each received widespread public attention in its day—and each followed its own course to closure in terms of public and regulatory response. For the same reason we have decided not to discuss certain current scares, such as the furor over breast implants, for which the final chapter has yet to be written. Some of the scares examined here led to products or substances being banned. Some led to financial and economic disasters for the producers and processors of the falsely accused products. In other cases, after an initial panic, consumers shrugged off their fears.

It is interesting to note that the decisions to ban or to forget generally depended not on the relative magnitude of the risk but on the perceived role the products in question played in consumers’ daily lives. In some cases a very small risk was exaggerated, or the risk was not compared with the benefits to be derived from the substance in question. In other cases the available evidence showed no risk to human health, and the people making the charges knew—or should have known—this all along.

Widespread public fears and concerns over matters of health and safety are not new to our era, of course. But what makes these particular scares unique in comparison with the panics of earlier times is that these specifically involved the products of technology,

rather than the natural plagues that claimed so many lives in the past. Often initiated by “environmental” or “consumer” organizations and fueled by modern mass media, these scares emerged at a time when Americans enjoyed better health, an ever-increasing life span, a higher standard of living, and a greater scientific understanding of the causes of human death and disease.

As you read this report, you will see common themes and patterns emerge in the accounts of the scares:

- The indiscriminate presumption that the results of laboratory tests involving rodents force-fed (usually via stomach tubes) huge doses of a given substance can be extrapolated to show that the tested substance causes cancer in humans.
- Ignorance of the basic principle of toxicology, “The dose makes the poison,” as consumers fret over the presence of even a single molecule of a substance that is not hazardous to humans unless it is consumed in large amounts.
- The acceptance—implicit or explicit—of the United Nations—conceived “precautionary principle,” which states, “where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent degradation.”¹ In other words, all that matters is whether a substance or a technology *may* do harm. If the risk of harm cannot be ruled out, then the risky product or activity should not be permitted.
- The fear of “synthetic” chemicals, even when some of those same substances exist abundantly, without causing harm, in nature.

These themes and patterns were all present in the first of our scares, the infamous “cranberry scare” of 1959. And they continued to pop up in almost every scare of the next three decades, reaching their zenith with the great Alar scare of 1989.

The response to scares in the post-Alar era has been more muted. This may be due to public “overload” and to growing skepticism in the face of regular front-page health warnings, such as the Center for Science in the Public Interest’s periodic admonitions against Chinese food, movie-theater popcorn, and other popular gustatory diversions.

The purpose of this report is not, of course, mere-

ly to reflect on modern society's propensity to fear the unfamiliar. This collection of scare stories is meant to serve as a cautionary tale of a different kind.

Scares that focus on trivial or nonexistent risks—and the media blitzes and public panics that follow—may serve to divert scarce resources away from real, significant public health risks. In this report we intend to show just how the American public has been manipulated by certain segments of the media, by a handful of scientists outside the scientific mainstream, and by a larger coterie of activists and government regulators, all of whom have, whether intentionally or not, frightened the public with hypothetical risks.

What we need is responsible, balanced, scientific reporting. Journalists must become more knowledgeable about science (and scientific reporting) so that they—and we—can avoid the sort of media blitzes and scares discussed in this report. In short, if we are to achieve the goal of providing consumers with an understanding of science—and of the real health risks they can incur—we need to bridge the gap between journalism and science.

It is ACSH's hope that after reading this report, you, as a consumer, will carefully consider the next headlined scare that comes along; that you will stop to determine critically whether the headline describes a real or a trivialized risk.

Remember that the worries such headlines can cause may be more dangerous than the "risks" themselves. Remember, too, that contrary to the assertions of the hobgoblins, technology is making our world safer.

¹ United Nations Conference on Environment and Development (UNCED). Declaration of Rio. Rio de Janeiro, Brazil: UN 1992 (Principle 15).

1. The 'Cranberry Scare' of 1959

Background

Aminotriazole, a weed killer, was first used on cranberry crops in 1957. Because the chemical had not yet been approved for use on crops, growers withheld 30,000 barrels of cranberries found to contain aminotriazole residue. The following year, the chemical was approved. Testing by the U.S. Food and Drug Administration (FDA) showed, however, that when aminotriazole was fed to rats in concentrations of 100 parts per million in the diet, it produced cancer of the thyroid. Although this dose was the equivalent of a human's ingesting 15,000 pounds of berries every day for a number of years, the FDA restricted aminotriazole

in cranberry bogs to postharvest use.¹ No residues were found during 1958.²

The Scare

On November 9, 1959, Secretary of Health, Education, and Welfare Arthur Fleming announced that a consignment of berries from Oregon examined by the San Francisco office of the FDA had been found to be contaminated with aminotriazole. Fleming warned that other berries from Oregon and Washington—9 percent of the crop—might also be contaminated. He added that berries from other states—Massachusetts, Wisconsin, and New Jersey—showed no evidence of contamination. But when asked by a reporter whether "a housewife" could be sure of the safety of the cranberries she was buying, Fleming replied, "To be on the safe side, she doesn't buy."¹

The Reaction

Fleming's comment—which came just 15 days before Thanksgiving—set off a full-fledged panic. State health officials in Ohio and city authorities in San Francisco and Chicago banned cranberry sales. The states of Michigan, Kentucky, and Washington called for voluntary suspensions. Supermarkets and restaurants in New York and other cities pulled products and dishes containing cranberries off their shelves and menus.³ A nightclub in Chicago even set a one-to-a-customer limit on cranberry cocktails.⁴

Cranberry growers agreed to join the FDA in searching for aminotriazole contamination⁵ but were nonetheless furious at Fleming for his comments. The growers demanded an apology, and the Massachusetts Farm Bureau called for Fleming's resignation.³ Wary of the effect of the scare, other government officials began to backtrack: Secretary of Agriculture Ezra Taft Benson announced that he would have cranberries on his Thanksgiving table.⁶ Even the candidates for the 1960 Presidential election got into the act. At campaign stops in Wisconsin, Vice President Richard Nixon ate four helpings of cranberry sauce and Senator John F. Kennedy downed two glasses of cranberry juice.¹

Cans of cranberry sauce reappeared on supermarket shelves in time for Thanksgiving, complete with labels assuring buyers that the fruit had been inspected and approved. Fleming himself promised to have cranberries on his holiday table.⁷ Cranberry growers had initially feared the total loss of the \$45 million to \$50 million revenue expected from Thanksgiving cranberry sales⁵—60 percent of annual sales⁸—but the actual loss was apparently much less. At least one U.S. senator suggested that the government reimburse growers for any

losses,⁹ but no such action was taken.

Conclusion

As noted, any risk from aminotriazole was infinitesimal at best, given the enormous amounts of it fed to rats in the tests that resulted in the FDA's declaring it a carcinogen. Additionally, as with many substances that are rodent carcinogens, any hypothetical harm done by aminotriazole is dwarfed by that of far more potent naturally occurring carcinogens. In this case Dr. Edwin Astwood, a professor of medicine at Tufts University, noted that certain turnips naturally contained 100 times as much antithyroid potency as did any cranberries contaminated with aminotriazole.¹⁰

The New York Times (among others) declared early on that Fleming "went too far" in provoking an unnecessary panic. *The Times* noted that even if humans should prove to be as susceptible to the chemical as rats, people would have to consume fantastic quantities of contaminated berries to suffer any ill effects.¹¹ These attempts to put the matter into perspective were ignored by the wider public, however.

Even in those days, the public suffered from chemophobia. One newspaper article noted the influence of "wildlife and conservation groups and . . . pure-food enthusiasts, who believe that chemical residues on agricultural products pose a threat to health."¹² The most important influence, though, was that of the Delaney clause, which had been passed as an amendment to the Federal Food, Drug, and Cosmetic Act the preceding year. It was the Delaney clause that first codified the "mouse-as-little-man" principle: the premise that any substance that causes cancer in rodents at extraordinarily high doses will also cause cancer in humans at more moderate doses.¹³ As one report noted, the Delaney clause tied the FDA's hands. The amendment prevented the FDA from "consider[ing] any food safe if it contains even the smallest amount of a substance (specifically, an additive: the Delaney Clause was not applied to substances naturally occurring in foods) which tests have shown will produce cancer in test animals."⁸

"Noncontaminated" cranberries soon returned to kitchen tables across America, but a precedent had been set: The public had been taught to fear trace amounts of chemicals regardless of the actual human health risk.

And this boggy little brouhaha laid the groundwork for scares yet to come: It paved the way for many of the other scares discussed in this report.

¹ Cranberry affair. *Newsweek*. November 23, 1959:35.

² U.S. widens taint check for cranberries. *The New York Times*. November 11, 1959:1.

³ Cranberry crop facing huge loss. *The New York Times*. November 11, 1959:1.

⁴ Mercy Ma! No cranberries. *Life*. November 23, 1959:28.

⁵ Weed-killer testing of cranberry crop aided by growers. *The New York Times*. November 12, 1959:1.

⁶ Benson won't abandon cranberries on holiday. *The New York Times*. November 11, 1959:29.

⁷ Cranberries, please. *Newsweek*. November 30, 1959:27.

⁸ The cranberry scare—here are the facts. *U.S. News and World Report*. November 23, 1959:44–45.

⁹ Williams in protest. *The New York Times*. November 12, 1959:20.

¹⁰ Hayes W. *Pesticides Studied in Man*. Baltimore: Williams & Wilkins; 1982:566.

¹¹ Cranberries and Mr. Fleming. *The New York Times*. November 14, 1959:44.

¹² Pesticide scare worries manufacturers. *The New York Times*. November 22, 1959:1.

¹³ Jukes TH. Chasing a receding zero: impact of the zero threshold concept on actions of regulatory officials. *J Amer Coll Toxicol*. 1983; 2(3):147–160.

2. DDT, 1962

Background

DDT (dichlorodiphenyltrichloroethane) was first synthesized in 1877,¹ but it was not until 1940 that a Swiss chemist discovered that it could be sprayed on walls and would cause any insect to die within the next six months, without any apparent toxicity to humans.² DDT's effectiveness, persistence, and low cost (only 17 cents per pound) resulted in its being used in antimalarial efforts worldwide. It was introduced into widespread use during World War II and became the single most important pesticide responsible for maintaining human health through the next two decades. The scientist who discovered the insecticidal properties of DDT, Dr. Paul Müller, was awarded the 1948 Nobel Prize in Physiology and Medicine.³

The Scare

In 1962 Rachel Carson's lyrical yet scientifically flawed book *Silent Spring* was released. The book argued eloquently but erroneously that pesticides, and especially DDT, were poisoning both wildlife and the environment and also endangering human health.

The emotional public reaction to *Silent Spring* launched the modern environmental movement.⁴ DDT became the prime target of the growing anti-chemical and anti-pesticide movements during the 1960s. Reasoned scientific discussion and sound data on the favorable human health effects of DDT were brushed aside by environmental alarmists who discounted DDT's enormous benefits to world health with two allegations: (1) DDT was a carcinogen, and (2) it endangered the environment, particularly for certain birds.

In 1969 a study found a higher incidence of leukemia and liver tumors in mice fed DDT than in unexposed mice.⁵ Soon, too, environmentalists were blaming the decline in populations of such wild bird species as the osprey and peregrine falcon on the contamination by DDT of their environment. A number of states moved to ban DDT, and in 1970 the U.S. Department of Agriculture announced a plan to phase out all but essential uses.⁶

The Reaction

Numerous scientists protested that the laboratory-animal studies flew in the face of epidemiology, given that DDT had been used widely during the preceding 25 years with no increase in liver cancer in any of the populations among whom it had been sprayed. And when the World Health Organization (WHO) investigated the 1969 mice study, scientists discovered that both cases and controls had developed a surprising number of tumors. Further investigation revealed that the foods fed to both mice groups were moldy and contained aflatoxin, a carcinogen.⁷ When the tests were repeated using noncontaminated foods, neither group developed tumors. In 1970 the National Academy of Sciences declared, "In little more than two decades, DDT has prevented 500 million human deaths due to malaria, that would otherwise have been inevitable."⁸

Additionally, the evidence regarding the effect of DDT on eggshell thinning among wild birds is contradictory at best. The environmentalist literature claims that the birds threatened directly by the insecticide were laying eggs with thin shells. These shells, say the environmentalists, would eventually become so fragile that the eggs would break, causing a decline in bird populations, particularly among raptors (birds of prey).

In 1968 two researchers, Drs. Joseph J. Hickey

and Daniel W. Anderson, reported that high concentrations of DDT were found in the eggs of wild raptor populations. The two concluded that increased eggshell fragility in peregrine falcons, bald eagles, and ospreys was due to DDT exposure.⁹ Dr. Joel Bitman and associates at the U.S. Department of Agriculture likewise determined that Japanese quail fed DDT produced eggs with thinner shells and lower calcium content.¹⁰

In actuality, however, declines in bird populations either had occurred before DDT was present or had occurred years after DDT's use. A comparison of the annual Audubon Christmas Bird Counts between 1941 (pre-DDT) and 1960 (after DDT's use had waned) reveals that at least 26 different kinds of birds became more numerous during those decades, the period of greatest DDT usage. The Audubon counts document an overall increase in birds seen per observer from 1941 to 1960, and statistical analyses of the Audubon data confirm the perceived increases. For example, only 197 bald eagles were documented in 1941¹¹; the number had increased to 891 in 1960.¹²

At Hawk Mountain, Pennsylvania, teams of ornithologists made daily counts of migrating raptors for over 40 years. The counts—published annually by the Hawk Mountain Sanctuary Association—reveal great increases in most kinds of hawks during the DDT years. The osprey counts increased as follows: in 1946, 191; in 1956, 288; in 1967, 457; and in 1972, 630.¹³ In 1942 Dr. Joseph Hickey—who in 1968 would blame DDT for bird population decline—reported that 70 percent of the eastern osprey population had been killed by pole traps around fish hatcheries.¹⁴ That same year, before DDT came into use, Hickey noted a decline in the population of peregrine falcons.¹⁵

Other observers also documented that the great peregrine decline in the eastern United States occurred long before any DDT was present in the environment.^{16,17} In Canada peregrines were observed to be "reproducing normally" in the 1960s even though their tissues contained 30 times more DDT than did the tissues of the midwestern peregrines allegedly being extirpated by the chemical.¹⁸ And in Great Britain, in 1969, a three-year government study noted that the decline of peregrine falcons in Britain had ended in 1966 even though DDT levels were as abundant as ever. The British study concluded that "There is no close correlation between the decline in population of predatory birds, particularly the peregrine falcon and the sparrow hawk, and the use of DDT."¹⁹

In addition, later research refuted the original studies that had pointed to DDT as a cause for eggshell thinning. After reassessing their findings using more

modern methodology, Drs. Hickey and Anderson admitted that the egg extracts they had studied contained little or no DDT and said they were now pursuing PCBs, chemicals used as capacitor insulators, as the culprit.²⁰

When carefully reviewed, Dr. Bitman's study revealed that the quail in the study were fed a diet with a calcium content of only 0.56 percent (a normal quail diet consists of 2.7 percent calcium). Calcium deficiency is a known cause of thin eggshells.²¹⁻²³ After much criticism, Bitman repeated the test, this time with sufficient calcium levels. The birds produced eggs without thinned shells.²⁴

After many years of carefully controlled feeding experiments, Dr. M. L. Scott and associates of the Department of Poultry Science at Cornell University "found no tremors, no mortality, no thinning of eggshells and no interference with reproduction caused by levels of DDT which were as high as those reported to be present in most of the wild birds where 'catastrophic' decreases in shell quality and reproduction have been claimed."²³ In fact, thinning eggshells can have many causes, including season of the year, nutrition (in particular insufficient calcium, phosphorus, vitamin D, and manganese), temperature rise, type of soil, and breeding conditions (e.g., sunlight and crowding).²⁵

In the years preceding the DDT ban, the National Academy of Sciences,^{26,27} the American Medical Association, the U.S. Surgeon General,²⁸ the World Health Organization,²⁹ and the Food and Agriculture Organizations of the United Nations³⁰ had been among those who spoke out in support of the continued use of DDT as a disease fighter and crop protectant.

In 1971 authority over pesticides was transferred from the Department of Agriculture to the newly formed Environmental Protection Agency (EPA). In April 1972, after seven months of testimony, Judge Edmund Sweeney stated that "DDT is not a carcinogenic hazard to man. . . . The uses of DDT under the regulations involved here do not have a deleterious effect on freshwater fish, estuarine organisms, wild birds, or other wildlife. . . . The evidence in this proceeding supports the conclusion that there is a present need for the essential uses of DDT."³¹

Two months later EPA head William Ruckelshaus—who had never attended a single day's session in the seven months of EPA hearings, and who admittedly had not even read the transcript of the hearings—overturned Judge Sweeney's decision. Ruckelshaus declared that DDT was a "potential human carcinogen" and banned it for virtually all uses.³²

Conclusion

The ban on DDT was considered the first major victory for the environmentalist movement in the U.S. The effect of the ban in other nations was less salutary, however. In Ceylon (now Sri Lanka) DDT spraying had reduced malaria cases from 2.8 million in 1948 to 17 in 1963. After spraying was stopped in 1964, malaria cases began to rise again and reached 2.5 million in 1969.³³ The same pattern was repeated in many other tropical—and usually impoverished—regions of the world. In Zanzibar the prevalence of malaria among the populace dropped from 70 percent in 1958 to 5 percent in 1964. By 1984 it was back up to between 50 and 60 percent. The chief malaria expert for the U.S. Agency for International Development said that malaria would have been 98 percent eradicated had DDT continued to be used.³⁴

In addition, from 1960 to 1974 WHO screened about 2,000 compounds for use as antimalarial insecticides. Only 30 were judged promising enough to warrant field trials. WHO found that none of those compounds had the persistence of DDT or was as safe as DDT. (Insecticides such as malathion and carbaryl, which are much more toxic than DDT, were used instead.) And—a very important factor for malaria control in less developed countries—all of the substitutes were considerably more expensive than DDT.³⁵

And what of the charges leveled against DDT? A 1978 National Cancer Institute report concluded—after two years of testing on several different strains of cancer-prone mice and rats—that DDT was not carcinogenic.³⁶ As for the DDT-caused eggshell thinning, it is unclear whether it did, in fact, occur and, if it did, whether the thinning was caused by DDT, by mercury, by PCBs, or by the effects of human encroachment.^{16,37} And as recently as 1998 researchers reported that thrush eggshells in Great Britain had been thinning at a steady rate 47 years before DDT hit the market; the researchers placed the blame on the early consequences of industrialization.³⁸

Regardless of whether DDT, exclusive of other chemicals, presented a threat to bird populations, it remains in the news. DDT has a long half-life, and residues sometimes persist for years in certain environments. Also, DDT is an organochlorine. Some organochlorines have been shown to have weak estrogenic activity, but the amounts of naturally occurring estrogens in the environment dwarf the amounts of synthetic estrogens.³⁹ A recent article in the journal *Environmental Health Perspectives* suggested that the ratio of natural to synthetic estrogens may be as much as 40,000,000 to 1.⁴⁰

In addition, Dr. Robert Golden of Environmental Risk Studies in Washington, DC, reviewed the research of numerous scientists and concluded that DDT and DDE (a breakdown product of DDT) have no significant estrogenic activity.⁴¹

The 1996 book *Our Stolen Future* speculated on a link between DDT and breast cancer, noting that DDE has been found in some breast tumors.⁴² Recently, charges have been made associating DDT and DDE with breast cancer—specifically, the finding that women with breast cancer had higher levels of DDE in their blood than did women without breast cancer.⁴³ However, elevated blood DDE could quite plausibly be a result of the mobilization of fat from storage depots in the body due to weight loss associated with breast cancer. Breast cancer thus may be a risk factor for elevated DDE, rather than DDE's being a risk factor for breast cancer.⁴⁴

In a 1994 study published in the *Journal of the National Cancer Institute*, researchers concluded that their data did not support an association between DDT and breast cancer.⁴⁵ The researchers did note that breast cancer rates are higher than the national average in many places in the northeastern United States; but the data also indicated that the higher levels could be accounted for by nonenvironmental factors among women living in these regions—factors such as higher socioeconomic status and deferral or avoidance of pregnancy, both of which increase the risks of breast cancer by up to twofold.^{45,46}

In October 1997 the *New England Journal of Medicine* published a large, well-designed study that found no evidence that exposure to DDT and DDE increases the risk of breast cancer.⁴⁷ In the accompanying editorial Dr. Steven Safe, a toxicologist at Texas A&M University, stated, “weakly estrogenic organochlorine compounds such as PCBs, DDT, and DDE are not a cause of breast cancer.”⁴⁸ Dr. Sheila Zahm, deputy chief of the occupational epidemiology branch at the National Cancer Institute, agrees that the body of evidence that DDT can cause breast cancer “is not very compelling.”⁴⁹

⁴ *Toxic Terror*:96.

⁵ Tarjan R, Kemeny T. Multigeneration studies on DDT in mice. *Food Cosmet and Toxicol.* 1969; 7:14–222.

⁶ *Toxic Terror*:110–114.

⁷ Edwards JG. Testimony and affidavit: California Department of Food and Agriculture. Los Angeles, CA. March 1978.

⁸ National Academy of Sciences. *The Life Sciences.* Washington, DC: National Academy of Sciences Press; 1970.

⁹ Hickey JJ, Anderson DW. Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. *Science.* 1968; 162:271–273.

¹⁰ Bitman J, Cecil HC, Harris SJ, Gries GF. DDT induces a decrease in eggshell calcium. *Nature.* 1969; 224:44–46.

¹¹ The 42nd Annual Christmas Bird Census. *Audubon Magazine.* 1942; 44:1–75.

¹² The 61st Annual Christmas Bird Census. *Audubon Field Notes.* 1961; 15(2): 84–300.

¹³ Taylor JW. Summaries of Hawk Mountain migration of raptors, 1934 to 1970. *Hawk Mtn Assn Newsletter.* 1970; 42.

¹⁴ Hickey JJ. *Guide to Bird Watching.* Madison, WI: University of Wisconsin Press; 1943.

¹⁵ Hickey JJ. Only 170 pairs of peregrines in eastern U.S. in 1940, before DDT. *Auk.* 1942; 59:176.

¹⁶ Beebe FL. *The Myth of the Vanishing Peregrine.* North Surrey, BC, Canada: Canadian Raptor Society Press; 1971.

¹⁷ Rice JN. *Peregrine Falcon Populations.* Madison, WI: University of Wisconsin Press; 1969:155–164.

¹⁸ Enderson JH, Berger DD. Chlorinated hydrocarbons in peregrines from northern Canada. *Condor.* 1968; 70:149–153.

¹⁹ Wilson Report. *Review of organochlorine pesticides in Britain.* Report by Advisory Committee on Toxic Chemicals. Department of Education and Science; 1969.

²⁰ Anderson DW, Hickey JJ, Risebrough RW, Hughes DF, Christensen RE. Significance of chlorinated hydrocarbon residues to breeding pelicans and cormorants. *The Canadian Field-Naturalist.* 1969; 83:91–112.

²¹ Greely F. Effects of calcium deficiency. *J Wildlife Management.* 1960; 70:149–153.

²² Romanoff AL, Romanoff AJ. *The Avian Egg.* New York: Wiley & Sons; 1949:154.

²³ Scott ML, Zimmermann JR, Marinsky S, Mullenhoff PA. Effects of PCBs, DDT, and mercury compounds upon egg production, hatchability and shell quality in chickens and Japanese quail. *Poultry Science.* 1975; 54:350–368.

¹ Whelan E. *Toxic Terror.* 2nd ed. Buffalo, NY: Prometheus Books; 1993:100.

² Desowitz R. *The Malaria Capers: More Tales of Parasites and People, Research and Reality.* New York: W.W. Norton; 1991:62–63.

³ Edwards JG. *Pesticides in Medicine & Politics.* Prepared address to Doctors for Disaster Preparedness. San Diego, CA. June 14, 1997.

- 24 Cecil HC, Bitman J, Harris SJ. No effects on eggshells, if adequate calcium is in DDT diet. *Poultry Science*. 1971; 50:656–659.
- 25 *The Avian Egg*:152–156, 266.
- 26 Handler P. The federal government and the scientific community. *Science*. 1971; 171(967):144–151.
- 27 *Toxic Terror*:112.
- 28 Steinfeld JL. Surgeon General testimony before EPA. September 9, 1971.
- 29 The place of DDT in operations against malaria & other vector-borne disease. *Official Records, WHO*, Geneva: No. 190, April 1971:176–182.
- 30 Special Report of United Nations Food and Agriculture Organization. *The New York Times*. November 29, 1969.
- 31 Sweeney EM. EPA Hearing Examiner's recommendations and findings concerning DDT hearings. 25 April 1972 (40 CFR 164.32).
- 32 Ackerly RL. DDT: a re-evaluation, part II. *Chemical Times and Trends*. October 1981:55.
- 33 Gerberg EJ, Wilcox III H. *Environmental Assessment of Malaria and Control Projects—Sri Lanka*. Agency for International Development. 1977; 20:32–33.
- 34 Bast J, Hill P, Rue R. *Eco-Sanity: A Common Sense Guide to Environmentalism*. Lanham, MD: The Heartland Institute; 1994:100–101.
- 35 Service MW. Some problems in the control of malaria. In: *Ecological Effects of Pesticides* (Perring, FH and Mellanby K, eds). New York: Academic Press; 1977:156.
- 36 Efron E. *The Apocalyptic*. New York: Touchstone/Simon & Schuster; 1985:268.
- 37 Jukes TH. Insecticides in health, agriculture and the environment. *Die Naturwissensch*. 1974; 61:6–16.
- 38 Milius S. Birds' eggs started to thin long before DDT. *Science News*. 1988; 153(17):261.
- 39 Jukes TH. Chasing a receding zero: impact of the zero threshold concept on actions of regulatory officials. *J Amer Coll Toxicol*. 1983; 2(3):147–160.
- 40 Safe S. Environmental and dietary estrogens and human health: is there a problem? *Environ Health Perspect*. 1995; 103:346–351.
- 41 Golden, R. *Proceedings of the International Environmental Conference*, Washington, DC, 1995.
- 42 Colborn T, Dumanoski D, Myers JP. *Our Stolen Future*. New York: Dutton. 1996:184.
- 43 National Cancer Institute. DDT and Breast Cancer. *NCI CancerFax*. National Cancer Institute Office of Cancer Communications; August 1996:1.
- 44 Ottoboni MA. Personal communication with Dr. N. Snyderman, 1993.
- 45 Krieger N, Wolff MS, Haitt RA, et al. Breast cancer and serum organochlorines: a prospective study among white, black, and Asian women. *JNCI*. April 20, 1994.
- 46 Sturgeon SR, Schairer C, Gail M, McAdams M, Brinton LA, Hoover RN. Geographic variation in mortality from breast cancer among white women in the United States. *JNCI*. December 20, 1995.
- 47 Hunter DJ, Hankinson SE, Laden F, Colditz G, Manson JE, Willett WC, Speizer FE, Wolff MS. Plasma organochlorines levels and the risk of breast cancer. *N Engl J Med*. 1997; 337:1253–1258.
- 48 Safe S. Xenoestrogens and breast cancer. *N Engl J Med*. 1997; 337:1303–1304.
- 49 Kolata G. Study discounts DDT role in breast cancer. *The New York Times*. October 10, 1997:A26

3. Cyclamates, 1969

Background

Cyclamates (salts of cyclamic acid) are synthetic, nonnutritive sweeteners used as a sugar substitute. They were discovered by accident by a researcher in 1937 and approved by the FDA as a drug in 1951. In 1958 they were reclassified as a food additive; at the time, based on their past history of safe use, the FDA declared them to be GRAS (generally recognized as safe) and thus exempt from regulation under the Food, Drug, and Cosmetic Act.¹

Cyclamates were originally intended only for the use of the obese and diabetics; but as worrying over excess pounds became a national concern in the 1960s, the use of cyclamates grew dramatically. They were used in everything from soft drinks and candy to canned fruits and salad dressings.² Between 1963 and 1970 national consumption of cyclamates rose from 5 million to 21 million pounds.³

The Scare

In the late 1960s FDA experiments showed that cyclohexamine, a byproduct of cyclamates, caused chromosome damage in male rats⁴; and in June 1969 a study found that some white Swiss mice developed tumors when cyclamate was implanted in their bladders.⁵ In both cases, however, the FDA said the route of

administration was inappropriate to draw any conclusion—that is, the afflicted rats were exposed in a way that was not comparable to the route of human exposure: ingestion in the diet.

Then, in October 1969, FDA scientist Dr. Jacqueline Verrett appeared on the NBC evening news declaring that baby chicks injected with cyclamate as embryos had suffered gross malformations—and displaying the deformed birds to the national television audience.⁶ Both FDA Commissioner Dr. Herbert Ley, Jr., and HEW Secretary Robert Finch criticized Dr. Verrett for going to the media before subjecting her findings to peer-reviewed scrutiny, and both men defended the safety of cyclamate.³

A few days later Abbott Laboratories, the manufacturer of cyclamate, released a study showing that 8 out of 240 rats fed a mixture of ten parts cyclamate to one part saccharin (the mixture most often used in food products) developed bladder tumors.⁷ As with all tests of this type, the rats were ingesting a dosage far higher than that of equivalent human consumption; in this case, it was the equivalent of 350 cans of diet soda per day.

On October 18, 1969, HEW Secretary Robert Finch declared that under the Delaney clause he was obliged to remove cyclamate from the market.¹ The following year the sale of cyclamate as a prescription product to dieters and diabetics was also banned.⁸

The Reaction

Except for the manufacturers of cyclamate-containing products, one of whom was stuck with \$31.5 million worth of unusable canned fruits,⁸ the public reaction to the cyclamate ban was generally positive. Even though, at the time, nearly 75 percent of Americans used cyclamate in one product or another,⁹ nearly 80 percent of the public felt “gratitude for the government protection.” At a time when the “back-to-nature” movement in general was picking up steam, the ban dovetailed nicely with the idea that anything “artificial” was dangerous. Many scientific publications, however, were critical of the FDA for acting so precipitously.¹⁰

And studies of cyclamates continued. Subsequent, large-scale tests on rodents failed to duplicate the results of the 1969 studies; none of the new studies showed any tumors that could be linked to cyclamates.^{11–15} As a result, numerous scientific bodies—among them the National Cancer Institute,¹⁶ the United Nations Food and Agriculture Organization (FAO), the World Health Organization (WHO),¹⁷ the FDA’s Center for Food Safety and Applied Nutrition,¹⁸

and the National Research Council of the National Academy of Sciences¹⁹—have all declared that that evidence shows cyclamates not to be carcinogenic.

As a result of these studies, Abbott Laboratories has on several occasions petitioned the FDA to revoke the ban on cyclamates; each time, the petition has been rejected. Cyclamates are once again available in Canada and in the nations of the European Community (EC), however; the EC, FAO, and WHO have set an acceptable daily intake of 11 milligrams per kilogram of body weight per day.²⁰

Conclusion

Experiments must be subject to peer review and be reproducible to be considered valid. In the case of the studies indicting cyclamates, neither criterion was met. All subsequent studies have drawn conclusions contrary to those that suggested that cyclamates were carcinogens.

The loss of cyclamates did not cause much of an uproar, both because of the mood of the times and because most consumers quickly adjusted by switching to saccharin—which did not become the subject of its own scare until 1977 (see page 18).

¹ Jukes TH. Cyclamate sweeteners. *JAMA*. 1976; 236(17):1987–1989.

² Whelan E, Stare FJ. *Panic in the Pantry*. New York: Atheneum; 1975:151–152.

³ Bitterness about sweets. *Time*. October 17, 1969:79.

⁴ Bitter sweeteners? *Newsweek*. September 29, 1969:83.

⁵ Osler BL, et al. Chronic toxicity study of cyclamate: saccharin (10:1) in rats. *Toxicol*. 1975; 4:315–330.

⁶ *Panic in the Pantry*. 1975:154–155.

⁷ Price JM, et al. Bladder tumors in rats fed cyclohexylamine or high doses of a mixture of cyclamate and saccharin. *Science*. 1970; 167:1131–1132.

⁸ FDA extends ban on sweeteners. *Science*. September 4, 1970:962

⁹ *Panic in the Pantry*. 1975:155

¹⁰ *Panic in the Pantry*. 1975:160.

¹¹ Roe FJ, et al. Feeding studies on sodium cyclamate, saccharin and sucrose for carcinogenic and tumor-promoting activity. *Food Cosmet Toxicol*. 1970; 8(2):135–145.

¹² Brantom PG, et al. Long-term toxicity of sodium cyclamate in mice. *Food Cosmet Toxicol*. 1973; 11(5):735–746.

- ¹³ Hardy J, et al. Long-term toxicity of cyclohexylamine hydrochloride in mice. *Food Cosmet Toxicol.* 1976; 14(4):269–276.
- ¹⁴ Kroes R, et al. Long-term toxicity and reproduction study (including a teratogenicity study) with cyclamate, saccharin and cyclohexylamine. *Toxicol.* 1977; 8(3):285–300.
- ¹⁵ Taylor JM, et al. Chronic toxicity and carcinogenicity to the urinary bladder of sodium saccharin in the in utero exposed rat. *Toxicol Appl Pharmacol.* 1980; 54(1):57–75.
- ¹⁶ *Panic in the Pantry.* Rev ed. Buffalo, NY: Prometheus Books; 1992:145.
- ¹⁷ Joint Expert Committee on Food Additives of the Food and Agriculture Organization and the World Health Organization. *Evaluation of Certain Food Additives: Eighteenth Report.* 1974.
- ¹⁸ Cancer Assessment Committee, Center for Food Safety and Applied Nutrition, FDA. *Scientific review of the long-term carcinogen bioassays performed on the artificial sweetener, cyclamate.* April 1984.
- ¹⁹ Committee on the Evaluation of Cyclamate for Carcinogenicity, National Research Council. *Evaluation of Cyclamate for Carcinogenicity.* Washington, DC: National Academy Press; 1985.
- ²⁰ Meister K. *Low-Calorie Sweeteners.* New York: American Council on Science and Health; 1993.

4. DES in Beef, 1972

Background

DES (diethylstilbestrol) is a synthetic form of estrogen first approved by the FDA in 1938 as a medication to prevent miscarriages and other pregnancy complications. In 1954 it was approved by the FDA as a cattle growth stimulant. DES shortens by 34 days the time required to bring a 500-pound animal to a marketable weight of 1,000 pounds. An animal thus treated requires 500 fewer pounds of feed than otherwise. Treatment with DES also increases by 7 percent the amount of protein and moisture in an animal's meat while decreasing the percentage of fat.¹

All estrogens are animal carcinogens. Therefore, when the Delaney clause was passed in 1958, the use of DES as a cattle growth stimulant was permitted only so long as no DES residue could be detected in meat.¹ Criticisms had issued from some quarters almost from the beginning of DES's use as a cattle growth stimulant, but during the 1950s these were largely confined to publications such as the *National Police Gazette*, a forerunner of today's supermarket tabloids (a typical *Police*

Gazette headline: "Poison by the Plateful").² Hearings were held on the safety and efficacy of DES during the 1950s and early 1960s, but those hearings were largely driven by a rivalry between two different business–university partnerships holding patents for two different means of DES administration.³

The Scare

In 1970 an Associated Press story charged that FDA officials were permitting DES residues "known to incite cancer . . . in violation of federal law" and that cattlemen were routinely ignoring the law requiring them to withdraw DES from cattle 48 hours prior to slaughter.⁴ The AP report sparked wide public reaction, and both government and industry vowed to enforce the laws more stringently.⁵

Then, in April 1971, a study in the *New England Journal of Medicine* reported incidences of clear cell adenocarcinoma, a rare form of vaginal cancer, in women aged 14 to 22. This type of cancer had been virtually unknown among women of this age group, and the study concluded that what all these women had in common was that their mothers had taken DES at doses of as much as 125 milligrams daily during the first trimester of pregnancy.^{6–8} (At the time, DES was the only treatment known to enable some women to carry a pregnancy to term.)

In November 1971 the FDA banned the use of DES during pregnancy but still allowed its use as a growth stimulant in cattle. Therefore, the issue of the far more minuscule amounts of DES used in cattle remained.

The Reaction

By the end of 1971 the FDA had extended the preslaughter DES withdrawal period to seven days.⁹ On August 2, 1972, after a special Senate hearing in which Senator Edward Kennedy described DES as a "known cancer-causing agent . . . on thousands of American dinner tables,"¹⁰ the FDA banned the use of DES in feed, noting that highly sensitive tracers measured 0.2 parts per trillion of the drug a week after ingestion.¹¹ The following April, after investigations found up to 0.7 parts per trillion in cattle livers 120 days after DES implantation, the use of DES implants was likewise banned.¹²

More than any other health or food issue at the time, the DES ban pointed up the dichotomy between public perceptions and the views of scientists. Self-styled consumer advocates formed groups with names such as the "Committee to Get the Drugs Out of Meat" (DOOM) and wrote letters to the FDA declaring that the DES ban would be worthwhile even if it raised prices:

“People would rather be broke (or vegetarian) than DEAD,” stated one such letter. *Sowing the Wind*, a book by a Nader activist accusing the FDA of negligence, reached the best-seller list.¹³

Scientists, meanwhile, sought to explain how estrogens occur naturally in milk, honey, eggs, and other substances—as well as in approved pharmaceuticals such as the birth-control pill—at levels thousands to millions of times higher than those found in the livers of DES-treated cattle. Scientists also pointed out that a woman would have to eat 62.5 tons of beef liver to equal the 125-milligram dosage received by the DES-treated mothers and that the human female (and male) body naturally produces estrogens—again at levels far higher than those found in cattle.¹⁴

Furthermore, the scientists explained, at a time when world hunger was a matter of great public concern, to ban DES would cost an extra seven million tons of corn per year. And, they added, the use of DES may actually decrease the risk of cancer by producing leaner beef.

But most of these protestations fell on deaf ears—or prompted rejoinders from zealots that the DES proponents “were condemning a number of young ladies to death [so that] beef will cost a few cents less.” This sort of thing silenced most of the critics in the political arena,¹⁵ and the subsequent legislative and court efforts to overturn the DES ban failed.

The FDA issued its final ban on DES in June 1979; the following year investigators turned up massive illegal use of DES by ranchers throughout the nation.¹⁶

Conclusion

The DES ban illustrates a basic fallacy held by both the regulatory community and much of the public: the belief that if a substance has an adverse effect in one instance (as DES did in pregnant women and their female offspring) that substance must therefore be dangerous in all instances. The belief in that fallacy, combined with a general fear of “artificial” substances and with what one historian would later describe as the “politicizing of the regulatory process and loss of special status for scientists”³—an attitudinal shift that occurred between the introduction of DES in 1954 and its ban in 1979—led to the elimination of DES in cattle ranching. And that elimination occurred in the absence of any evidence that DES in beef might cause even a single case of human cancer.

² Marcus A. *Cancer from Beef: DES, Federal Food Regulation and Consumer Confidence*. Baltimore: Johns Hopkins Press; 1994:32.

³ Browne WP. The DES case. *Science*. January 20, 1995:394.

⁴ Lang JS. Cancer-inciting hormone found in U.S. beef supply. *Des Moines Register*. June 24, 1970.

⁵ *Cancer from Beef*:96.

⁶ National Cancer Institute/National Institutes of Health. Long-term health effects of exposure to DES—Background. NCI *CancerFax*. National Cancer Institute Office of Cancer Communications; May 1, 1996.

⁷ Herbst AL, et al. Prenatal exposure to stilbestrol: a prospective comparison of exposed female offspring with unexposed controls. *N Engl J Med*. 1975; 292(7):334–339.

⁸ Herbst AL, Scully RE. Adenocarcinoma of the vagina. *N Engl J Med*. 1971;284:

⁹ U.S. to require seven-day withdrawal period, mandatory certification for stilbestrol. *Feedstuffs*. 1971; 43:164.

¹⁰ *Panic in the Pantry*. 1992:166.

¹¹ Diethylstilbestrol—Order denying hearing and withdrawing approval of new animal drug applications for liquid and dry premixes, and deferring ruling on implants. *Federal Register*. 1972; 37:15747–15750.

¹² Diethylstilbestrol. *Federal Register*. 1973; 38:10485–10488.

¹³ *Cancer from Beef*:125–126.

¹⁴ Jukes TH. A quantitative evaluation of estrogens, including DES, in the diet. *The American Statistician*. 1982; 36(3):273–277.

¹⁵ *Cancer from Beef*:139–141.

¹⁶ Jukes TH. Chasing a receding zero: impact of the zero threshold concept on actions of regulatory officials. *J Amer Coll Toxicol*. 1983; 2(3):147–160.

5. Nitrites, 1972

Background

Salt and spices have been used to preserve foods and “cure” meat since the beginning of civilization. By the late 19th century, scientists had identified sodium nitrate as a substance that acted as such a preservative in meat while providing the meat with a pleasant taste

¹ Whelan E, Stare FJ. *Panic in the Pantry*. Rev ed. Buffalo, NY: Prometheus Books; 1992:137.

and color. Sodium nitrate was approved as a food additive in 1906, under the earliest federal food safety laws.

In the 1920s it was discovered that sodium nitrite, a breakdown product of sodium nitrate, performed the same function more effectively, and the USDA approved it as a direct additive.¹ By the 1950s scientific studies had also shown that nitrite prevented germination of the bacterial spores that cause deadly botulism² in canned goods and other foods stored under airtight conditions.

The Scare

In 1970 a paper in the journal *Nature* concluded that nitrites reacted in the body with other agents in food to form nitrosamines—substances known to be animal carcinogens.³ The following year Congress held hearings, and in August 1972 a Congressional committee released a report declaring that “nitrites and nitrates pose a potential danger to public health.”⁴

Because nitrites were most closely associated with many meat products—such as hot dogs and bacon—already under attack as being too fattening, too artificial, or out-and-out “junk food,” these charges became fuel to the fire for many so-called “consumer advocates.” Ralph Nader declared that hot dogs were “among America’s deadliest missiles;”⁵ and the recently formed Center for Science in the Public Interest called for an immediate ban,⁶ which was denied.

The nitrite issue leapt back into the news in October 1975 when a wire-service report announced that the Agriculture Department “plans to force the nation’s bacon producers to remove a dangerous cancer-causing agent from the breakfast table.” In fact, the wire-service report was untrue; when the regulations were released the following month, they merely set limits on the amount of sodium and potassium nitrite allowed in various foods.⁷

The Reaction

In 1977 Carol Tucker Foreman became Assistant Secretary of Agriculture. Almost immediately, she set out to actively pursue the nitrite issue, going so far as to say that she was not concerned whether or not a nitrite ban “would burden the consumer or the industry” and that she would not “balance the desire for . . . botulism protection against the fact that nitrosamines may be injurious.”⁸

Then, in 1978, a Massachusetts Institute of Technology (MIT) study found that 13 percent of test animals receiving nitrite contracted lymphatic cancer, as compared with 8 percent receiving no nitrite. Rumors of a nitrite ban were widely circulated, but the FDA

reminded the public that any decision was a “difficult balance of risks,” given nitrite’s protective effect against botulism.⁹

Some observers speculated that the FDA’s cautious approach, in contrast to their often rapid actions in the face of similar reports in the past, was a reaction to the public outcry over the attempted ban on saccharin the previous year (see page 18). In other words, “once consumers realized that their bacon, hot dogs, and bologna might look and taste odd once the additive was removed, they might take to the ramparts as they did for sugar-free Diet Pepsi.”¹⁰ In December 1978 the USDA began a monitoring program to assure that the level of nitrosamines in bacon was no more than 9 parts per billion;¹¹ otherwise, the furor died down significantly in the years that followed.

And the 1978 MIT report was questioned, not only by the FDA and many Congressmen, but also by leading pathologists, who discovered “gross irregularities” in the report’s tumor diagnoses and records. By 1980 the FDA, the USDA, and the Universities Associated for Research and Education in Pathology had all concluded that many of the “tumors” found in the MIT study could not be confirmed as such; even Assistant Secretary Foreman acknowledged that “there is no basis for the FDA or USDA to initiate any action to remove nitrite from foods at this time.”¹² The National Academy of Sciences did suggest that research be continued on reducing nitrite levels and searching for alternative curing products.¹³

Conclusion

Because nitrites were “grandfathered” at the time of the Delaney clause (see page 7) and thus were not subject to its zero-risk rule, their fate differed from that of many of the other substances banned in the 1970s. In this instance, regulators were able to place the risks from nitrites into perspective. For example, nitrates, which are converted to nitrites, occur naturally in human saliva¹⁴ as well as in many vegetables. They occur in spinach in concentrations of as high as 3,000 parts per millions (0.3%) and are also present in lettuce, celery, radishes, and beets.¹⁵ And nitrites in cured meat products account for only about 9 percent of nitrite ingestion.¹⁶

Additionally, this situation again involves the question of extrapolation from animal testing and the fact of minuscule human doses. This case also centers on the very real need to prevent botulism—a genuine public health threat. And in Norway, where nitrite curing was allegedly banned in the 1970s, the smoking process adapted as an alternative resulted in 50 percent-

higher levels of nitrite than those found in similar products in the U.S.¹⁷

Recently it has been reported that levels of nitrites used as meat preservatives have dropped 80 percent since the 1970s.¹⁸ And more importantly, vitamin C—which inhibits the formation of nitrosamines—has been added to nitrite-preserved foods.¹⁹

In 1995 a group headed by one of the coauthors of the original, 1970 *Nature* paper petitioned the USDA to require a warning label on hot dogs. But scientific evidence points in the opposite direction. The American Cancer Society states, “Nitrites in foods are not a significant cause of cancer among Americans.”²⁰ The Council for Agricultural Science and Technology agreed, in their recent review of the issue, that “the scientific evidence does not support restrictions in the consumption of salted, smoked, or nitrite-preserved foods by the U.S. population.”²¹ And there is no mention of nitrites in the National Research Council’s comprehensive report: *Carcinogens and Anticarcinogens in the Human Diet*.²²

So the best advice—as stated recently by a scientist who was on the USDA Expert Panel that reviewed the issue in the 1970s—is simply to “enjoy your cook-out.”¹⁶

¹ Cassens RG. *Nitrite-Cured Meat: A Food Safety Issue in Perspective*. Trumbull, CT: Food and Nutrition Press; 1990:18–21.

² Steinke PKW, Foster EM. Botulinum toxin formation in liver sausage. *Food Res*. 1951; 16:477.

³ Lijinsky W, Epstein SS. Nitrosamines as environmental carcinogens. *Nature*. 1970; 225:21.

⁴ Nineteenth Report by the Committee on Government Operations. *Regulation of Food Additives—Nitrates and Nitrites*. August 15, 1972. House Report No. 92-1338.

⁵ Kanfer S. The decline and fall of the American hot dog. *Time*. October 2, 1972:63.

⁶ *Food Chemical News*. February 14, 1972:35.

⁷ *Food Chemical News*. October 27, 1975:45.

⁸ *Food Chemical News*. January 16, 1987:44.

⁹ *Food Chemical News*. August 15, 1978:65.

¹⁰ Light L. Nervous over nitrite. *The Progressive*. February 1979:30–31.

¹¹ *AMI Newsletter*. August 26, 1988.

¹² *Food Chemical News*. August 25, 1980.

¹³ National Academy of Sciences. *Alternatives to the current use of nitrite in foods*. Washington, DC: National Academy of Sciences; 1982.

¹⁴ Jukes TH. Chasing a receding zero: impact of the zero threshold concept on actions of regulatory officials. *J Amer Coll Toxicol*. 1983; 2(3):147–160.

¹⁵ Jukes TH. *Nitrates and nitrites as components of the normal environment*. Proceedings of the Meat Industry Research Conference. March 25–26, 1976:41–48.

¹⁶ *Food Chemical News*. December 1997; 39:6.

¹⁷ Greenberg R. Have another hot dog, sweetie. *Miami Herald*. November 24, 1995.

¹⁸ *Food Chemical News*. December 1997; 39:21.

¹⁹ Berlitz HD, Grosch W. *Food Chemistry*. New York: Springer-Verlag; 1987:374.

²⁰ American Cancer Society. 1996 *Guidelines on Diet, Nutrition and Cancer Prevention*. Atlanta, GA: The American Cancer Society 1996 Dietary Guidelines Advisory Committee; 1996.

²¹ Council for Agricultural Science and Technology. *Examination of dietary recommendations for salt-cured, smoked, and nitrite-preserved foods*. Issue Paper. November 1997; 8:1–9.

²² National Research Council. *Carcinogens and Anticarcinogens in the Human Diet*. 1996. National Academy Press, Washington, DC

6. Red Dye Number 2, 1976

Background

Red Dye Number 2, also known as amaranth, was first synthesized in 1878. For much of the 20th century it was the most widely used of all food colorings. In the early 1900s the Department of Agriculture chose it as one of only seven dyes suitable for use in food, and in 1938 it underwent further testing before being approved under the Food, Drug, and Cosmetic Act.

In the 1950s, after some children became ill from eating Halloween candy and colored popcorn, more general tests were ordered, but Red No. 2 was exonerated. Subsequent FDA tests on rats concluded that Red No. 2 “had no significant influence in the formation of

tumors.” Further FDA tests during the 1960s indicated that Red No. 2 did not damage laboratory animals even under the most stringent test conditions.

As of 1970 Red No. 2 was being used in over \$10 billion worth of food products ranging from soft drinks, candy, and pet foods to baked goods, sausages, ice cream, and processed fruits. It was also used in pill coatings and in cosmetics.¹

The Scare

In a study published in 1970, scientists at the Moscow Institute of Nutrition found tumors in 13 of 50 male rats fed amaranth for 33 months but found no tumors in the control group.² A second Russian study found that the use of amaranth caused stillbirths and deformities among female rats.³ The methodology of both studies was immediately criticized by American scientists: The species of rats used are naturally prone to tumors, and the effects of the dye were actually greater at lower doses than at higher ones. It was also unclear whether the purity of the dye used by the Russians was similar to the purity of that used in the United States.^{1,4} Nevertheless, the FDA immediately ordered additional studies.

One reproductive study by Dr. Jacqueline Verrett—the researcher who had conducted the infamous studies on chicks exposed to cyclamates (see page 12)—showed that chicks exposed to Red No. 2 suffered high death rates and a variety of other birth defects. As happened with her cyclamate studies, however, other FDA scientists questioned the significance of her findings, noting that the introduction of any substance into a closed system—such as that of a chick’s egg—might be harmful.

Another study of pregnant rats dosed with Red No. 2 showed some increase in birth defects and miscarriages as dosage increased, although at a level not considered statistically significant. Based on this study, the FDA issued a ruling in November 1971 significantly curtailing use of the dye, only to reverse itself the following June after subsequent studies on rats, hamsters, mice, and rabbits failed to show any link to birth defects or miscarriage.⁵

A study of 500 female rats, completed in 1975, aimed to determine definitively whether Red No. 2 had carcinogenic effects. Unfortunately, even before any results could be drawn from that study, FDA officials acknowledged that it had been carried out in an unprofessional manner: The scientist originally in charge of the experiment had left the agency midway through; handlers had mixed up the rats, blurring the distinction between those given dye and those not; and most of the

rats that died during the experiment had been left to rot in their cages. One scientist called it “the lousiest experiment I had seen in my life.”^{4,6}

Nevertheless, the FDA—under public pressure from U.S. Senator Gaylord Nelson and the Ralph Nader–affiliated Health Research Group to act on Red No. 2—attempted to draw some conclusions from the flawed study. An FDA advisory committee concluded in November 1975 that Red 2 had “no apparent adverse effect on rats”⁷—only to have its findings contradicted a month later by a statistician at another government agency who reviewed the same data but accused the FDA of not performing a correct statistical analysis.

FDA Commissioner Alexander Schmidt was not used to the level of media scrutiny he was receiving. (He had recently endured a grilling on CBS’s *Face The Nation*.) So Schmidt tried to find a way out of this public-relations quandary. On January 19, 1976, noting that Red No. 2 had been under only “provisional approval” since 1960 while testing had been going on, Commissioner Schmidt announced that the dye’s provisional approval was being revoked. This prohibited any future use of the dye.⁷

Schmidt nonetheless declared that “there was no evidence of a public health hazard” from the dye and added that even if the “botched” study were to show a cancer link, a human would have to drink 7,500 cans of soda a day to reach the rats’ level of consumption. Schmidt did not order companies to pull Red 2-containing products from store shelves,⁸ and he held out the possibility that the dye could be reapproved in the future if its manufacturers could prove that “Red No. 2 has a safe and useful place in the food supply.”⁹

The Reaction

Manufacturers of Red No. 2 were outraged that the dye was banned without any new evidence. They noted that Canadian health authorities did not find any “biological significance to the FDA’s data” and so did not move to ban the dye.¹⁰ Perhaps the most curious action came on the part of the M&M/Mars candy company, which ceased manufacturing red M&Ms—an odd move because the candies were never made with Red No. 2, but with two other dyes. The company announced that it was withdrawing the red candies “to avoid any consumer confusion or concern.”¹¹ (Red M&Ms made a much-ballyhooed return in 1988.)

The primary replacement for Red 2 was Red No. 40—a dye that costs more, does not project exactly the same hues,⁸ and, ironically, at the time had not been tested anywhere as extensively as Red No. 2.¹²

Conclusion

One science writer at the time of the Red 2 ban called the FDA's action "a panicky response to outside pressures."¹³ The trade publication *Food Chemical News* summed up the verdict on Red 2's safety by saying it boiled down to "one positive test and dozens of negative tests."¹⁴ Clearly, much of the impetus to ban Red 2 was not so much that there was any proof of harm to human health as the fact that this was not considered a "necessary" product: Consumers Union noted at the time that Red 2 was not a "valuable drug or indispensable food staple."⁵ Another article commented that the dye was "mainly used . . . in products that consumer activists consider 'junk foods.'"¹⁵ Yet there are many "nonnecessary" items that most consumers are unwilling to give up, even in the face of negative news.

7. Saccharin, 1977

Background

Saccharin, a noncaloric, white crystalline powder 300 times sweeter than sugar, was first synthesized in 1879. It was the first artificial sweetener marketed in the U.S. and has been produced commercially for over 80 years. Saccharin was declared "generally recognized as safe" (GRAS) in 1958.¹ As with cyclamates, its use by the general public increased rapidly during the 1960s, although its popularity was not as great as that of cyclamates due to its bitter and metallic aftertaste.² After the cyclamates ban, the use of saccharin as a sugar substitute increased dramatically.

The Scare

Almost immediately after the cyclamate scare (see page 11), saccharin became the next target. A 1970 study suggested that saccharin caused bladder cancer in mice.² When a 1972 study showed that it also caused bladder cancer in rats, the FDA removed it from the GRAS list.³ Then, after a 1977 Canadian study showed similar results, the FDA moved to ban saccharin altogether, basing the decision on the Delaney clause prohibition on the use of food additives that cause cancer in animals. Again, the amounts ingested by the rodents were enormous, up to 5 percent of their total diets.⁴

The Reaction

Unlike the generally agreeable response to the cyclamate ban, the public reaction to the FDA's proposed action on saccharin was overwhelmingly negative. Consumers voted first with their wallets, "sweeping the shelves clean," as one housewife recalls, of pink saccharin packets and any saccharin-containing products in an attempt to stock up against the coming ban.⁵ Diabetics lobbied Congress to reverse the ban, given that no other nonsugar sweetener was available. And the diabetics were joined by many weight-conscious members of the general public: During 1977 Congress received more mail on the saccharin issue than on any other topic.⁶

Under public pressure, Congress imposed a moratorium on the ban, requiring instead that saccharin-containing products carry a warning label. The moratorium has been extended six times.¹ Meanwhile, continued studies of saccharin show that while it clearly is a carcinogen in male rats (although a very weak one, in terms of the dose required to produce tumors), it has not been shown to produce tumors in female rats or in any other species.⁷⁻¹⁰ Many scientists believe that these tumors are due to two proteins found in the urine of

¹ Boffey PM. Death of a dye? *The New York Times Magazine*. February 29, 1976:9.

² Andrianova MM. Carcinogenic properties of the red food dyes amaranth, Ponceaux SX, and Ponceaux 4R [in Russian]. *Voprosy Pitanya*. 1970; 5:29.

³ Shtenberg AI, Gavrilenko YU. Effect of amaranth food dye on reproductive function and progeny development in experiments with albino rats [in Russian]. *Voprosy Pitanya*. 1970; 29:66-73.

⁴ Corwin E, Pines WL. Why FDA banned Red No. 2. *FDA Consumer*. April 1976:18-23.

⁵ Red food coloring: how safe is it? *Consumers Reports*. February 1973.

⁶ Boffey PM. Color additives: botched experiment leads to banning of Red Dye No. 2. *Science*. 1976; 191:450-451.

⁷ Boffey. *Death of a dye?*:11-13.

⁸ Death of a dye. *Time*. February 2, 1976:53.

⁹ Boffey. *Death of a dye?*:13.

¹⁰ Boffey. *Death of a dye?*:9.

¹¹ Seeing red. *The Atlantic Monthly*. October 1988:34-35.

¹² Boffey PM. 1976. "Color additives: botched experiment leads to banning of Red Dye No. 2" *Science* 191; as cited in Efron E. 1984. *The Apocalypitics*. New York: Simon & Schuster:12.

¹³ Boffey. *Death of a dye?*:13.

¹⁴ Whelan E, Stare FJ. *Panic in the Pantry*. Rev ed. Buffalo, NY: Prometheus Books; 1992:131.

¹⁵ Boffey. *Death of a dye?*:9.

male rats, but not found in humans or other animals, that react with high levels of saccharin to produce crystals that damage the bladder.^{11–13}

Furthermore, human epidemiological studies have failed to show a link between bladder cancer and heavy saccharin use.^{14–16} For example, people born in Denmark during World War II (when sugar was largely unavailable) and diabetics, who have been constant users of this sweetener for several decades, have failed to show higher-than-normal rates of bladder cancer.^{17,18} In another study, 3,000 individuals recently diagnosed with bladder cancer were found not to show heavier use of saccharin than members of a control group without bladder cancer.¹⁹

As a result of these findings, the FDA withdrew its proposal to ban saccharin, although the Congressionally imposed warning label remains in place.²⁰ Saccharin continues to be available; but its use in the U.S. has decreased since 1983, when the FDA approved aspartame, a sugar substitute with no after-taste.²¹

Conclusion

The charges leveled against saccharin and the research used to justify its banning illustrate the many problems policymakers have when they indiscriminately extrapolate results from animals to man. These include:

- the enormous doses necessary for such experiments—doses so large that they may overwhelm the animal’s natural defenses—and
- the question of whether a substance that is a carcinogen in one species is necessarily a carcinogen in others, given the physiological differences between species, and especially between rodents and humans.

There is also a regulatory “double standard” implicit in the fact that many “natural” substances—everyday items such as table pepper and vitamins A and D among them—have also been proved to be rodent carcinogens under such testing regimens; but these natural substances are not subject to the sort of regulatory action that would be taken with a synthetic additive.²²

Due to the accumulation of more than two decades of extensive research discounting the risk of bladder cancer due to saccharin use, the then-director of the National Cancer Institute stated in 1988 that “to be harmed by saccharin, one would have to take enough to turn yourself [*sic*] into a giant saccharin crystal.”²³ In its 1996 Dietary Guidelines, the American Cancer

Society concluded the following: “Several years ago, experiments on rats suggested that saccharin might cause cancer. Since then, however, studies of primates and humans have shown no increased risk of cancer from either saccharin or aspartame.”²⁴

The tide seemed finally to have turned for saccharin. In September 1996 the industry group Calorie Control Council petitioned that saccharin be delisted from the “anticipated” carcinogen list of the National Toxicology Program (NTP)—a branch of the National Institute of Health.

But the controversy continued. Despite the ruling of two preliminary NTP subcommittees in favor of delisting, the seven-person NTP committee—ignoring the wealth of information and data pointing to the contrary—voted 4 to 3 on October 31, 1996, to continue listing saccharin as *reasonably anticipated to be a human carcinogen*.²⁵ The committee even chose to ignore the testimony of Dr. Samuel Cohen, a human pathologist at the University of Nebraska who had performed extensive research on saccharin. Dr. Cohen stated that human urine is vastly different from rat urine and does not react with saccharin in the same way: “We now have enough understanding to know that this is a rat-specific phenomenon.”²⁶

The continued listing of saccharin as an “anticipated human carcinogen” is unlikely to have any effect on its availability, however. The sweetener continues to be widely used in low-calorie, sugar-free foods; and its supposed link to human cancer remains one of the greatest unfounded health scares of the last 20 years.

What was different in the saccharin case was that the public viewed saccharin, unlike most “artificial” chemicals, as a product it needed and wanted. Saccharin served a purpose in people’s lives—and they ardently objected to the efforts being made to remove it from the market. This contrasts with other banned substances that were implicated as carcinogens on equally dubious evidence but that elicited less public understanding regarding their purpose or use. Today’s highly urbanized public has little knowledge of the food production system, for example, or of the vital role pesticides and other agricultural chemicals play in that system. The public is thus willing to fear the worst when such a substance is implicated as a carcinogen.

¹ Meister K. *Low-Calorie Sweeteners*. New York: American Council on Science and Health; 1993:18.

² Bryan GT, Erturk E, Yoshida O. Production of urinary bladder carcinomas in mice by sodium saccharin. *Science*. 1970; 168:1238–1240.

- 3 Whelan E, Stare FJ. *Panic in the Pantry*. Buffalo, NY: Prometheus Books; 1992:146.
- 4 Whelan E, Stare FJ. *The 100% All Natural Nutrition Hoax*. New York: Atheneum; 1984:161.
- 5 Conversation between Adam Lieberman and Dr. Elizabeth Whelan, January 1996.
- 6 *Panic in the Pantry*. 1992:147.
- 7 Schoenig GP, Goldenthal EI, Geil RG, Frith CH, Richter WR, Carlborg FW. Evaluation of the dose-response and in utero exposure to saccharin in the rat. *Food Chem Toxicol*. 1985; 23(4-5):475-490.
- 8 Cohen SM, Ellwein LB. Risk assessment based on high-dose animal exposure experiments. *Chem Res Toxicol*. 1992; 5:742-748.
- 9 Whysner J, Williams GM. Saccharin mechanistic data and risk assessment: urine composition, enhanced cell proliferation, and tumor promotion. *Pharmacol Ther*. 1996; 71:225-252.
- 10 Takayama S, Sieber SM, Adamson RH, Thorgeirsson UP, Dalgard DW, Arnold LL, Cano M, Eklund S, Cohen SM. Long-term feedings of sodium saccharin to nonhuman primates: implications for urinary tract cancer. *J Natl Cancer Instit*. 1998; 90:19-25.
- 11 Cohen SM, Cano M, Earl RA, Carson SD, Garland EM. A proposed role for silicates and protein in the proliferative effects of saccharin on the male rat urothelium. *Carcinogenesis*. 1991; 12:1551-1555.
- 12 Olson MJ, et al. A comparison of male rat and human urinary proteins: implications for human resistance to hyaline droplet nephropathy. *Toxicol Appl Pharmacol*. 1990; 102:524-536.
- 13 Zurlo J, Squire RA. Is saccharin safe? Animal testing revisited. *J Natl Cancer Instit*. 1998; 90(1):2-3.
- 14 Armstrong B, Doll R. Bladder cancer mortality in England and Wales in relation to cigarette smoking and saccharin consumption. *Br J Prev Soc Med*. 1974; 28(4):233-240.
- 15 Wynder EL, Stellman SD. Artificial sweetener use and bladder cancer: a case-control study. *Science*. 1980; 207(4436):1214-1216.
- 16 Morrison AS, Verhoek WG, Leck I, Aoki K, Ohno Y, Obata K. Artificial sweeteners and bladder cancer in Manchester, U.K., and Nagoya, Japan. *Br J Cancer*. 1982; 45(3):332-336.
- 17 Ellwein LB, Cohen SM. The health risks of saccharin revisited. *Crit Rev Toxicol*. 1990; 20:311-326.
- 18 Jensen OM, Kamby C. Intra-uterine exposure to saccharin and risk of bladder cancer in man. *Int J Cancer*. 1982; 29:507-509.
- 19 Hoover RN, Strasser PH. Artificial sweeteners and human bladder cancer. (preliminary results). *Lancet*. 1980; 1(8173):837-840.
- 20 *Low-Calorie Sweeteners*:19.
- 21 *Low-Calorie Sweeteners*:6.
- 22 Havender W, Whelan E. Sweet truth. *Reason*. October 1984:33-38.
- 23 DeVita V Jr. Director of National Cancer Institute—Presentation to American Cancer Society science writers' forum. March 1988.
- 24 American Cancer Society. *1996 Guidelines on Diet, Nutrition and Cancer Prevention*. Atlanta, GA: The American Cancer Society 1996 Dietary Guidelines Advisory Committee; 1996.
- 25 *Food Chemical News*. November 10, 1997:31-33.
- 26 Stolberg SG. Bid to absolve saccharin is rebuffed by U.S. Panel. *New York Times*. 1997.

8. Hair Dyes, 1977

Background

Commercial hair dyes have been available since about 1920. The key ingredients in most permanent dyes—colors that last until the hair grows out—are so-called “coal-tar” dyes (actually petroleum derivatives). Under the Food, Drug, and Cosmetic Act of 1938, which first placed cosmetic products under federal regulation, products containing coal-tar dyes must carry a warning label noting that they may cause skin irritation in some individuals.¹ Today, anywhere from 20 to 60 percent of Americans are estimated to use some type of hair coloring; most of it contains coal-tar dyes.²

The Scare

In 1975 students in a biochemistry class at the University of California at Berkeley engaged in a class project in which common household products were tested on bacteria to see if they caused genetic mutations. A permanent hair dye was the only substance, other than cigarette tar, to cause such a mutagenic reaction. Subsequent tests showed similar mutagenic reactions in bacteria from many such dyes.¹

The National Cancer Institute began testing 13 coal-tar chemicals on laboratory rodents. In late 1977 they published a study reporting that seven of the 13 caused tumors in the animals. The most potent of the tested chemicals was 4-methoxy-m-phenylenediamine-sulfate (4-MMPD), which caused thyroid and skin tumors in 24 percent of the rodents.³ The FDA announced in early 1978 that all dyes containing 4-MMPD would carry a warning label noting that it was an animal carcinogen. Before the label order could go

into effect, however, hair-dye manufacturers responded by reformulating their products, removing 4-MMPD as well as three other chemicals commonly used in the dyes and implicated in the rodent tests.¹

The Reaction

This was not enough for some consumer activists, who accused hair-care companies of using substitute chemicals that were as hazardous as the substances removed. Dr. Benjamin Van Durren of New York University's Institute of Environmental Medicine claimed there was "not one iota" of difference between the replacement chemicals and those they replaced, even though the replacement chemicals tested negative as animal carcinogens.

The cosmetics companies objected to the methodology of the original tests, noting that the rodents drank the dye—obviously not the method of human exposure. Although hair-dye chemicals applied to the scalp can penetrate the skin, an earlier FDA test had shown that only 3 percent of the dye was absorbed, and about half of this was excreted in urine. Furthermore, the doses used on the laboratory rodents were the equivalent of a woman's drinking 25 bottles of hair dye every day for her entire life.¹

At the same time, epidemiological studies were launched to assess whether regular users of dyes were suffering a greater incidence of cancer. Two early studies from Great Britain and Canada failed to show that women using hair dye faced a greater risk of cancer; a third study indicated that women over 50 years old who had used dyes for 10 years or more had a greater incidence of breast cancer. All three studies were limited by the small number of subjects involved.¹ The public concern over the issue subsequently died down, although henna and other "natural" plant-based colorings, which are not as long-lasting, gradually became more popular.

Conclusion

Epidemiological studies continued in an attempt to assess whether the dyes—old or reformulated—ever posed a cancer risk. In a 1992 study of 373 women with non-Hodgkin's lymphoma, researchers concluded that women who used hair dyes had a 50 percent greater chance of developing the disease⁴; but the FDA said that that study "does not allow the establishment of a causal link between hair dye and increased cancer"⁵ and so refused to impose a warning label on hair-care products. A much larger study carried out by the FDA and the American Cancer Society showed that "women who use hair dyes do not have an overall greater risk of dying from cancer," although a much smaller sub-

group—those who used black hair dyes for more than 20 years—showed an increase in non-Hodgkin's lymphoma and multiple myeloma.⁴

Most recently, Brigham and Women's Hospital conducted a study involving more than 99,000 women and specifically designed to determine whether a link existed between cancer and hair dyes. That study showed no greater risk of blood or lymph cancers among users of dyes.⁶ The National Cancer Institute is currently on record as concluding that, while further research is needed in this area, "no recommendation to change hair dye use can be made at this time."

¹ Are Hair Dyes Safe? *Consumer Reports*. August 1979:456-459.

² National Cancer Institute. Personal use of hair coloring products. *NCI CancerFax*. National Cancer Institute; August 1, 1996:1.

³ Gwynne P, Copeland JB. Household worries. *Newsweek*. October 31, 1977:109.

⁴ Zahm SH, Weisenburger DD, Babbitt PA, et al. Use of hair coloring products and the risk of lymphoma, multiple myeloma, and chronic lymphocytic leukemia. *Am J Public Health*. 1992; 82:990-997

⁵ Newman J. Hair dye distress. *American Health*. November 1992:39.

⁶ Grodstein F, Hennekens CH, Colditz GA, et al. A prospective study of permanent hair dye use and hematopoietic cancer. *JNCI*. 1994; 86:1466-1470.

9. Tris, 1977

Background

In 1953, to protect the public from unreasonable risk of fires, the government passed legislation designed to regulate the manufacture of highly flammable clothing. The act was amended several times during the 1960s and 1970s to set flammability standards for additional products. In 1972 the first such standard was set for children's sleepwear. To meet the new standards, most manufacturers chose to produce sleepwear made of polyester or acetate, to the surface of which was added the flame retardant tris-(2,3-dibromopropyl) phosphate, familiarly called "Tris." The change in industry and consumer patterns in this area was dramatic: In 1971, 56 percent of children's sleepwear was constructed of cotton and 27 percent of polyester-cotton; by 1975, 87 percent was constructed of various synthetics.¹

The Scare

In January 1977 Berkeley biochemistry professor Bruce Ames published an article in *Science* magazine in which he declared that Tris was a mutagen (a substance capable of causing changes in genetic material) in bacteria. He based this conclusion on the salmonella/microsome test (“the Ames test”) he had developed (a test that had also been used in the 1975 hair-dye study; see page 20).

The test showed Tris to be more mutagenic than many known carcinogens (cancer-causing substances). Ames noted further that 90 of the known carcinogens were also mutagens, whereas few noncarcinogens were. Furthermore, Ames suggested that Tris could be absorbed through the skin (he based this suggestion on previous studies involving rats and rabbits in which Tris was directly applied to their skin) and that certain impurities found even in “high-purity” Tris caused squamous carcinomas when fed to rats and mice.

While acknowledging that there was no direct evidence that humans could absorb Tris through the skin (the only such experiment, with two human volunteers, had tested negative), Ames nevertheless concluded that “the risk from cancer might be very much higher than the risk from being burned . . . the use of an untested chemical as an additive to pajamas is unacceptable in view of the enormous possible risks.”¹

The Reaction

The response to this single study was incredibly rapid: No sleepwear manufacturer wanted to be accused of spreading cancer to children, and all of them immediately ceased manufacturing garments containing Tris. Three months later, after the release of a study showing that Tris caused kidney cancer when ingested by mice and rats, the Consumer Products Safety Commission imposed a ban on any further sale of such garments.² Exports of Tris-containing garments were banned the following year.³

Ironically, one of the strongest criticisms against this action came from the editorial page of *Science* itself. Editor Phillip Abelson noted that no evidence had been presented showing that Tris baked into a fiber (as it was in the fibers used in the manufacture of sleepwear) rather than applied directly to the skin caused cancer. Abelson also noted that the rodents that had developed kidney cancer had been bred to be especially cancer prone.²

Conclusion

At the time, the Tris ban was touted by both the scientific and popular press as an example of how a sin-

gle scientist could make a difference in public policy. But the Ames bacterial test—the test Dr. Ames used in 1977 to show that Tris was a mutagen—has itself come under increasing criticism. Subsequent studies by the National Cancer Institute and the National Toxicology Program have shown that there was only about a 50- to 70-percent correlation between carcinogenicity and mutagenicity.⁴ And Dr. Ames did not receive the same degree of mass media attention or public admiration in 1983 when he published a landmark paper in *Science* declaring that natural carcinogens were as much as 10,000 times more prevalent in the human environment than synthetic ones—and that any regulatory policy that focused on man-made carcinogens alone was therefore scientifically unfounded.^{5,6}

So the architect of the Tris ban—a scientist who at the time was also outspoken on the danger of a variety of other synthetic chemicals⁷—has now become perhaps the premier scientific spokesman for the belief that the products of modern technology are nowhere near as hazardous to our health as many in the media and in the environmentalist community have led us to believe.

¹ Ames B, Blum A. Flame-retardant additives as possible cancer hazards. *Science*. January 7, 1977:17–22.

² Abelson P. The Tris controversy. *Science*. July 8, 1977:13.

³ Hinds M deC. Reagan signs law on pajamas makers. *The New York Times*. January 1, 1983.

⁴ Proctor R. *Cancer Wars*. New York: Basic Books; 1995:300–301.

⁵ *Cancer Wars*:133–137.

⁶ Ames BN. *Science*. 1983; 221:1256.

⁷ *Cancer Wars*:137.

10. Love Canal, 1978

Background

Love Canal took its name from William Love, an entrepreneur who in the 1890s had an unsuccessful plan to build a massive city near Niagara Falls. The unfinished canal that was dug in the area in Love’s time was used between 1942 and 1953 by both the Hooker Chemical Company and the U.S. military to dispose of industrial and chemical wastes, which were sealed under clay lining.

In 1957, as the population of the surrounding area grew, Hooker sold the property above the canal for \$1 to the local school board to build a school on the site, with the caveat that the land should not be excavated due to the wastes buried underneath. Despite Hooker's warning, the city of Niagara Falls subsequently constructed sanitary and storm sewers at the site, disturbing the wastes.¹

The Scare

Starting in 1976, local residents began to complain of chemical odors from the landfill, and of chemicals seeping into basements. A local reporter began to write about suspected cases of illness among residents of the canal area. In response to these reports, in 1978 the New York State Health Commissioner called for the evacuation of families, pregnant women, and young children from the area immediately surrounding the canal. Later that year the state announced the relocation of all families living within a two-block radius of the canal.

These announcements led to a ripple effect throughout the community, with homes outside the immediate area losing value. Residents conducted their own informal surveys, which appeared to show elevated incidence of numerous ailments.² A report by Dr. Beverly Paigan in 1979 found a high rate of birth defects and miscarriages among Love Canal residents. The study was not a scientific, controlled study, however; it was based only on anecdotal reports from interviews with families in the area.³ In May 1980 an EPA study reported possible chromosome damage among Love Canal residents; two days later another EPA study concluded that a degree of peripheral nerve damage existed among residents.

The Reaction

The two EPA studies immediately became national news. In Love Canal itself, hysteria ensued, with two EPA officials being "involuntarily detained" for several hours. A few days later, the EPA announced that 2,500 residents would be temporarily relocated, at a cost of \$3 to 5 million. (The relocation eventually became permanent, at a cost of over \$30 million.)⁴

Was there a public health justification for these actions? Both of the EPA studies have since been criticized by other health and scientific authorities, not only for being released prior to peer review, but for errors in statistical analysis, for small sample size, and for improperly drawing conclusions that were in some cases contrary to the evidence. (The chromosome study, for example, actually found that cases of chromosome

damage were lower overall among Love Canal residents than among a control group.) Subsequent, peer-reviewed studies from the New York State Department of Health failed to show any abnormal health trends among Love Canal residents. And additional studies made in later years by the Centers for Disease Control and Prevention, the American Medical Association,⁵ and the National Research Council reached the same conclusion.⁶

Conclusion

In 1982 the EPA conducted a study showing that outside the area immediately surrounding the canal, there was no unusually high level of chemical contamination. By 1990, after many court battles with environmental groups, a New York state agency created to manage homes near the canal began putting houses on the market.

Despite the obstacle of negative publicity and of banks refusing to grant mortgages for fear of being held liable for environmental contamination,⁷ by June 1994, 193 out of 280 available homes had been sold. Appraisers originally deducted 20 percent from house prices because of the location, but this later was reduced to 15 percent as demand increased. Some 30 percent of the purchasers were pre-1980 residents.⁸

The legal battles that have gone on since 1980 also appear to be winding down. In March 1994 a federal judge rejected a claim of \$250 million in punitive damages filed by New York state against Occidental Chemical, which had purchased Hooker in 1968.⁹ Occidental settled out of court with the EPA in December 1995, agreeing to pay \$129 million to cover the costs of cleaning up the site in exchange for the federal and state governments' dropping all other claims against the company.¹⁰

One immediate result of the 1980 panic—a result that remains not only a continuing legacy but a burden—is the so-called "Superfund." Authorized by Congress just months after Love Canal hit the headlines, Superfund spends about \$1.7 billion annually—about what the National Cancer Institute spends on research and development—to clean up approximately 2,000 waste sites. Today, over 15 years later, most of the Superfund sites have not been reclaimed; and most of the budget has been spent on legal and consulting fees. It remains unclear whether there will be any benefit to public health if and when all the sites are ever cleaned up.¹¹

Efforts have been made to reform the Superfund program, but so long as any attempt at reform is portrayed as a means of getting polluters "off the hook"—

as EPA head Carol Browner recently charged¹⁰—rather than as an attempt to put the public health risks from such sites into perspective, any such change seems unlikely.

¹ Whelan E. *Toxic Terror*. 2nd ed. Buffalo, NY: Prometheus Books; 1993:125–129.

² Wider Range of Illness Suspected. *Niagara Gazette*. August 4, 1978.

³ New York Governor’s Panel, October 1980.

⁴ *Toxic Terror*:131–132.

⁵ American Medical Association News Release. *Love Canal Residents Not at Risk, Says CDC*. March 16, 1984.

⁶ *Toxic Terror*:134–140.

⁷ *Toxic Terror*:141–142, 144–145.

⁸ Kirschner E. Love Canal settlement. *Chemical & Engineering News*. June 27, 1994:4–5.

⁹ Reisch M. Court rejects punitive damages for Love Canal. *Chemical & Engineering News*. March 28, 1994:7.

¹⁰ Love Canal settlement highlights Superfund debate. *Chemical Marketing Reporter*. January 1, 1996:3.

¹¹ *Toxic Terror*:69–70.

11. Three Mile Island, 1979

Background

Located 10 miles south of Harrisburg, Pennsylvania, Three Mile Island (TMI) Nuclear Station started commercial operations in September of 1974. In 1978 TMI began running a second reactor, Unit 2. More than 800,000 people benefit from the electrical services this facility provides.

The idea of radiation has long inspired apprehension and misunderstanding in the general population in the United States. Often, people reflexively associate radiation with the devastating effects of the atomic bomb. Unfortunately, too, the media tend to publicize health risks of nuclear power; as a result, the media have generated fear of radiation energy in the public mind.

What many people do not know is that all forms of life on earth have evolved in the presence of natural background radiation. Such radiation is present every-

where in small amounts. The radiation doses people receive from nuclear plants may, in fact, be lower than the doses associated with such “nonnuclear” activities as smoking, flying, or burning coal for power generation. These low levels of radiation do not produce the health effects observed after prolonged exposure to high levels of radiation—effects such as damage to genes and chromosomes, damage to developing human embryos and fetuses, and damage to cells that can increase their risk of becoming cancerous.

The Scare

On Wednesday, March 28, 1979, at 4:00 A.M., what normally would have been a minor plumbing problem occurred in the cooling system at Three Mile Island. Even though the Unit 2 reactor was immediately shut down by the automatic safety system, the reactor core continued to produce what is called “decay heat” and needed to be cooled. Because the instruments in the control room did not provide the operators with a ready understanding of key plant conditions, they subsequently shut down the main pumps that would have provided cooling water to the reactor core. The problem was exacerbated because the pressure relief valve was stuck open, resulting in the loss over the next few hours of some 30,000 gallons of water needed for the primary cooling system. This loss ultimately led to the uncovering of the top portion of the reactor core and the meltdown of some of the reactor’s fuel.

Due to increased radiation readings at various points outside the plant, the operators declared a “site emergency” at about 7:00 A.M. and a “general emergency” about 30 minutes later. During the week following the initial accident, 2.5 to 10 million curies of radioactivity escaped into the atmosphere in the form of steam and gas—an amount slightly above the normal background radiation level.

The Reaction

Initial reports in the media were calm in tone and citizens were informed that the situation at TMI was under control. Public concern immediately following the accident was negligible. Fear of radioactive contamination gradually increased among the local populace, however. Five days after the accident the governor advised all pregnant women and preschool children living within five miles of TMI to evacuate the area. In all, 60 percent of those living within the five-mile range left. Within 15 miles of TMI, approximately 39 percent of the population evacuated.

The greatest number of those who evacuated did so on March 30; estimates of the numbers leaving that

day range from 55 to 72 percent of total evacuees. Hershey, Pennsylvania, was designated the official evacuation center; on more than one occasion there were more reporters than evacuees at Hershey.¹

The governor's evacuation advisory was lifted on April 4, but the median date of the evacuees' return to the affected area was April 2. Schools within five miles of Three Mile Island reopened on April 11.¹

Although monitors were established both inside and outside the TMI plant, questions arose as to whether those monitors provided accurate data on the radiation doses received by the public. Because of these questions the U.S. Department of Health, Education, and Welfare decided to collect unexposed film from photographic shops in the area; the agency used them to provide an independent estimate of the exposures. The U.S. Department of Energy brought whole-body counters into the area and checked some 2,000 people for any radioactive materials that might have been deposited in or on their bodies. The U.S. Department of Health, Education, and Welfare; the Environmental Protection Agency; and the U.S. Nuclear Regulatory Commission organized an Ad Hoc Population Dose Assessment Group to provide an accurate assessment of the doses received by area residents.

The ad hoc group's review of the records, supplemented by the data generated by the analyses of the photographic film and the whole body counts, led the researchers to conclude that the maximum possible radiation dose received by an individual standing on the border of the plant site for the duration of the 10-day period following the accident was about 80 millirems.²

This dose is comparable to natural background exposure levels, which—exclusive of indoor radon—contribute about 100 millirems per year. The average likely dose to persons living within five miles of the plant was estimated to be 9 millirems, an amount similar to the dose an airline passenger receives during two round-trip transcontinental flights. And these estimates are, in fact, overestimations of the doses received: The numbers are calculated on the basis of an affected person having remained outside continuously from March 28 to April 7.^{3,4}

Despite the low levels of the estimated radiation doses, the residents—mainly responding to the scare stories in the media—remained apprehensive of their risks. To allay the community's fears and reduce confusion, the Commonwealth of Pennsylvania and the federal government announced that they would follow up and study the exposed population in the years ahead to monitor any possible changes in their physical and mental health.⁵ The agencies involved in the follow-up stud-

ies included the Pennsylvania Department of Health (DOH), the Centers for Disease Control and Prevention (CDC), and the U.S. Bureau of the Census. Accordingly, every five years since the accident, the Pennsylvania DOH has conducted special health surveys on the TMI-affected population.

Conclusion

What was the health legacy of the Three Mile Island accident?

The principal effect seems to have been on mental health. The director of the Public Health and Safety Task Force of the President's Commission on the Accident at TMI stated that "the major health effect of the accident at Three Mile Island was that of a pronounced demoralizing effect on the general population in the Three Mile Island area." He went on to identify teenagers and mothers of preschool children living within five miles of TMI as the most susceptible members of the affected population. Thus, the demoralization factor—the feeling of not being able to cope with the stress of the imposed environment—was the accident's chief residual danger.⁶

On the other hand, studies designed to determine the physical effects of the accident indicated no changes. Studies of such high-risk groups as pregnant women found no effects.^{7,8} Pennsylvania Department of Health studies confirmed that pregnant women exposed to the accident showed no measurable differences for prematurity, for congenital abnormalities, for neonatal death, or for infant hypothyroidism.^{9,10} No increased risk of cancer due to radiation emissions was found.¹¹

The Three Mile Island Public Health Fund—a court-supervised fund created to address the public health concerns of the residents—commissioned Columbia University's Division of Epidemiology to perform a comprehensive study of cancer incidence around Three Mile Island. In 1990 the researchers reported that they had found no excess cancer due to the radiation releases from the TMI nuclear facility.¹² In addition, at the request of U.S. Senator Edward M. Kennedy, the National Cancer Institute conducted its own study, which was released in September 1990. Again, no evidence was found of excess occurrence of cancer in people living near the TMI nuclear facility.⁹

On June 7, 1996, Judge Sylvia H. Rambo of the United States District Court of Pennsylvania dismissed all 2,100 lawsuits claiming injury from the TMI accident. She based her decision on "the paucity of proof alleged in support" of the case against the Three Mile Island Nuclear Station.¹³

But although there have been dozens of major independent studies (including more than 30 conducted by the Pennsylvania DOH alone) showing a lack of association between radiation releases at TMI and health effects on the people and environment around TMI, apprehension and distrust still exist.

Researchers at the University of North Carolina recently reevaluated the data from the Three Mile Island Public Health Fund study.¹⁴ The reevaluation study confirmed the original statistical findings but offered a different interpretation of the results.

The earlier study chose to concentrate on those cancers believed to be most radiosensitive—and especially on leukemia, which has been found to be generated by low-dose radiation and which has a short latency period.¹⁵ Because the original researchers found no increase in those cancers most susceptible to radiation, they determined that the radiation released by the accident did not cause excess cancers.

The North Carolina researchers chose to look, instead, at all cancers. Because they saw small increases for lung cancer and non-Hodgkin's lymphoma, the North Carolina researchers concluded that cancer risk did increase.

Non-Hodgkin's lymphoma is not thought to be a cancer commonly induced by radiation, however; and the North Carolina researchers' findings for lung cancer are inconsistent with the magnitude of the exposure. In addition, lung cancer due to radiation requires years to develop; it is unlikely, therefore, that the lung cancers the North Carolina researchers found were caused by the TMI radiation exposure. (Two recent studies of people living near nuclear installations in England and Wales found no increased risk for either lung cancer or non-Hodgkin's lymphoma.^{16,17}) The scientific weight of evidence thus continues to support a conclusion of no effect from the radiation released by the TMI accident.

Although the maximum dose estimated to have been received at TMI is less than the dose a person normally receives each year from natural background radiation, the TMI accident continues to evoke public fear. Memories of the accident still bolster opposition to nuclear power in the United States.¹⁸ It is particularly noteworthy, however, that the only measurable effect of the TMI accident was the affected population's increased level of stress—stress brought about by their unfounded fear that they were being exposed to harmful levels of radiation.

Today TMI's Unit 2 remains in monitored storage, even after an approximately \$973 million cleanup program. TMI Unit 1 resumed operations in October 1985 and still services parts of Pennsylvania and New

Jersey. In 1989 TMI Unit 1 received an efficiency rating of 100 percent, making it the number-one nuclear reactor in the world for cost efficiency.¹⁹

- 1 Flynn CB. Local public opinion. *Ann NY Acad Sci.* 1981; 146–151.
- 2 Ad Hoc Population Dose Assessment Group. Preliminary dose and health impact of the accident at the Three Mile Island nuclear station. *Nucl Safety.* 1979; 20:591–594.
- 3 Maxon HR. Fallout at Three Mile Island. *Ann Internal Med.* 1979; 91(3):486.
- 4 Upton AC. Health impact of the Three Mile Island accident. *Ann NY Acad Med.* 1981:63–75.
- 5 Committee on Federal Research into the Biological Effects of Ionizing Radiation. *Follow-up studies on biological and health effects resulting from the Three Mile Island nuclear power plant accident of March 28, 1979.* NIH Publication No. 79-2065. Washington, DC: U.S. Department of Health, Education, and Welfare, 1979.
- 6 Fabrikant JI. Health effects of the nuclear accident at Three Mile Island. *Health Phys.* 1981; 40:151–158.
- 7 Goldhaber MK, Staub SL, Tokuhata GK.. Spontaneous abortions after the Three Mile island nuclear accident: A life table analysis. *Am J Public Health.* 1983; 73(7):752–759.
- 8 Bratz JR, Tokuhata GK, Kim JS, et al. Three Mile Island pregnancy outcome study: Final report (abstract). Presented at the 116th annual meeting of the American Public Health Association, Boston, MA, November 16, 1988.
- 9 Health studies find no cancer link. *GPU Backgrounder.* July 1996.
- 10 Houts PS, Tokuhata GK, Bratz J, Bartholomew MJ, Sheffer KW. Effect of pregnancy during TMI crisis on mothers' mental health and their child's development. *Am J Public Health.* 1991; 81(3):384–386.
- 11 Hatch MC, Wallenstein S, Beyea J, Nieves JW, Susser M. Cancer rates after the Three Mile Island nuclear accident and proximity of residence to the plant. *Am J Public Health.* 1991; 81(6):719–724.
- 12 Hatch MC, Beyea J, Nieves JW, Susser M. Cancer near the Three Mile Island nuclear plant: Radiation emissions. *Am J Epidemiol.* 1990; 132(3):397–412.
- 13 Rambo, SH. Order and Judgment. Chief Judge, U.S. District Court for the Middle District of Pennsylvania, Harrisburg, PA. June 7, 1996.
- 14 Wing S, Richardson D, Armstrong D, Crawford-Brown D. A reevaluation of cancer incidence near the Three Mile Island Nuclear Plant: The collision of evidence and assumptions. *Environ Health Perspectives.* 1997; 105(1):52–57.
- 15 Committee on the Biological Effects of Ionizing Radiation. *The effects on populations for exposure to low levels of ioniz-*

ing radiation: 1980 (BEIR III). Washington, DC: National Academy Press, 1980.

- 16 Forman D, Cook-Mozaffari PH, Darby S, et al. Cancer near nuclear installations. *Nature*. 1987; 329:499–505.
- 17 Cook-Mozaffari PH, Darby SC, Doll R, et al. Geographical variation in mortality from leukemia and other cancers in England and Wales in relation to proximity to nuclear installations, 1969–78. *Br J Cancer*. 1989; 59:476–485.
- 18 Shulman S. Legacy of Three Mile Island. *Nature*. 1989; 338:190.
- 19 GPU Nuclear. *TMI-1 Operations Fact Sheet*. July, 1996.

12. Asbestos in Hair Dryers, 1979

Background

Unlike the other substances discussed so far, asbestos—a substance used widely in countless industrial applications for most of the 20th century—is a proven human carcinogen.

“Asbestos” is the name given to a group of naturally occurring minerals. Four types of asbestos have been commonly used in industry: chrysotile, or “white asbestos,” which consists of curly, flexible fibers; amosite; crocidolite; and anthophyllite. The latter three types consist of needlelike fibers and are collectively known as amphiboles.¹

By the mid 1960s it had become clear that occupational exposure to asbestos, and especially to amphiboles, was capable of causing several serious diseases: asbestosis, a scarring of lung tissue; lung cancer (especially among those who also smoke cigarettes); and mesothelioma, a cancer of a membrane surrounding the lungs.² As a result, emissions of these minerals were placed under tight control by the EPA in the early 1970s,³ and their use subsequently declined.

The Scare

In June 1978 a freelance photographer contacted reporters at WRC-TV in Washington, DC, and presented them with a hair dryer, complaining that it was spraying asbestos on prints he was drying with it. So reporter Lea Thompson (now the consumer reporter on *Dateline* NBC) contacted the federal Consumer Products Safety Commission. The commission informed her that, according to the most recently commissioned study, only one manufacturer was using asbestos in its hand-held dryers, and that manufacturer was phasing the asbestos out.

Still skeptical, Thompson’s producer sent the

photographer’s dryer to a testing lab in Rockville, Maryland. The lab found that it did, indeed, spray asbestos—as did six of 33 other hairdryers the station also sent to the lab. So, on March 29, 1979, WRC broadcast their story, declaring that 20 percent of hair dryers were spewing a known carcinogen.⁴

The Reaction

Congress immediately called hearings on hair dryers, with Thompson as the star witness. Department stores pulled some dryers off the shelves.⁴ The Consumer Product Safety Commission acknowledged that their previous study had been in error⁵ and began considering a ban on asbestos in hair dryers. Even before this action could be taken, however, nearly every company that manufactured hair dryers agreed to retrofit the models containing asbestos⁶ and to accept, at company expense, the return of dryers containing asbestos, which they would replace with models containing mica. This action was taken “to alleviate public concern,” even though both industry leaders and the government’s National Institute of Environmental Health Sciences questioned whether the amount of asbestos used in hand-held dryers posed any risk. Industry and government alike also questioned the validity of the Rockville lab study due to the small size of the sample.⁷

Conclusion

Since asbestos-containing dryers were pulled from the market before any conclusive data were in, it is unclear whether there ever was a risk from these products. In this particular case, “an adequate, nontoxic alternative”⁷ was available. The media’s fixation on the dangers of asbestos in the 1970s made it difficult to put asbestos-related risks in perspective, however—even though failure to do so may actually endanger public health (see “Asbestos in Schools” on page 43).

¹ National Cancer Institute. *Cancer Facts: Questions and Answers About Asbestos Exposure*. November 30, 1995.

² Elmes PC. The epidemiology and clinical features of asbestosis and related diseases. *Postgrad Med J*. 1966; 42:623–635.

³ Craighead JE, Mossman BT. The pathogenesis of asbestos-associated diseases. *N Engl J Med*. 1982; 306(24):1446–1455.

⁴ We were all so excited, says Thompson. *People*. April 16, 1979:15–16.

⁵ And federal officials learn of hazards in homes. *Science*. April 20, 1979:285.

⁶ Environmental Defense Fund. The campaign to end use of deadly asbestos. *EDF Letter*. Vol XIX, No. 3, June 1988:1–2.

⁷ Powledge F. The asbestos pistol. *The Nation*. January 5–12, 1980:14–15.

13. 2,4,5-T, 1979

Background

2,4,5-trichlorophenoxyacetic acid (2,4,5-T) was first developed as a herbicide in 1948. It soon was in wide use as a weed killer, both in agriculture and along highways and railroads. Its “crop specificity”—that is, its ability to affect undesirable plants while sparing crops—and its ability to increase crop yields on marginal farmlands led even the author of a highly negative newspaper article to describe 2,4,5-T as “one of the building blocks of the Green Revolution”¹ that brought about a dramatic increase in the world’s food supply in the post–World War II era.

2,4,5-T first received negative attention in the Vietnam War era of the late 1960s because of its association with Agent Orange (a mixture of 2,4,5-T and a related chemical, 2,4-D). After a 1969 study showed that large doses of 2,4,5-T were teratogenic—that is, caused birth defects—in mice, the USDA announced in April 1970 that it would cancel the use of 2,4,5-T for most domestic food crops.

A subsequent review by the National Academy of Sciences found that the earlier study had used batches of 2,4,5-T contaminated with levels of dioxin 30 times greater than the levels present in the 2,4,5-T actually used in manufacturing. The NAS review also found that “pure” 2,4,5-T did not produce similar effects in lab animals except at toxic doses. The NAS committee recommended overturning the ban.

This NAS decision was denounced by Ralph Nader, and the scientists on the academy’s review committee were publicly attacked by Nicholas Wade of *Science* magazine as allegedly having an “industry bias.” In August 1971 Environmental Protection Agency chief William Ruckelshaus announced that he was overruling the NAS committee and keeping the ban.² 2,4,5-T continued to be used for other purposes, however.

A study commissioned by the EPA in the late 1970s—after eight women in Alsea, Oregon, reported 10 miscarriages that they blamed on 2,4,5-T—showed insufficient evidence of a relationship between crop spraying and the miscarriages.^{3,4}

The Scare

In response to continued anxieties over the issue, the EPA commissioned a second study, Alsea II. In February 1979 the Alsea II researchers reached the opposite conclusion to Alsea I, declaring that “there was an unusually high number of spontaneous abortions [i.e., miscarriages] in the area,” and that the incidence of spontaneous abortions may be related to 2,4,5-T in that area.⁵ The EPA immediately invoked an emergency ban—the first time this action, the most drastic the agency can take, was ever invoked—on all uses of 2,4,5-T except on range fields and rice lands.⁶

The Reaction

Products containing 2,4,5-T were immediately pulled from the shelves. In New York, a state official threatened to quarantine any store that still sold any such product. But in a subsequent legal action taken by the chemical’s prime manufacturer in an attempt to reverse the ban, a Federal court judge in Michigan tellingly described the EPA’s data as “inconclusive.” The judge added that he would not have “ordered the emergency suspensions on the basis of the information of the EPA,” but that since “the EPA has been vested with broad powers in this area,” the ban would have to stand.⁷

There was also widespread questioning within the scientific community of the EPA’s use of data, and at least 18 reviews drew the opposite conclusion. Many reviewers noted that while the EPA claimed there had been a “peak” of miscarriages each June in the six years studied—a peak they attributed to the spraying done earlier each spring—in fact, this “peak” deviated from the norm in only one year.³

Any attempt to oppose the ban probably became moot later in 1979, however, after two television documentaries—“A Plague on Our Children,” produced by Boston PBS affiliate WGBH, and “The Politics of Poison,” produced by San Francisco station KRON—blamed 2,4,5-T for causing miscarriages. The television reports did not contain any new scientific information, but that mattered little in the face of heartrending anecdotal reports such as that of a woman who described her miscarried fetus as looking like “chopped hamburger.”^{8,9}

Conclusion

Approvals for all uses of 2,4,5-T were canceled in February 1985. What remains to emerge is not only any sound evidence linking the product to harmful health effects, but a replacement product that would be as effective as 2,4,5-T in eliminating the undesirable plants that otherwise grow wild in grasslands.

The current alternatives to 2,4,5-T require more applications to be effective, which renders them prohibitively expensive. One weed-control specialist predicted that the ultimate effect of the 2,4,5-T ban will be treatment of fewer acres, with a resulting loss of the grass production needed for livestock, increased soil erosion, and the fact that “some areas will grow so dense that there will be a loss of habitat for wildlife.”¹⁰

- ¹ Severo R. Two crippled lives mirror dispute on herbicides. *The New York Times*. May 27, 1979:1.
- ² Claus G, Bolander K. *Ecological Sanity*. New York: David McKay; 1977:238–253.
- ³ Witt, JM. *A Discussion of the Suspension of 2,4,5-T and the EPA Aalsea II Study*. Northeast Weed Science Society. January 8, 1980.
- ⁴ *Agent Orange and Dioxin*. American Medical Association; 1981:24.
- ⁵ Whelan E. *Toxic Terror*. 2nd ed. Buffalo, NY: Prometheus Books; 1993:285.
- ⁶ McFadden R. E.P.A., citing miscarriages, restricts 2 herbicides. *The New York Times*. March 2, 1979.
- ⁷ Severo R. Dispute over the risks of 2,4,5,T continues despite a federal ban. *The New York Times*. May 27, 1979:42.
- ⁸ *A Plague on Our Children*. WGBH Educational Foundation, 1979.
- ⁹ *The Politics of Poison*. KRON-TV, 1979.
- ¹⁰ *Toxic Terror*:284.

14. Coffee and Pancreatic Cancer, 1981

Background

Between 1974 and 1979 Harvard School of Public Health epidemiologist Dr. Brian MacMahon and his colleagues studied 369 patients admitted to New England hospitals with pancreatic cancer, along with 644 patients admitted for other diseases.¹ The purpose of the study was to determine whether alcohol and tobacco use increased the risk of pancreatic cancer. But MacMahon and his colleagues also made another “discovery”—one that was totally unexpected.²

The Scare

As MacMahon reported in the *New England Journal of Medicine* in March 1981, “an unexpected

association of pancreatic cancer with coffee consumption was evident.” Pancreatic cancer was 2.1 times as likely among people drinking up to two cups of coffee per day; it was 3 times as likely among those drinking five cups a day or more. If these trends were extrapolated to the general public, the report went on, “we estimate . . . the proportion of pancreatic cancer that is attributable to coffee consumption to be more than 50 percent.” While admitting that the findings did not provide a solid basis for such a conclusion, MacMahon himself said he was giving up coffee.²

The Reaction

Although pancreatic cancer is one of the most fatal of cancers (only about 4 percent of those diagnosed with it survive five years),³ and this story received extensive media coverage, the public reaction was not as great as might have been anticipated. Perhaps the reaction reflected the importance of coffee in most Americans’ daily ritual. Indeed, the price of coffee on the futures market went up slightly after the report was released.²

And the findings of the MacMahon study were almost immediately questioned by other researchers. For one thing, this study offered no plausible explanation as to why coffee might lead to pancreatic cancer. Since heavy tea drinkers showed no such risk, caffeine was presumably not to blame. Furthermore, both scientists and trade groups noted that by using hospital patients as a control group, the study ignored the fact that many of these hospitalized non-coffee drinkers—including many hospitalized for gastrointestinal disorders—may have stopped drinking coffee only when they became ill; the report made no attempt to track patterns of coffee drinking prior to illness.⁴ MacMahon acknowledged several months later that these criticisms were “reasonable.”⁵

Conclusion

MacMahon’s group repeated its study in 1986 and failed to confirm its previous findings. They reported, “in contrast to the earlier study, no trend in risk was observed for men or women.”⁶ In addition, subsequent animal tests and epidemiological studies have failed to indicate any link between coffee and cancer.^{7–13} The American Cancer Society agrees, “the most recent scientific studies have found no relationship at all between coffee and the risk of pancreatic, breast, or any other type of cancer.”¹⁴ This brief scare illustrates the danger of putting too much credence in a single study without analyzing any possible biases or confounding factors. Meanwhile, as a walk through any fashionable neigh-

borhood will show, coffee consumption continues to be strong, particularly among young, health-conscious people.

- ¹ MacMahon B, Yen S, Trichopoulos D, Warren K, Nardi G. Coffee and Cancer of the Pancreas. *N Engl J Med*. 1981; 304:630–633.
- ² Coffee nerves. *Time*. March 23, 1981:117.
- ³ American Cancer Society. *Cancer Facts and Figures—1996*. Atlanta, GA: American Cancer Society. 1996.
- ⁴ Clark M. Coffee—a cancer culprit? *Newsweek*. March 23, 1981:87.
- ⁵ Cancer and coffee: concern percolates. *Science News*. July 4, 1981:6.
- ⁶ Hsieh CC, MacMahon B, Yen S, Trichopoulos D, Warren K, Nardi G. Coffee and Pancreatic Cancer (Chapter 2). *N Engl J Med*. 1986; 313:587–589.
- ⁷ Whelan E, Stare FJ. *Panic in the Pantry*. Rev ed. Buffalo, NY: Prometheus Books; 1992:168.
- ⁸ Curatolo PW, Robertson D. The health consequences of caffeine. *Ann Int Med*. 1983; 98:641–653.
- ⁹ Wynder EL, Hall NE, Polanksy M. Epidemiology of coffee and pancreatic cancer. *Cancer Res*. 1983; 43:3900–3906.
- ¹⁰ Nomura A, Heilbrun LK, Stemmermann GN. Prospective Study of Coffee Consumption and the Risk of Cancer. *J Natl Cancer Instit*. 1986; 76:587–590.
- ¹¹ Hiatt RA, Klatsky AL, Armstrong MA. Pancreatic cancer, blood glucose and beverage consumption. *Int J Cancer*. 1988; 41:794–797.
- ¹² Gordis L. Consumption of methylxanthine-containing beverages and risk of pancreatic cancer. *Cancer Let*. 1990; 52:1–12.
- ¹³ Bueno De Mesquita HB, Maisonneuve P, Moerman CJ, Runia S, Boyle P. Lifetime consumption of alcoholic beverages, tea and coffee and exocrine carcinoma of the pancreas: A population-based case-control study in the Netherlands. *Int J Cancer*. 1992; 50:514–522.
- ¹⁴ American Cancer Society. 1996 *Guidelines on Diet, Nutrition and Cancer Prevention*. Atlanta, GA: The American Cancer Society 1996 Dietary Guidelines Advisory Committee; 1996.

15. Times Beach, 1982

Background

Between 1969 and 1971 a chemical plant in Verona, Missouri, manufactured the antiseptic hexachlorophene. After the company went out of business in

1971, one of its suppliers hired a waste hauler to dispose of the plant's wastes. In May 1971 about 2,000 gallons of the waste, mixed with oil, were sprayed on a horse arena in the town of Moscow Mills to reduce dust and flies prior to a horse show. Additional oil was sprayed on the unpaved streets of the nearby town of Times Beach to reduce dust.¹

During the horse show a number of previously healthy horses became ill. Over the next few months a dozen horses showed symptoms of ill health, and the daughter of the horse arena's owner became ill. The Centers for Disease Control and Prevention (CDC) was contacted. Three years later the CDC concluded that the cause of these problems was the oil sprayed on the arena—oil that was found to contain 33,000 parts per billion of the chemical 2,3,7,8-TCDD, also known as dioxin. Nonetheless, the CDC concluded that the oil no longer posed a health risk because the soil in question had already been excavated and buried.²

Dioxin is a byproduct of various industrial processes and combustion. It is an animal carcinogen and teratogen—that is, it causes cancer and birth defects in animals. There is no evidence showing that it has either effect in humans. Indeed, studies of humans exposed to large doses of dioxin during industrial accidents—particularly in West Virginia in 1949, in Michigan in 1964, and in Italy in 1976—all showed no increased rates of cancer or birth defects in the following years. The only proven human health effect of dioxin exposure is a skin disorder called chloracne.³

In the fall of 1982 the EPA placed the Times Beach site on its list for soil sampling.² But on December 5, before the results of the soil-sample tests were in, the residents of Times Beach faced another crisis: the worst flood in the town's history. The deluge damaged virtually every one of the town's 800 dwellings.⁴

The Scare

On December 23, just as the flooded-out Times Beach residents were about to return to their homes, the EPA findings were released: The soil in some parts of the community showed up to 100 parts per billion of dioxin.² This was less than 1 percent of what had been found around the horse arena eight years earlier, but it was still 100 times higher than what the EPA considered a safe level (a level corresponding to one extra case of cancer per million people over a 70-year exposure), presuming that children would be ingesting dirt in dioxin-contaminated areas.⁵ It was unclear what effect the flooding had had on the dioxin levels. Nevertheless, the CDC immediately warned Times Beach residents not to return to their homes.⁶

The Reaction

Most residents heeded the warning. The 200 or so who did return came back to find EPA workers in plastic, spacesuit-like clothing taking soil samples and attempting to repair water mains, even as residents walked around without any such protection. County police fled the town. Looting broke out. A psychologist described the mental condition of most residents as a “state of shock.”⁷

Initial CDC studies of Times Beach residents showed no unusual health problems—not even chloracne, typically the hallmark of dioxin exposure.⁵ And preliminary EPA silt samples taken after the flood showed far lower levels of dioxin exposure.¹ Nonetheless, in February 1983 the EPA announced that it would buy all the homes in Times Beach—the first such buyout under Superfund—at a cost of \$33 million.⁸

To the weary residents of Times Beach, this news came as a relief. Scientific authorities were more skeptical, however. A few months later the American Medical Association adopted a resolution at its convention criticizing the “irrational reaction and unjustified public fright and . . . erroneous information about the health hazards of dioxin.” And *Science* magazine noted that dioxin, when bound tightly to soil, as was the case at Times Beach, “does not constitute much of a hazard.”⁹

Conclusion

Subsequent follow-up tests on the Times Beach residents continued to show no evidence of increased illness.^{10,11} And in 1991—after an EPA reassessment showed that dioxin exposure at low doses may have no adverse health effects, even in rats—the government did something it seldom does: It admitted it made a mistake. Dr. Vernon Houk, the CDC official who had ordered the evacuation, acknowledged, “Given what we know about this chemical’s toxicity and its effect on human health, it looks as though the evacuation was unnecessary. Times Beach was an overreaction. It was based on the best scientific information we had at the time. It turns out we were in error.”¹²

EPA chief William Reilly agreed: “We are seeing new information on dioxin that suggests a lower risk assessment for dioxin should be applied. . . . There isn’t much precedent in the federal establishment for pulling back on a judgment of toxicity. But we need to be prepared to adjust, to raise and lower standards, as new science becomes available.”¹³

The total cost of the Times Beach buyout and cleanup has come to at least \$138 million. And, incredibly, there are signs that the federal government has not

learned its lesson: Recently, the federal government agreed to pay \$18 million to relocate residents of a Florida town where the dioxin level was found to be 0.2 parts per billion—far less than the standard used at Times Beach. The Florida decision—which one senior EPA scientist claimed was ordered by the White House “in response to political pressures in an election year”—was described by another EPA engineer as likely to cause many other communities similarly to demand relocation.¹⁴

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- 1 Garmon L. Dioxin in Missouri: troubled Times. *Science News*. January 22, 1983:61–63.
 - 2 Sun M. Missouri’s costly dioxin lesson. *Science News*. January 28, 1983:367–368.
 - 3 Letts R. *Dioxin in the Environment*. New York: American Council on Science and Health; 1991:5–12.
 - 4 Lerner M. The trouble at Times Beach. *Newsweek*. January 10, 1983:24.
 - 5 *Dioxin in the Environment*:14.
 - 6 The “river rats” want to stay. *Time*. January 10, 1983.
 - 7 County pulls police out of poisoned Missouri town. *The New York Times*. January 1, 1983:A18.
 - 8 Garmon L. The buying of Times Beach: a town unfit for human beings. *Science News*. February 26, 1983:132–133.
 - 9 Chlorinated dioxins. *Science*. June 24, 1983.
 - 10 Edgar M. Times Beach residents pass first dioxin tests. *St. Louis Globe-Democrat*. July 1, 1983.
 - 11 Council for Agricultural Sciences and Technology. *The Missouri Dioxin Controversy: Scientific Overview*. April 1983:3
 - 12 Schneider K. Times Beach warning: regrets a decade later. *The New York Times*. August 15, 1991.
 - 13 Napier K. Re-evaluating dioxin: the implications for science policy. *Priorities*. Winter 1992:35–37.
 - 14 Nossiter A. Villain is dioxin. Relocation is response. But judgment is in dispute. *The New York Times*. October 21, 1996:A12.

16. EDB, 1983

Background

Ethylene dibromide (EDB) was used as far back as the 1930s: primarily as an additive to prevent “knock” in leaded gasoline, but also as an agricultural fumigant to control nematodes (worms) in citrus fruit, to prevent insect and mold infestation of grain stored over long periods of time, to eliminate insects from the milling machinery used to grind grain into flour, and to control tropical fruit flies in fruit. Because EDB evaporates quickly after use, for many years it was exempt from the normal regulations requiring tolerance levels for pesticide residues. The exemption was based on the belief that the EDB would have evaporated completely by the time a treated agricultural product reached the consumer. As the ability to detect residues became more sensitive in the 1970s, however, it was discovered that minuscule levels of EDB residues were indeed present in some food products.¹

The Scare

In September 1983 the Environmental Protection Agency announced that EDB residues had been found in groundwater in Florida, and that this posed an “imminent hazard” to human health. There was no doubt that EDB was an animal carcinogen²; indeed, it was one of the strongest animal carcinogens found in the range of synthetic chemicals. The degree of hazard from any substance is determined by both its potency and its dose, however; and the rodents fed EDB ingested 250,000 times more, proportionately, than the average human dose. Nevertheless, the EPA moved to ban the use of EDB as a soil fumigant and announced plans to phase out the other agricultural uses of EDB.³

In December 1983, after high concentrations of EDB were found in processed cornmeal, grits, and hush puppies, the state of Florida banned the sale of any food containing EDB residues.⁴ In February 1984 the EPA announced an immediate ban on the further use of EDB in grain, with the use of EDB in fruit to be phased out over the next six months. Existing foods already treated with EDB could only be sold if the residues were below newly set tolerance levels.⁵

The Reaction

The EPA’s action led to a “pattern of confusion,” according to one newspaper report. Some states rigorously enforced the EPA guidelines. Others, such as Florida, set even stricter guidelines and pulled any product with detectable residue off the shelves. Still others took no action whatsoever. In Florida, one com-

munity incurred a cost of \$1 million to replace water wells judged contaminated; in another community the residents were forced to travel 10 miles to buy “uncontaminated” water.⁶

Much of the scientific community was more skeptical, however. Scientists noted that EDB had never been proven to cause cancer in humans and pointed out further that if it was a carcinogen, it was a far less potent one than many naturally occurring substances (such as aflatoxin, which is commonly found and legally permitted in peanuts at up to 20 parts per billion). Even the head of the EDB regulation team at the EPA declared that “the press has given the public a sense of panic about EDB that is undeserved.”⁷

A year later an outside consultant hired by the EPA concluded that the EPA had failed to communicate clearly the facts about the actual risk of EDB and thus had helped contribute to the unwarranted panic.⁴

Conclusion

The amount of EDB to which humans were being exposed was infinitesimal. The EPA’s estimate before the ban—an estimate that many observers felt was far greater than actual exposure—was five to ten micrograms (millionths of a gram) per day. In comparison, the average American ingests 140,000 micrograms of table pepper per day; and pepper contains the potent animal carcinogen safrole. Furthermore, studies of workers engaged in EDB manufacturing—people exposed to doses five to ten thousand times the average doses ingested by consumers—failed to show a higher-than-average incidence of cancer.⁸

Since the removal of EDB from the marketplace, fruit and grain producers have had to use one of three alternatives to prevent insect infestation: methyl bromide, carbon disulfide mixed with carbon tetrachloride, or phosphine. The first two have been shown to be animal carcinogens; the third has never been subject to chronic toxicity testing and is highly flammable.⁹ None of the three is as effective a fumigant as EDB, and all three need to be applied in far greater doses to be effective.⁸

The use of low-dose gamma irradiation has been approved by the FDA as a safe alternative means of disinfestation,^{10,11} but irradiation still faces hurdles in industry and public acceptance, due in part to the activities of advocacy groups that have raised safety concerns about irradiation—concerns not supported by scientific fact. In any case, food irradiation facilities have not yet been built on a large scale.

EDB may have been forgotten by almost all consumers, but the ban has left one visible—and revolt-

ing—legacy. Anecdotal reports from throughout the country over the last few years indicate an increasing number of flying insects in the pantry and live bugs in newly opened bags of flour, corn meal, and similar products—a finding that, if it is consistently true, might be attributable to the loss of EDB and its replacement by less effective alternatives. And this is not merely an aesthetic unpleasantness: many of these bugs may themselves carry highly toxic and carcinogenic molds, spores, and microbes. It remains to be seen whether this poses a new, real human health hazard.

- ¹ Craigmill AL. Ethylene dibromide and methyl bromide. *Environmental Toxicology Newsletter*. 2(1); August 1981.
- ² Wong LC, et al. Carcinogenicity and toxicity of 1,2-dibromoethane in the rat. *Toxicol Appl Pharmacol*. 1982; 63(2):155–165.
- ³ Gold LS, et al. Rodent carcinogens: setting priorities. *Science*. 1992; 258:261–265.
- ⁴ Pesticide scare laid to communication gap. *The New York Times*. March 12, 1985:A18.
- ⁵ Ban on pesticide is weighed by the U.S. *The New York Times*. February 3, 1984:1.
- ⁶ States' actions on EDB in food resulting in patterns of confusion. *The New York Times*. February 18, 1984:1.
- ⁷ Some experts say EDB risk is small. *The New York Times*. February 18, 1984:1.
- ⁸ Havender W. *Ethylene Dibromide (EDB)*. New York: American Council on Science and Health; May 1984.
- ⁹ Whelan E. *Toxic Terror*. 2nd ed. Buffalo, NY: Prometheus Books; 1993:85.
- ¹⁰ Greenberg R. *Irradiated Foods*. 4th ed. New York: American Council on Science and Health; 1996:7.
- ¹¹ Council for Agricultural Science and Technology. Radiation Pasteurization of Food. *Issue Paper*. April 1996; 7:1–10.

17. Alar, 1989

Background

Alar was the trade name for daminozide, a growth regulator of ripening apples first developed in the 1960s. It was used to prevent apples from dropping off the tree prematurely and rotting prior to harvest. Alar went through two years of FDA carcinogenicity tests and was approved as safe by the FDA in 1968.

In 1973 a study showed that UMDH, a byproduct of Alar, caused blood vessel, lung, and kidney tumors in mice. Subsequent EPA analysis of this study declared it to be flawed, however, because the mice had been treated with such a high dosage of UMDH that the “maximum tolerated dose” (MTD) was exceeded by eightfold and the toxicity of the high dose might have caused the tumors.¹ (For any substance, no matter how benign, a maximum tolerated dose exists above which the substance will damage tissues merely from its high concentration.² Current guidelines for conducting bioassays for carcinogenic effects specify that the maximum tested dose should not exceed the maximum tolerated dose.)

Further tests conducted by the National Cancer Institute in 1978 and by the EPA in 1986 also failed to indicate that Alar was a carcinogen.³ Nevertheless, the EPA insisted on further tests—tests in which mice were given doses of UMDH at four to eight times the MTD,² or 133,000 to 266,000 times the highest estimated daily intake of UMDH by preschool children.⁴ To put it another way, a child would have to drink 19,000 quarts of apple juice a day for life to equal this degree of exposure.⁵

At four times the MTD one mouse out of a group of 45 developed a benign lung tumor. At eight times the MTD—close to the level of the discredited 1973 study—11 out of 52 mice developed either benign or malignant tumors. Unfortunately for the other mice in the experiment, 80 percent of the mice died from toxicity, not cancer.

The EPA acknowledged that the study may have been compromised by such high doses but concluded nonetheless that Alar posed a risk of 45 cancers per one million exposed humans. The agency ordered Alar’s manufacturer, Uniroyal, to phase out its use by July 31, 1990.

The Scare

This gradual phaseout didn’t satisfy the Natural Resources Defense Council, however. The NRDC had been calling for years for an EPA ban on Alar. Then, on February 26, 1989, over 50 million Americans saw a segment on CBS’s *60 Minutes* called “A is for Apple.” The program labeled Alar “the most potent cancer-causing agent in our food supply” and called it a cause of childhood cancer.³ The source for these allegations was the NRDC report *Intolerable Risk: Pesticides in Our Children’s Food*, which the NRDC had prereleased exclusively to CBS with the assistance of Fenton Communications, a public-relations firm hired to help coordinate the effort.²

The Reaction

Fenton and the NRDC achieved their goal to have “the [Alar] ‘story’ have a life of its own” far beyond their wildest dreams. In the days following the *60 Minutes* broadcast the claims in the NRDC report were repeated by virtually every major print and broadcast outlet. Fenton also enlisted some high-profile help—actress Meryl Streep and the wife of NBC newsman Tom Brokaw, who formed a group called Mothers and Others For Pesticide Limits.⁶

Public reaction verged on the hysterical. One consumer called the International Apple Institute to ask if it was safe to discard apple juice in the kitchen sink, or whether it was necessary to take it to a toxic waste dump. A parent sent state troopers chasing after her child’s school bus to confiscate the forbidden fruit her child was carrying.⁷ Fenton and NRDC even received an added bonus when the FDA announced that some grapes from Chile were tainted. Although the Chilean fruit had nothing to do with Alar, the incident fed the general atmosphere of panic.⁸

Not everyone joined in the chorus of alarmism, however. Many science reporters questioned the methodology of the studies on which the EPA and NRDC had based their conclusions. Even other environmentalist groups (such as the Environmental Defense Fund) declared that the NRDC’s data were inconclusive. And a few days after the *60 Minutes* report, the National Research Council declared that there was “no evidence that pesticides or natural toxins in food contribute significantly to cancer risk in the United States.”⁹

In an unusual step, *60 Minutes* devoted a second broadcast to the issue. Several critics of the earlier program, including ACSH president Dr. Elizabeth Whelan, appeared on the second show. But *60 Minutes* correspondent Ed Bradley spent much of the program impugning the motives of the critics, suggesting that they were being influenced by the chemical industry.¹⁰

Finally, under pressure from apple growers—who were suffering losses whether or not they used Alar—Uniroyal withdrew Alar from use on edible products in June of 1989.¹¹

When the dust had cleared, apple growers had lost an estimated \$250 million. Apple processors had lost another \$125 million. Some growers who had owned their farms for generations lost them to foreclosure. And the U.S. taxpayer lost, too: The U.S. Department of Agriculture made an emergency purchase of \$15 million worth of leftover apples.¹²

This was the end of Alar—but it was not the end of the story. Since 1989 a stream of criticism by inde-

pendent scientists and scientific authorities has continued to be leveled at the NRDC and the EPA. In Great Britain a group appointed by Parliament declined to ban Alar, stating that, unlike the EPA, “we don’t always make the assumption that animal data are transferable to man” or that high-dose responses can predict low-dose responses.¹³ A United Nations panel of scientists from the World Health Organization and the Food and Agriculture Organization also concluded that Alar was not oncogenic (tumor-causing) in mice.²

In subsequent years, the anti-Alar campaign has been criticized on the editorial page of *Science* magazine (which compared the NRDC’s actions to those of an embezzler)¹⁴; by former Surgeon General C. Everett Koop,¹⁵ the Chairman of the National Safe Kids Campaign; and by the senior medical advisor to the American Medical Association.¹⁶ In a 1993 article in *ECO* magazine, *New York Times* reporter Keith Schneider, a veteran of the environmental movement, described the NRDC report on Alar as “specious” and as having “not much scientific evidence.”¹⁷

Even the EPA—although bound by the Delaney clause definition of a carcinogen and so unable to change its judgment on Alar—nonetheless released a new toxicological analysis in late 1991 that showed Alar to be only half as potent as earlier estimates had indicated.¹⁸

The NRDC continued to defend its action, noting that the Supreme Court had recently refused to hear an appeal of a defamation suit filed by Washington state apple growers against CBS, the NRDC, and Fenton.¹⁹ However, the court’s denial had been based on the conclusion that as long as the EPA held Alar to be a carcinogen, the CBS report could not be proved false.²

Alar itself is clearly too stigmatized ever to be returned to the market, but there is no doubt that public perception has changed since 1989. The climate has changed to the point where a writer in the *Columbia Journalism Review* recently bemoaned the continued attention given to exposing the NRDC’s false claims about Alar. The *CJR* piece alleged that this was due to a “concerted disinformation campaign by industry trade groups,”²⁰ but did so without providing a single example of how the critics of the anti-Alar campaign were in error or of how “industry trade groups” were responsible for pointing out the anti-Alar campaign’s scientific inaccuracies.

The *Columbia Journalism Review* article was correct, though, in pointing out that the media have become far more circumspect about giving uncritical publicity to health scares without first consulting with mainstream scientists. To cite one recent example, *Our*

Stolen Future—an alarmist book alleging that chlorinated compounds in the environment pose grave health risks to humans—was published in the spring of 1996. The book’s release was handled with public-relations assistance from Fenton Communications. Most media reports on *Our Stolen Future* included the views of scientists skeptical about the book’s claims and also made note of Fenton Communications’ activities during the Alar scare.²¹

Conclusion

Back on the apple farm the effects of Alar’s loss are still being felt. Farmers from Ohio to New Hampshire are reporting decimation of their crops and, ironically, a need to use additional pesticides to enable the trees to hold their fruit.²²

And Alar lives on as a symbol: a symbol, first, of a model of risk assessment increasingly under criticism from scientists as having no relation to actual human cancer risks and, second, of the manipulation of the media by interest groups acting in contravention of the consensus of mainstream science.

The fallout from the Alar campaign was also a blow to the entire “mouse-as-a-little-man” premise. As the public followed the Alar story, it learned of the basis for the government’s risk estimates—and it began to see how poorly such tests actually predicted human cancer risks. More generally, many consumers started to grow skeptical of the countless health scares popping up almost daily in the media.

¹ Rosen J. Much ado about Alar. *Issues in Science and Technology*. 1990; 7:85–90.

² Smith K. *Alar Five Years Later*. New York: American Council on Science and Health; 1994.

³ Winter C, Craigmill A, Stimmann M. NRDC report and Alar. *Environmental Toxicology Newsletter*. 9(2); May 1989.

⁴ Marshall E. A is for Apple, Alar, and . . . Alarmist? *Science*. 1991; 254:20–22.

⁵ Whelan E, Stare FJ. *Panic in the Pantry*. Buffalo, NY: Prometheus Books; 1992:4.

⁶ Whelan E. *Toxic Terror*. 2nd ed. Buffalo, NY: Prometheus Books; 1993:189–190.

⁷ *Panic in the Pantry*. 1992:2.

⁸ *Toxic Terror*:190.

⁹ *Washington Post*. March 1, 1989.

¹⁰ *Toxic Terror*:191–194.

¹¹ *Toxic Terror*:198.

¹² Rice J. Speech to Virginia Horticultural Society by International Apple Institute Chairman. January 1990.

¹³ Evaluation on Daminozide. U.K. Ministry of Agriculture, Fisheries and Food, Pesticides Safety Division; 1989.

¹⁴ Koshland DE. Credibility in science and the press. *Science*. 1991; 254:629.

¹⁵ Koop CE. Letter to Elizabeth M. Whelan. December 20, 1991.

¹⁶ American Medical Association, Council on Scientific Affairs. Report of the Council on Scientific Affairs; diet and cancer: where do matters stand? *Arch Intern Med*. 1993; 153:50–56.

¹⁷ Schneider K. A policy that set the world standard goes off track. *ECO*. June 1993:17–22.

¹⁸ Marshall E. A is for apple, Alar, and . . . alarmist? *Science*. 1991; 254:20–22.

¹⁹ Letter to the editor. *Wall Street Journal*. March 29, 1996.

²⁰ Negin E. The Alar ‘scare’ was real. *Columbia Journalism Review*. September–October 1996:13–15.

²¹ For one example, see: Clamorous pro and con campaign heralds book’s launch. *Wall Street Journal*. March 7, 1996:B1.

²² *Toxic Terror*:194–196.

18. Electric Blankets, 1989

Background

The technology used to make electric blankets—first designed by General Electric in the mid-1940s—was adapted from that used to make “electrically warm” suits for flyers in World War II.¹ Electromagnetic field (EMF) exposure is ubiquitous in modern societies; people’s total EMF exposure includes occupational, residential, and appliance uses. Electric blankets (like other electrical appliances) are sources of residential magnetic field exposure. Despite substantial agreement within the scientific community that EMF health effects remain unproved,² public anxiety persists. Electric blankets have come under particular scrutiny because, unlike other appliances, they are used close to the body

for extended periods of time.

The Scare

The first report of a possible relationship between childhood cancer mortality and high-current residential power lines—a report suggestive of elevated EMF levels—came in 1979.³ Then, in 1989, *Consumer Reports*, noting the uncertainties surrounding EMF exposure, recommended that children and pregnant women avoid electric blankets and mattress pads in favor of comforters. A 1990 study added to the uneasiness when researchers reported finding a modest increased risk of childhood cancer in relation to the mother's use of an electric blanket during pregnancy and, to a lesser extent, the child's use of electric blankets.⁴

The Reaction

After remaining stable for many years, sales of electric blankets dropped by 11 percent, to five million units, in 1989.¹ Eighteen congressmen, responding to the anxiety of their constituents, asked that electric blankets be labeled as hazardous for children and pregnant women. As a result, all U.S. blanket manufacturers now include warnings with their products, advising that children not be permitted to use electric blankets.

Conclusion

Subsequent studies of brain tumor occurrence and electric blanket use have not supported the 1990 study.^{5–7} A multicenter, large-scale study performed in 1996 found no evidence to support a relationship between brain-cancer occurrence in children and EMF exposure from the use of electric blankets and heated water beds.⁸ In addition, the 1996 study showed that maternal use of electric blankets or heated water beds was not associated with subsequent brain-tumor risk in children. A large-scale study conducted by the National Cancer Institute and published in 1998 concluded that a causal relationship between childhood brain tumors and EMF from appliances, including electric blankets, is unlikely.⁹

Other studies have investigated the possible association of electric blanket exposure in adults with leukemia,^{10,11} testicular cancer,¹² and breast cancer.^{13,14} All of these studies have shown that electric blankets are not associated with increased cancer risk. Additionally, the use of electric blankets during pregnancy has been found to be unrelated to fetal growth,¹⁵ to risk of spontaneous abortions,¹⁶ or to birth defects.¹⁷

Electric blankets have been found to contribute substantial magnetic field exposure to children¹⁸; but if EMF exposure is a risk factor for brain-tumor occur-

rence, some suggestion of an increased risk among users would have been found—and none has been.

Despite these encouraging findings, the electric blanket industry did not escape unscathed. Of the original four major U.S. manufacturers of electric blankets, only Sunbeam-Oster remains a major manufacturer. Moreover, in response to the controversy over the effects of EMF on health, blanket manufacturers have redesigned their product. Electric blankets were, in fact, the first consumer product to undergo such a change.¹ All of the electric blankets and pads tested by *Consumer Reports* in a recent evaluation registered EMF levels close to “the ‘background’ level produced by any house’s wiring.”¹⁹

Today the warning labels remain, but the weight of the scientific evidence indicates that pregnant women and children can once again sleep soundly as they cuddle under their electric blankets.

¹ Stix G. Field Effects: A health worry for electric blanket makers. *Scientific Am.* December 1990:122–123.

² National Research Council. *Possible Health Effects of Exposure to Residential Electric and Magnetic Fields.* Washington, DC: National Academy Press. 1996.

³ Wertheimer N, Leeper E. Electrical wiring configurations and childhood cancer. *Am J Epidemiol.* 1979; 109:273–284.

⁴ Savitz DA, John EM, Kleckner RC. Magnetic field exposure from electric appliances and childhood cancer. *Am J Epidemiol.* 1990; 131:763–773.

⁵ Bunin GR, Buckley JD, Boesel CP, et al. Risk factors for astrocytic glioma and primitive neuroectodermal tumor of the brain in young children: a report from the Children's Cancer Group. *Cancer Epidemiol Biomarkers Prev.* 1994; 3:197–204.

⁶ London SJ, Thomas DC, Bowman JD, et al. Exposure to residential electric and magnetic fields and risk of childhood leukemia. *Am J Epidemiol.* 1991; 134:923–937.

⁷ Gurney JG, Mueller BA, Davis S, Schwartz SM, Stevens RG, Kopecky KJ. Childhood brain tumor occurrence in relation to residential power line configurations, electric heating sources, and electric appliance use. *Am J Epidemiol.* 1996; 143:120–128.

⁸ Preston-Martin S, Gurney JG, Pogoda JM, Holly EA, Mueller BA. Brain tumor risk in children in relation to use of electric blankets and water bed heaters. *Am J Epidemiol.* 1996; 143:116–122.

⁹ Hatch EE, Linet MS, Kleinerman RA, et al. Association between childhood acute lymphoblastic leukemia and use of electrical appliances during pregnancy and childhood. *Epidemiol.* 1998; 9(3):234–245.

- 10 Preston-Martin S, Peters JM, Yu MC, et al. Myelogenous leukemia and electric blanket use. *Bioelectromagnetics*. 1988; 9:207–213.
- 11 Severson RK, Stevens RG, Kaune WT, et al. Acute nonlymphocytic leukemia and residential exposure to power frequency magnetic fields. *Am J Epidemiol*. 1988; 128:10–20.
- 12 Verreault R, Weiss NS, Hollenbach KA, et al. Use of electric blankets and risk of testicular cancer. *Am J Epidemiol*. 1990; 131:759–762.
- 13 Vena JE, Graham S, Hellman R, et al. Use of electric blankets and risk of postmenopausal breast cancer. *Am J Epidemiol*. 1991; 134:180–185.
- 14 Vena JE, Freudenheim, JL, Marshall JR, Laughlin R, Swanson M, Graham S. Risk of premenopausal breast cancer and use of electric blankets. *Am J Epidemiol*. 1994; 140:974–979.
- 15 Bracken MB, Belanger K, Hellenbrand, et al. Exposure to electromagnetic fields during pregnancy with emphasis on electrically heated beds: association with birthweight and intrauterine growth retardation. *Epidemiol*. 1995; 6:263–270.
- 16 Belanger K, Leaderer B, Hellenbrand K, et al. Spontaneous abortion and exposure to electric blankets and heated water beds. *Epidemiol*. 1998; 9:36–42.
- 17 Dlugosz L, Vena J, Byers T, Sever L, Bracken M, Marshall E. Congenital defects and electric bed heating in New York State: A register-based case-control study. *Am J Epidemiol*. 1992; 135:1000–1011.
- 18 Florig HK, Hoburg FJ. Power-frequency magnetic fields from electric blankets. *Health Phys*. 1990; 58:493–502.
- 19 Keeping warm in bed. *Consumer Reports*. November 1995:724–725.

19. Video Display Terminals, 1989

Background

Video display terminals (VDT), most of which are based on common television technology, were originally used in industry—notably for such tasks as airline seat reservations and stock control—and for military activities. Within the last two decades, however, the use of VDTs in the workplace has exploded. They have become a common sight, not only in offices, banks, and other business sites, but in our homes.

The Scare

On July 23, 1980, the *Toronto Globe and Mail* reported that four women in the classified advertising department of another Toronto newspaper, the *Star*, had given birth to children with birth defects. The defects including a cleft palate, complex heart defects, an underdeveloped eye, and club feet. All four mothers had worked with VDTs during the early stages of their pregnancies.¹

To defuse the rising anxiety among VDT workers, the Canadian Radiation Protection Bureau tested radiation emissions from the terminals. The acting director of the bureau stated: “The machines are safe. . . There’s absolutely nothing of any hazard emitted by VDTs.”² The Toronto Department of Health reported that “There is no scientific evidence whatsoever that radiation from VDTs is a health hazard even to pregnant women, nor is there any evidence that four abnormal births by Toronto Star VDT operators were caused by VDT radiation.”³

But one cluster of birth defects had been reported even before 1979. And by 1984 seven more clusters had been widely publicized. These involved workers in the offices of Canada’s Solicitor General in Ottawa (7 adverse outcomes in 8 pregnancies); at the office of the Attorney General in Toronto (10 in 19); at Air Canada’s offices in Montreal (7 in 13); at Sears, Roebuck in Dallas, Texas (8 in 12); at the Defense Logistics Agency in Atlanta, Georgia (10 in 15); at Pacific Northwest Bell in Renton, Washington (3 in 5); at Surrey Memorial Hospital in Vancouver, British Columbia (5 in 6); at United Airlines in San Francisco, California (18 in 132); and at Southern Bell, again in Atlanta (6 in 8). The problems included birth defects, spontaneous abortions, and premature births.⁴

The scare gained momentum in 1989 with the publication of *Currents of Death: Powerlines, Computer Terminals and the Attempt to Cover Up the Threat to Your Health*, a book that claimed EMFs had been proved unsafe beyond doubt. *The New Yorker* ran three lengthy excerpts from the book in July 1989.⁵

The Reaction

Meanwhile, in both North America and Europe the cluster findings were pitting management against employees. Pregnant employees at the *Montreal Gazette* refused to work on VDTs until the terminals could be tested to verify that they did not emit dangerous levels of radiation. The Federal Labour Minister of Canada endorsed a task force’s recommendation that pregnant workers should have the right to transfer without loss of seniority to jobs not requiring the use of

VDTs.⁴ In 1982 a public service arbitration board in Ontario ruled that the belief that radiation from a terminal could harm an unborn child was reasonable grounds for a pregnant government employee to refuse to work on a VDT.⁶ The Workman's Compensation Board of Quebec systematically supported and compensated every pregnant VDT user who wished to leave her job because she considered it potentially harmful to her fetus.⁷ In Sweden, pregnant women were allowed to request to leave jobs involving VDTs on the grounds of reducing worry.⁸

The culprit in the clusters was thought originally to be X radiation, but studies confirmed that levels of radiation around VDTs were, in fact, extremely low; in the overwhelming majority of cases, they were unmeasurable.⁹ The Canadian Health Protection Branch stated that "There is no scientific or medical evidence at the present time to indicate that any person, male or female, young or old, pregnant or not, [should] be concerned about radiation health effects from VDTs."¹⁰

Researchers' attention now turned to weak electromagnetic fields. Animal data partially supported these suggestions. Damage to chick embryos or fetal malformations in mice were reported after exposures to pulsed electromagnetic and magnetic fields,¹¹⁻¹³ but other results have been inconsistent. A review of the laboratory data indicates that the findings are inconclusive, but it is possible that under some experimental conditions adverse reproductive and developmental effects do occur in laboratory animals.¹⁴ The extrapolation of these findings to humans is tenuous at best, however.¹⁵

Two 1988 studies did find an increased risk of spontaneous abortion among female VDT workers,^{16,17} but researchers criticized both studies for biases and flawed study design.¹⁵ The World Health Organization (WHO)—after a thorough evaluation of the studies on VDT use and adverse pregnancy outcomes—concluded that there is no evidence of adverse effects of VDTs on pregnancies.¹⁸ Indeed, the number of studies that have failed to show a link between VDT use and adverse pregnancy outcomes is impressive.¹⁹⁻²³

Conclusion

No positive explanation has been found for the clusters, but miscarriages and other adverse reproductive outcomes are not evenly spread in time and space; some clustering is to be expected.²⁴ After careful examination and calculations, the Centers for Disease Control and Prevention concluded that the clusters likely were random occurrences.²⁵ Another research group determined that the clusters could be considered a "nat-

ural" phenomenon.²⁶ A large number of groups of women work with VDTs; the mere observation of a certain number of clusters among these groups is, therefore, not surprising. The observed clusters thus may have no epidemiological significance.

March 1991 saw the publication of the most detailed study of its kind on risk of spontaneous abortions and VDT use, a study performed by the National Institute for Occupational Safety and Health (NIOSH). After studying over 5,000 women over a six-year period, the NIOSH researchers concluded that the use of VDTs is not associated with an increased risk of spontaneous abortions.²⁷

In 1992 the Federal Committee on Interagency Radiation Research and Policy Coordination contracted the Oak Ridge Associated Universities to review all the VDT health risk literature. The authors of the *Oak Ridge Review* concluded that "There is no convincing evidence in the published literature to support the contention that exposures to extremely low frequency electric and magnetic fields generated by . . . VDT . . . are demonstrable health hazards." The committee pointedly stated that "no plausible biological mechanism is presented that would explain causality."²⁸

The Committee on Man and Radiation (COMAR) of the Institute of Electrical and Electronics Engineers (IEEE) reviewed the information as well. The IEEE committee concluded that the use of VDTs in the workplace is not a risk factor for either miscarriage or birth defects but added that VDT users do need to be aware of ergonomic problems—particularly possible eye strain and the effects of improper posture—which can be easily ameliorated.²⁹

The absolute safety of VDTs can be demonstrated only by the absence of even the smallest increased risk. This is impossible to demonstrate, however: A hazard can be shown to exist; the absence of a hazard cannot. In 1965, well before the current computer age began, the noted English industrialist Sir Leon Bagrit stated, "In putting automation into practice, its very novelty, its unfamiliarity is likely to arouse instinctive caution on the part . . . of organised labour."³⁰ This remark may best sum up the hysteria and uncertainty that occurred with the increasingly widespread use of VDTs.

¹ Work conditions probed at *Star* as defects found in 4 employees' babies. *Toronto Globe and Mail*. July 23, 1980.

² *Toronto Star*. July 26, 1980.

³ *Toronto Star*. July 31, 1980.

- 4 Foster KR. The VDT Debate. *American Scientist*. 1986; 74:163–168.
- 5 Brodeur P. *Currents of death: Power lines, computer terminals and the attempt to cover up their threat to your health*. New York: Simon and Schuster. 1989.
- 6 Slesin L, Zybko M. Video display terminals: health and safety. *Microwave News*. 1983:41–46.
- 7 Group de travail. *Les terminaux à écrans de visualisation et la santé des travailleurs*. Institut de recherche en santé et en sécurité du travail du Québec. IRSST, Montréal, Publication E-008, 1984.
- 8 Bergquist U, Knave B. Video display work and pregnancy—research in the Nordic countries. In: Pearce GB, ed. *Allegations of reproductive hazards from VDUs*. Loughborough: Human Technology. 1984:49–53.
- 9 Zuk WM, Stuchly MA, Dvorak P, Deslauriers Y. *Investigations of Radiation Emissions from Video Display Terminals*. Public Affairs Director, Department of Health and Welfare Canada, Report 83-EHD-91. 1983:16.
- 10 Liston AJ. *Information Letter*, December 4, 1985. Health Protection Branch, Health and Welfare Canada, 1985.
- 11 Delgado JMR, Leal J, Montegaudo JL, Gracia MG. Embryological changes induced by weak, extremely low frequency electromagnetic fields. *J Anat*. 1982; 134:533–551.
- 12 Ubeda A, Leal J, Trillo MA, Jimenez MA, Delgado JM. Pulse shape of magnetic fields influences chick embryogenesis. *J Anat*. 1983; 137:513–536.
- 13 Tribukait B, Cekan E, Paulsson LE. Effects of pulsed magnetic fields on embryonic development in mice. In Knave B, Wideback PG, eds. *Work with Display Units 86*, International Scientific Conference on Work with Display Units. Amsterdam: Elsevier Science Publication B.V. 1987:129–134.
- 14 Chernoff N, Rogers JM, Kavet R. A review of the literature on potential reproductive and developmental toxicity of electric and magnetic fields. *Toxicol*. 1992; 74:91–126.
- 15 Windham GC, Fenster L, Swan SH, Neutra RR. Use of video display terminals during pregnancy and the risk of spontaneous abortion, low birthweight, or intrauterine growth retardation. *Am J Indust Med*. 1990; 18:675–688.
- 16 Goldhaber MK, Polen MR, Hiatt RA. The risk of miscarriage and birth defects among women who use visual display terminals during pregnancy. *Am J Ind Med*. 1988; 13:695–706.
- 17 McDonald AD, McDonald JC, Armstrong B, Cherry N, Nolin AD, Robert D. Work with visual display units in pregnancy. *Br J Ind Med*. 1988; 45:509–515.
- 18 WHO Working Group. *Visual display terminals and workers' health*. Geneva: World Health Organization. offset publication no. 99. 1987.
- 19 Bryant HE, Love EJ. Video Display Terminal Use and Spontaneous Abortion Risk. *Int J Epidemiol*. 1989; 18(1):132–138.
- 20 Ong CN, Thein MM, Berquist U. A Review of Adverse Effects on reproduction amongst female computer terminal workers. *Ann Acad Med*. 1990; 19(5):649–655.
- 21 Ericson A, Kallen B. An epidemiological study of work with video screens and pregnancy outcome: II. A case-control study. *Am J Ind Med*. 1986; 9:459–475.
- 22 Westerholm P, Ericson A. Pregnancy outcome and VDU-work in a cohort of insurance clerks. In: Knave B, and Wideback, PG, eds. *Work with Display Units 86: Selected Papers from the International Scientific Conference on work with Display Units*. North-Holland: Elsevier. 1987:104–110.
- 23 Butler WJ, Brix KA. Video display terminal work and pregnancy outcome in Michigan clerical workers. In: Pearce BG, ed. *Allegations of reproductive hazards from VDUs*. Nottingham: Humane Technology. 1986:31–52.
- 24 Lee WR. Working with visual display units. *Brit Med J*. 1985; 291(6501):989–991.
- 25 Centers for Disease Control and Prevention, Family Planning Evaluation Division. *Cluster of Spontaneous Abortions*. Centers for Disease Control and Prevention. Report EPI-80-113-2. 1981.
- 26 Abenheim L, Lert F. Methodological Issues for the assessment of clusters of adverse pregnancy outcomes in the workplace: the case of video display terminal users. *J Occup Med*. 1991; 33(10):1091–1096.
- 27 Schnorr TM, Grajewski BA, Hornung RW, Thun MJ, Egeland GM, Murray WE, Conover DL, Halperin WE. Video display terminals and the risk of spontaneous abortion. *NEJM*. 1991; 324:727–733.
- 28 Oak Ridge Associated Universities. *Health effects of low frequency electric and magnetic fields*. Report ORAV 92/F9, prepared for the Committee on Interagency Radiation Research and Policy Coordination, Oak Ridge, TN. 1992.
- 29 COMAR. Biological and health effects of electric and magnetic fields from video display terminals. *IEEE Engineering in Medicine and Biology*. 1997 16(3):87–92.
- 30 Bagrit L. *The age of automation*. London: Weidenfeld and Nicholson, 1965:61.

20. Benzene in Perrier, 1990

Background

Benzene is a hydrocarbon essential to many industrial processes, particularly in the chemical, tire-manufacturing, and petroleum industries. During the 1970s epidemiological studies of rubber-company workers in Ohio who were regularly exposed to high levels of benzene (probably at levels of at least 100 parts per million for a number of years, and possibly higher at times) showed that they suffered fatal leukemia at a rate seven times that of nonexposed workers in the same plant.¹ Similar studies of shoemakers in Turkey and Italy showed that such high-level exposures can also cause chromosomal aberrations and aplastic anemia (failure of the bone marrow to produce new blood cells).²

As a result of the studies, the federal Occupational Safety and Health Administration (OSHA) set limits on occupational benzene exposure. The original limits were set in 1971 at 10 parts per million.¹ In 1980 OSHA's attempt to reduce this level to 1 part per million was invalidated by the Supreme Court on the grounds that OSHA had not presented any scientific evidence that this tighter standard would result in any improved benefits to workers' health.²

In 1989 a report in *Environmental Health Perspectives* concluded that people living near oil refineries and petrochemical installations had no higher exposure to benzene than the rest of the general public, and that "more than half the entire nationwide exposure to benzene results from smoking tobacco or being exposed to tobacco smoke."³ But the most unexpected report on the health risks of benzene came from a very unexpected "source."

The Scare

In Mecklenburg County, North Carolina, some laboratory workers thought of a way to save time when testing local water for contamination: Rather than make their own purified water to use as a control sample in the tests, they would purchase bottles of Perrier—the French mineral water, famed for its purity, that ever since the late 1970s had been seen both as a status symbol and as an emblem of healthfulness.⁴

In January 1990 a spectrometer the lab workers used to detect organic compounds began displaying some odd readings. After checking all their other equipment, the lab workers analyzed the Perrier—and, to their amazement, found traces of benzene.

The FDA was notified. Further tests of various Perrier shipments all found benzene contamination, at

levels ranging from 12.3 to 19.9 parts per billion—above the EPA standards (a maximum of 5 parts per billion) set for public drinking-water supplies.⁵

Perrier immediately announced a recall of its entire U.S. inventory of over 72 million bottles. The next day it halted production worldwide.⁶ The company chairman declared, "We don't want the least doubt, however small, to tarnish the product's image of quality and purity."⁷

The Reaction

The FDA emphasized that the risk was small: Lifetime consumption of 16 ounces of Perrier a day might increase your lifetime risk of cancer by one in one million.⁶ Few people panicked; yet, as one Washingtonian dryly commented, "an entire class of people have just had their weekends ruined." The irony of a product so associated with a healthy lifestyle posing such a risk was not lost on many: Then-Senator Al Gore remarked that he had been worried about fluoride in tap water and so had switched to Perrier. Now, however—he went on in apparent seriousness—"I am not going to be satisfied until thousands of rats have consumed millions of bottles of Perrier and survived."⁸

Conclusion

The irony only increased the following week when Perrier officials discovered the cause of the problem: It turned out that benzene is naturally present in the spring that is the source of Perrier. Workers at Perrier's spring at Vergeze, France, were supposed to change the filters at the spring every six weeks. They had failed to do so for four months and had thus allowed the contamination.⁹ In other words, this was an "all-natural" health scare.

The filters were changed, the spring was certified as pure, and Perrier soon returned to the shelves. Putting this scare into perspective, the 1986 Surgeon General's report stated that a pack of cigarettes had up to 2,000 times the level of benzene found in the tainted Perrier¹⁰—and cigarettes remain on grocery shelves to this day.

¹ Carter LJ. Dispute over cancer risk quantification. *Science*. March 30, 1979:1324.

² Smith RJ. A light rein falls on OSHA. *Science*. August 1, 1980:567.

³ Raloff J. Biggest benzene risks hide close to home. *Science News*. October 14, 1989:245.

⁴ Perrier woes began with blip on Carolina screen. *The New York Times*. February 12, 1990:A18.

- ⁵ James G. Perrier recalls its water in U.S. after benzene is found in bottles. *The New York Times*. February 10, 1990:1.
- ⁶ Meier B. Perrier production halted worldwide. *The New York Times*. February 11, 1990:26.
- ⁷ Miller A. Perrier loses its fizz. *Newsweek*. February 26, 1990:53.
- ⁸ Dowd M. What, no Perrier? Status bubble bursts. *The New York Times*. February 11, 1990:26.
- ⁹ Wickens B. Bursting the bubble. *McLean's*. February 26, 1990:34.
- ¹⁰ Kluger R. *Ashes to Ashes*. New York: Alfred A. Knopf; 1996:708.

21. Amalgam Dental Fillings, 1990

Background

Silver fillings—actually a compound consisting of a tightly bound mixture (called an amalgam) of silver, copper, tin, mercury, and zinc—came into common use in the 1830s. Silver amalgam became the most common filling material after G.V. Black, the father of American dentistry, published research in 1896 supporting its use for filling teeth. Today such fillings are preferred for their durability (most fillings will last at least 10 years; some last as long as 40) and for their low cost.^{1,2}

For many years it was believed that mercury, once bound in a filling, could not escape into a patient's mouth. In 1979, however, researchers using a newly created device to measure mercury vapor in the workplace discovered that small amounts of mercury could be released into the body through chewing. The exposure lasts for only a few seconds, and most of the mercury is exhaled.^{3,4}

A small number of dentists began making claims that patients had been “poisoned” by their mercury fillings and also began recommending the removal of those fillings. A Colorado dentist named Hal Huggins propelled the movement by promoting a theory that amalgam fillings caused a wide variety of diseases ranging from anxiety and acne to multiple sclerosis, premenstrual syndrome, and cancer. In 1985, Huggins published a book called *It's all in Your Head* in which he detailed the “harmful effects” of mercury fillings.⁵

Several animal studies conducted during the late 1980s also suggested possible ill effects from mercury amalgam as well as a relationship between fillings and

higher blood levels of mercury.^{6–8} These studies were more than countered, however, by numerous epidemiological studies indicating no evidence of mercury poisoning or other health problems from amalgam fillings.^{9–17}

The one health concern that has been noted in connection with amalgam fillings is allergic reactions to mercury, a condition affecting only a very small percentage of people. (Although at least 100 million Americans have had such fillings over the years, there have been fewer than 50 cases of amalgam allergy reported in the scientific literature in this century.³)

A statistical analysis of a questionnaire survey of 20,000 dental professionals—people who are most likely to encounter any adverse effects of mercury, and who do, indeed, have blood mercury levels nearly twice as high as those found in the general population—found no toxic or other ill effects.¹⁸

But these reassuring findings did not deter some dentists from urging their patients to have their fillings removed—at a cost of between \$65 to \$500 per filling, and often with disastrous results. A \$100,000 settlement was awarded to a California woman in 1985 after her dentist removed her silver fillings and caused severe nerve damage.

Hal Huggins, the leader of the anti-amalgam movement, was censured by the FDA in 1985 for using a galvanometer-type device, allegedly to measure the amount of mercury in the body. The FDA said that “there is no scientific basis for the removal of dental amalgams for the purpose of replacing them with other materials.”³ Huggins later lost a \$159,000 malpractice suit to a patient who lost several teeth under his treatment. He subsequently had his license revoked.^{19,20}

Because of episodes such as these, the American Dental Association has warned dentists that the removal of amalgams “for the alleged purpose of removing toxic substances” is considered unethical behavior.²¹

The Scare

In December 1990 *60 Minutes* aired a report entitled “Poisons in Your Mouth?” that suggested that amalgam fillings were a health hazard. Among the more outlandish claims in the report was a testimonial from a young woman who had suffered from multiple sclerosis, and who declared that by the night of the day she had her fillings removed—a procedure that actually increases the body's mercury level, albeit temporarily—she was able to throw away her cane and go dancing.¹⁹

The Reaction

In response to the many dental professionals and

consumer advocates who condemned the program as irresponsible, CBS's director of audience services replied that they had attempted "to ensure that our report was balanced in presenting arguments on both sides of the issue." This was little solace to some of the many people who subsequently rushed to have their fillings removed, including one woman with amyotrophic lateral sclerosis ("Lou Gehrig's disease") who endured \$10,000 in expenses and 18 hours of excruciatingly painful dental work—only to see no improvement in her condition.²²

A survey conducted by the ADA several months after the *60 Minutes* broadcast showed that 4 percent of adults had had their fillings removed (not all since the broadcast), and another 16 percent were considering such an action. The ADA reiterated their strong position in opposition to such measures. The National Multiple Sclerosis Society also disavowed the *60 Minutes* report, declaring that "there is absolutely no evidence that mercury amalgam fillings have any connection to MS, or that their replacement will help patients with the disease."¹⁹

Lawsuits have also been filed against the manufacturers of dental amalgam; a recent case alleged that amalgam fillings had caused Guillain-Barre syndrome, a neurologic problem due to damaged nerves. The judge in that case concluded, however, that ". . . there is no support within the scientific community for the hypothesis that mercury vapor released in any amounts from dental amalgam can cause Guillain-Barre syndrome or, for that matter, any of the illnesses or diseases or conditions that the plaintiff might allege were contained within the general description of injury in his complaint."²³

In 1993 a comprehensive inquiry by the U.S. Public Health Service concluded that any mercury released from amalgams does not contribute to disease, immune-system disorders, or birth defects; and that allergies from such fillings are extremely rare. (Furthermore, when such allergies do exist, they are so acute that they will be felt within days—even within hours—of receiving the filling.¹⁸) Thus, "available data do not justify discontinuing the use of silver-containing dental amalgam fillings or recommending their replacement."¹⁹

The World Health Organization,²⁴ the FDI World Dental Federation, and the ADA all support the continued use of dental amalgam as a safe, durable, and cost-effective restorative material. According to the ADA, there is no credible scientific evidence that exposure to mercury from dental amalgam poses a serious health risk to humans.²⁵

Conclusion

It must be remembered that while high levels of mercury are toxic, the element is widespread in the natural environment. Mercury occurs naturally in almost all rocks, plants, drinking water, and food—even in our own bodies—at levels that do not pose a health risk. The minuscule amounts that amalgam fillings add to the body's overall mercury "load" are inconsequential; the Public Health Service study showed that even among people with more than 30 amalgams in their mouth, the highest level of mercury found in the urine was 4.8 micrograms per liter. (The urine level at which adverse health effects might occur is at least 25 micrograms per liter).¹⁹

While research continues on new dental technologies, all of the existing alternatives to amalgam fillings have drawbacks. Gold is far more expensive and too soft. Porcelain, while used to fill front teeth, is too fragile to be used in back teeth. The various "tooth-colored" plastics and resins, while praised for their cosmetic appearance, lack durability—most need to be replaced after two years—and may trigger allergies of their own.^{26,27}

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- ¹ DeMaar FE. Historically, when and by whom was silver amalgam introduced? Part II. *International College of Dentists Scientific and Educational Bulletin*. 1972; 6:59–64.
 - ² Black GV. The physical properties of the silver-tin amalgams. *Dental Cosmos*. 1896; 38:965–992.
 - ³ Dodes JE. *Dubious Dental Care*. New York: American Council on Science and Health. 1991:6.
 - ⁴ American Dental Association. When your patients ask about mercury in amalgam. *JADA*. 1990;120:395–398.
 - ⁵ Huggins HA, Huggins SA. *It's All in Your Head*. Colorado Springs: Huggins. 1985.
 - ⁶ Danscher G, Horsted-Bindslev P, Rungby J. Traces of mercury in organs from primates with amalgam fillings. *Exp Mol Pathol*. 1990; 52:291–299.
 - ⁷ Han LJ, Kloiber R, Leininger RW, Vimy MJ, Lorscheider FL. Whole-body imaging of the distribution of mercury released from dental fillings into monkey tissues. *FASEB J*. 1990; 4:3256–3260.
 - ⁸ Boyd ND, Benediktsson H, Vimy MJ, Hooper DE, Lorscheider FL. Mercury from dental "silver" tooth fillings impairs sheep kidney function. *Am J Physiol*. 1991; 261:R1010–R1014.
 - ⁹ Ahlqwist M, Bengtsson C, Furunes B, Hollender L, Lapidus L. Number of amalgam tooth fillings in relation to subjective-

- ly experienced symptoms in a study of Swedish women. *Community Dent Oral Epidemiol.* 1988; 16:227–331.
- 10 Ahlquist M, Bengtsson C, Lapidus L. Number of amalgam fillings in relation to cardiovascular disease, diabetes, cancer and early death in Swedish women. *Community Dent Oral Epidemiol.* 1993; 21:40–44.
 - 11 Eley BM, Cox SW. The release, absorption and possible health effects of mercury from dental amalgam: a review of recent findings. *Br Dental J.* 1993; 175:161–168.
 - 12 Herrström P, Högstedt B. Allergic diseases, dental health, and socioeconomic situation of Swedish teenagers. Allergy dental health, and social situation. *Scand J Prim Health Care.* 1994; 12:57–61.
 - 13 Österblad M, Leistevuo J, Leistevuo T, et al. Antimicrobial and mercury resistance in aerobic gram-negative bacilli in fecal flora among persons with and without dental amalgam fillings. *Antimicrob Agents Chemother.* 1995; 39:2499–2502.
 - 14 Berglund A, Molin M. Mercury vapor release from dental amalgam in patients with symptoms allegedly caused by amalgam fillings. *Eur J Oral Sci.* 1996; 104:56–63.
 - 15 Björkman L, Pedersen NL, Lichtenstein P. Physical and mental health related to dental amalgam fillings in Swedish twins. *Community Dent Oral Epidemiol.* 1996; 24:260–267.
 - 16 Edlund C, Björkman L, Ekstrand J, Sandborgh-Englund G, Nord CE. Resistance of the normal human microflora to mercury and antimicrobials after exposure to mercury from dental amalgam fillings. *Clin Infect Dis.* 1996; 22:944–950.
 - 17 Sandborgh-Englund G, Nygren AT, Ekstrand J, Elinder CG. No evidence of renal toxicity from amalgam fillings. *Am J Physiol.* 1996; 271:R941–R945.
 - 18 Nanadakavuakren A. *Our Global Environment: A Health Perspective.* Prospect Heights, IL: Waveland Press. 1995.
 - 19 *Dental Mercury Fillings.* Loma Linda, CA: National Council Against Health Fraud, 1996.
 - 20 Public Health Service Subcommittee on Risk Management. *Dental amalgam: a scientific review and recommended public health service strategy for research, education and regulation.* Department HHS/PHS. January 1993.
 - 21 Begley S. Drilling for danger? *Newsweek.* October 15, 1990:80.
 - 22 Barrett S, Jarvis WT. *The Health Robbers: A Close Look at Quackery in America.* Buffalo, NY: Prometheus Books; 1993:451–452.
 - 23 Tollhurst v Johnson & Johnson Consumer Products, Inc., Case No. CV 718228 (Superior Court of the State of California, in and for the County of Santa Clara) Memorandum of Decision and Order After Hearing Defendants' Motion in Line to Exclude Testimony of Plaintiff's Expert Regarding Causation and Related Matters. October 1, 1996.
 - 24 World Health Organization. *WHO Consensus Statement on Dental Amalgam.* Geneva: WHO. September 1997.
 - 25 No international bans on dental amalgams. *Asbury Park Press.* March 18, 1997:B3.
 - 26 *How safe are your dental fillings? Glamour.* May 1987:330–331.
 - 27 Barrett S, Jarvis WT, Kroger M, London WM. *Consumer Health: A Guide to Intelligent Decisions.* 6th ed. Madison, WI: Brown and Benchmark Publishers. 1997.

22. Asbestos in Schools, 1993

Background

From 1940 to 1973 schools across the United States were required to have asbestos insulation as a fire safety measure.¹ Asbestos was also widely used in many schools in tiles and plaster.² The EPA banned the use of asbestos in schools in 1973; by the late 1970s the agency started formulating regulations in an attempt to reduce the exposure of schoolchildren to the substance.³

In 1982 the EPA required that all schools be inspected for friable (easily crumbled) asbestos. In 1984 Congress passed the Asbestos School Hazard Abatement Act to provide financial assistance to schools with serious asbestos hazards. And in 1986 Congress passed the Asbestos Hazard Emergency Response Act (AHERA), which required all private and public schools to inspect for asbestos, develop asbestos management plans, and implement appropriate actions.⁴ By 1990 it was estimated that the cost of such abatement work was over \$6 billion.⁵

The public health benefit of all this was unclear, as asbestos experts have estimated that the lifetime risk to schoolchildren exposed to 0.001 fibers of chrysotile asbestos per milliliter of air for a minimum of 10 years is one additional death in 100,000 (that's three times less than the risk of being struck by lightning)—and most schools had asbestos levels far lower than 0.001 fibers per milliliter.⁶

The Scare

An EPA report showed that in buildings where asbestos had been removed, postremoval asbestos levels were often significantly higher than preremoval levels. Poorly conducted asbestos removals thus may actually

increase health risks by releasing more particles into the air.⁷ Additionally, while EPA guidelines do not require removal of asbestos if the asbestos-containing materials are not significantly damaged, many school districts misunderstood the guidelines, taking them as a mandate to remove all asbestos. Such misunderstanding led to unnecessary expense and to the diversion of already scarce school funds from other needs.⁸

In August 1993 it was revealed that an independent contractor to whom the New York City Board of Education had paid \$2 million to inspect city schools for asbestos had failed to perform the inspection properly. (The contractor did, of course, pocket the city's money.) To avoid being held in contempt of the 1986 AHERA law, New York Mayor David Dinkins announced that no school would be allowed to open in September until it had been inspected and found safe by both the Department of Environmental Protection and the Health Department. As a result, the opening of school for nearly 1,000,000 New York City schoolchildren was delayed by two weeks, with a few schools remaining shuttered even longer, their students placed in other buildings.⁹

The Reaction

Most schoolchildren were probably grateful for the respite; most scientists were less so. The American Medical Association had already declared that the type of low-level exposure found in school buildings—averaging 0.0005 fibers per milliliter (probably no more than the level in ambient air in Manhattan)—did not pose a health hazard.² This was especially true because the type of asbestos used in schools—chrysotile, or “white” asbestos—is considered far less hazardous than other types of asbestos—specifically, crocidolite and amosite see also “Asbestos in Hair Dryers, 1979” page 27).¹⁰ Chrysotile makes up 95 percent of all the asbestos ever used in the United States; it is easily expelled by the lungs rather than attaching itself to lung tissue.^{9,11}

Conclusion

The 1993 New York City asbestos scare may actually have done some good, in the sense that it alerted many people in the general community to the hazards of hyperbolizing about environmental risks. Many parents asked whether such a hypothetical risk was worth disrupting their and their children's lives for. Today the asbestos abatement laws—and the \$4 billion to \$6 billion-a-year abatement industry—remain in place; but more people are now aware that asbestos, as chemist P. J. Wingate puts it, is “like a big sleeping dog. If not stirred up, it does no harm. If hampered or sawed on, it may bite anyone near it.”¹

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- 1 Campbell S. Asbestos in schools: how much hazard? *ACSH News and Views*. September/October 1985.
 - 2 Whelan E. Asbestos in schools: the latest phantom risk. *Priorities*. Fall/Winter 1993:38.
 - 3 Environmental Defense Fund. The campaign to end use of deadly asbestos. *EDF Letter*. Vol XIX, No. 3, June 1988:2.
 - 4 U.S. Congress Asbestos Hazard Emergency Response Act (AHERA), 1986. Publication L. No. 99-519, 100 Stat 2970. Section 2 of AHERA addenda New Title II, 88201-214 to the Toxic Substances Control Act codified at 15 U.S.C. 992641-54 (supplement IV, 1986).
 - 5 Whelan E. *Toxic Terror*. 2nd ed. Buffalo, NY: Prometheus Books. 1993:267–268.
 - 6 Corn M, et al. Airborne concentrations of asbestos in 71 school buildings. *Regul Toxicol Pharmacol*. 1991; 13:99–114.
 - 7 *Toxic Terror*:270–272.
 - 8 EPA tells schools asbestos can stay, but it's too late. *Detroit Free Press*. September 14, 1990:1.
 - 9 Byron C. The phony asbestos scare. *New York*. September 15, 1993:22–23.
 - 10 McDonal JC, McDonald AD. Chrysotile, tremolite and carcinogenicity. *Ann Occup Hyg*. 1997; 41:699–705.
 - 11 National Cancer Institute. *Cancer Facts: Questions and Answers About Asbestos Exposure*. November 30, 1995.

23. Cellular Phones, 1993

Background

Since a 1979 report suggested that electromagnetic fields (EMFs) from power lines might increase the risk of childhood cancer,¹ sporadic scares have developed over the health effects of a wide variety of electrical appliances—devices ranging from electric blankets to computer terminals and from electric razors to alarm clocks (see “Electric Blankets, 1989,” page 35, and “Video Display Terminals, 1989,” page 37).

Subsequent studies have revealed methodological errors in the 1979 report: It failed to account for other carcinogenic factors, and studies of occupational exposure among electrical workers and others exposed to high levels of EMFs have given conflicting data.^{2,3} Scientists have also pointed out that such electromagnetic fields are far too weak to affect human tissue by

any of the known mechanisms by which the far stronger X rays and ultraviolet radiation can break apart cellular components and cause cancer.⁴ But the most publicized health scare involving an electromagnetic field was based on no scientific data at all.

The Scare

David Reynard of Tampa, Florida, gave his wife a cellular phone when she became pregnant in 1988. Two years later she was diagnosed with a brain tumor. The tumor was located just behind her right ear, where she typically had placed the phone's antenna.⁵ After his wife's death, Reynard filed suit against the phone's manufacturer, alleging that electromagnetic energy from the phone's antenna had caused the cancer. On January 21, 1993, Reynard appeared on CNN's *Larry King Show* to air his claims; in the weeks that followed, three similar lawsuits were filed by cellular phone users who had developed brain tumors in similar locations.¹

The Reaction

These anecdotal charges were, as one report noted, "not the kind of evidence that would be accepted by the *New England Journal of Medicine*"; but, for a time, it didn't matter: The authoritative voice of Larry King intoning, "cellular phones can kill you" struck a nerve.

One national journalist who had "become addicted to her cellular phone" described it as "yet another technology that is out to get us."⁴ Sales of the phones, which had been growing at rate of from 20 to 70 percent a year since 1982, fell off sharply. Stock prices of the three largest cellular companies dropped about 10 percent during the week following the King broadcast.⁶ Even more than Alar, the scare quickly entered the national consciousness, perhaps because of the image of the cellular phone as a "yuppie accessory." One entrepreneur offered a device to shield users' heads from the supposedly deadly antennae.⁷

Congressional hearings were held. The FDA, the National Cancer Institute, and the EPA all declared that there was no reason to put your phone on hold,² although an FDA spokesperson did suggest that if consumers were concerned, "they should pay attention to their usage."⁸ It was also noted that while the number of brain cancers had increased slightly between 1973 and 1989—from five per 100,000 to six per 100,000—there had been no change in the pattern of location of the tumors—as might be expected if the phones were, indeed, the culprit.⁵

At present it is not possible to tell whether an individual's cancer arose because of cellular phone use,

or whether it would have happened anyway. At the current brain cancer incidence rate, about 180 cases would be expected among the approximately 3 million owners of hand-held cellular phones—whether or not they actually used their phones.⁹

Conclusion

Cellular trade associations promised to spend \$25 million for research into cellular safety. An independent research entity was organized to implement a research program, including laboratory and epidemiological studies.¹⁰

Within months, however, the scare was largely forgotten. By mid 1993 sales of cellular phones were up 30 percent over the previous year, stock prices had recovered, and consumers had apparently lost concern. When one phone company offered its customers a free phone if they signed up for cellular service and offered a choice of three models, most customers chose the pocket model—with the antenna next to the user's head—over two other models with different configurations.⁷

Very few studies on electromagnetic fields are relevant to the evaluation of exposure to radio frequency (the frequency used by cellular phones) and the development of cancer in humans. Cancer studies in animals provide no clear evidence of an increase in tumor incidence.¹¹ In fact, studies to date show no evidence of serious medical effects from routine use of cellular phones.¹²

But while no study has proved that the electromagnetic fields from phones or other devices are harmful, it remains impossible to prove that EMFs are not harmful. Additionally, difficulties in designing, implementing, and interpreting epidemiological studies—particularly with respect to identifying populations with substantial exposures—are frustrating efforts to evaluate the effects of EMFs.¹¹

The brief life of the cellular phone scare may be a sign that consumers are beginning to show a more measured reaction to reports of this type. At the present time there is no convincing evidence that EMFs from cellular phones are harmful.⁹ Indeed, as suggested by a recent study reported in the *New England Journal of Medicine*, if there is any significant danger from cellular phones, it is from trying to drive while talking on one: The study showed that drivers who use cellular phones have four times the risk of accidents as drivers who do not.¹³

¹ Wertheimer N, Leeper E. Electrical wiring configurations and childhood cancer. *Am J Epidemiol.* 1979; 109:273–284.

- 2 Conkling W. Shocking charges. *American Health*. May 1993:50–55.
- 3 Savitz DA, Pearce NE, Poole C. Methodological issues in the epidemiology of electromagnetic fields and cancer. *Epidemiol Rev*. 1989; 11:59–78.
- 4 Elmer-Dewitt P. Dialing ‘P’ for panic. *Time* February 8, 1993:56.
- 5 Call at your own risk. *20/20* [television program]. ABC News, Transcript #1305, January 29, 1993.
- 6 Moore M. Jury still out on safety of cellular technology. *PC Week*. February 15, 1993.
- 7 Ziegler B. Remember the cellular-phone scare? *Business Week*. July 19, 1993:83.
- 8 The cellular phone controversy. *CNN Moneyline*. [television program]. Transcript #828, February 2, 1993.
- 9 Center for Devices and Radiological Health, Food and Drug Administration. Center addresses cellular phone issues. *Radiological Health Bulletin*. Spring 1993; 227:2–4.
- 10 Graham JD, Putnam S. Cellular telephones and brain cancer. *Risk in Perspective*. Harvard Center for Risk Analysis. June 1995; 3:1–2.
- 11 International Commission on Non-Ionizing Radiation Protection (ICNIRP). Health issues related to the use of hand-held radiotelephone and base transmitters. *Health Physics*. 1996; 70:587–593.
- 12 Salvatore JR. Low-frequency magnetic fields and cancer. *Postgrad Med*. 1996; 100:183–190.
- 13 Redelmeier DA, Tibshirani RJ. Association between cellular-telephone calls and motor vehicle collisions. *N Engl J Med*. 1997; 336:453–458.

24. Perchloroethylene in a Harlem School, 1997

Background

Perchloroethylene—also known as tetrachloroethylene and commonly referred to as “perc”—is the primary dry-cleaning solvent in use today. First used in the 1930s, perc is now used, alone or in combination with other solvents, by almost 90 percent of dry cleaners in the United States.

Perc is less toxic and less flammable than many of the dry-cleaning fluid alternatives, and it can be reclaimed for reuse more efficiently.¹

But while perc has been a boon to the dry-cleaning industry, environmental activists, regulatory officials, and pro-regulation public-interest organizations are less pleased with the prevalence of its use. *Consumer Reports* claims, “You’re likely to be exposed to some level of perc simply by wearing recently dry-cleaned clothes or storing them in your house.”² An opinion piece in *The New York Times* labeled perc “highly toxic” and called on the city government to “remove all the city’s cleaners from apartment buildings.”³

Certainly, chemicals such as perc can pose risks at high exposure levels and when improperly handled. Prolonged exposure to 200 parts per million of perc can induce headaches, dizziness, nausea, and eye and skin irritation.¹ (One part per million is equivalent to one facial tissue in a stack of tissues higher than the Empire State Building.) Perc, like most chemical substances, is safe if it is handled properly and exposures are limited; but it may be dangerous if it is used carelessly.

Although no scientific consensus has been reached, the Environmental Protection Agency (EPA) classifies perc as an animal carcinogen and places it on the continuum between possible and probable human carcinogens. Initial claims that perc might be a human carcinogen were based on animal tests. A 1977 National Cancer Institute study indicated that perc could induce liver cancer in mice but not in rats.⁴ A 1985 National Toxicology Program study done on rats and mice of both sexes also concluded that there was “clear evidence” of the rodent carcinogenicity of perc.⁵ But while positive results in animal tests can provide some indication of whether a compound is potentially carcinogenic to humans, such tests can be inconclusive.

If low- to-moderate exposure to perc presents a cancer threat to humans, one would expect to see increased rates of cancers among dry-cleaning workers—people who are exposed daily to significant amounts of perc in their working environment. Some studies have found a slight increase in cancer mortality rates for laundry and dry-cleaning workers.^{6–10} Other studies have suggested that this increase could result from other environmental and behavioral factors such as a smoking habit, socioeconomic status, and alcohol use.¹⁰ In studies in which it was possible to subdivide workers by exposure to different solvents, the slight increase in cancer deaths was not observable in those subgroups exposed only to perc. In short, if dry-cleaning workers are at risk from perc, scientific studies have yet to bear that out. The EPA’s Science Advisory Board has stated that perc “is an example of a chemical for which there is no compelling evidence of human cancer risk.”¹¹

The EPA has set its recommended exposure limit for a 40-hour work week at 25 parts per million (ppm) of perc. The New York State Department of Health (NYSDOH) recommends that the average air level of perc in a residential community not exceed 15 parts per billion (ppb)—a minuscule level at which no health effects of any kind have been noted (one part per billion is equivalent to one sheet of toilet paper in a roll stretching from New York to London).

The NYSDOH doesn't recommend taking action to reduce perc exposure until the air level is 150 ppb or higher. The NYSDOH goes as far as to state, "The guideline of 15 ppb is not a line between air levels that cause health effects and those that do not. . . . Thus, the possibility of health effects is low even at air levels slightly above the guideline. In addition, the guideline is based on the assumption that people are continuously exposed to perc in air all day, every day, for as long as a lifetime."¹²

The Scare

During June and July of 1997 the air in New York City's P.S. 141—an elementary school housed in a building that once had held a dry-cleaning facility—was tested for levels of perc. In the days of the building's use as a cleaning plant, the dry-cleaning machinery had been positioned at the back of the sprawling building; the present-day school was located at the front.

The landlord's consultant (a company called AKRF Incorporated) and the School Construction Authority tested the air in selected rooms at the school and found the perc levels in all to be at or below the NYSDOH's 15-ppb guideline. Based on those tests, on August 19 the New York State Department of Health cleared P.S. 141 for opening.

Then, on September 13, more than a week after the New York City school year had begun, the air in P.S. 141 was tested again. This time six rooms were tested—and perc levels in four of the rooms were found to be above the 15-ppb guideline. The levels found in three of the rooms were 16.1 ppb, 16.8 ppb, and 19 ppb; disparate readings of 16.1 and 36 ppb were recorded for the fourth room.

The Reaction

On October 6, 1997, under pressure from parents and advocacy groups, the school board shut down P.S. 141. Trembling parents were interviewed on local news stations, and *The New York Times* ran an article subtitled "A Toxic Lease." The Times piece stated that "chemical fumes were at potentially dangerous levels" at the Harlem school.¹³

Conclusion

Twenty-one readings in all were taken at P.S. 141 between June 30 and September 20. The readings were taken in a classroom, in the cafeteria, and in various other rooms. Only five of the readings recorded perc levels above 15 ppb.¹⁴

According to Andrew Rudko, a vice president of AKRF Incorporated (the company that had conducted the tests for the landlord), the fluctuation in the readings at the school was not surprising, given the nature of the tests and the small amounts being measured. "The precision," said Rudko of such air tests, "is not that great."¹⁵ At the levels being measured, the perc readings could spike up if one or more persons standing near the sampled air happened to be wearing clothes fresh from the dry cleaner.

As of this writing, in the spring of 1998, P.S. 141—a building renovated at a cost of \$5 million to house 500 students in a school district plagued by overcrowding—remains closed. The P.S. 141 students, from kindergarten through fourth grade, have been reassigned to more than a dozen other schools in the district, all of them already overcrowded.

The decision to shut down P.S. 141 was based on potentially meaningless fluctuations in readings of minuscule levels of perc—levels at which no adverse health effects have ever been documented. And, although the school was closed for the benefit of the children's health, the only effects the decision to close will have are in fact detrimental to the children. There is a direct detrimental effect in that the students of P.S. 141 have been scattered to other, overcrowded schools and an indirect detrimental effect in that the taxpayer dollars wasted on this scare might otherwise have been spent on such things as school libraries.

¹ International Fabricare Institute. *The Facts on Perc*. Silver Spring, MD: International Fabricare Institute; November 1992.

² This "perc" isn't a benefit. *Consumer Reports*. September 1992:559.

³ Pfeifer MB. The Enemy Below. *The New York Times*. September 5, 1992:19.

⁴ National Cancer Institute. *Bioassay of Tetrachloroethylene for Possible Carcinogenicity*. Carcinogenesis Technical Report series, no. 13. Washington, DC: National Institutes of Health; 1977.

⁵ *Federal Register*. (2686) January 19, 1989.

- 6 Brown DP, Kaplan SD. Retrospective cohort mortality study of dry cleaner workers using perchloroethylene. *J Occupational Med.* 1987; 29(6):535.
- 7 Blair, et al. Causes of death among laundry and dry cleaning workers. *Am J Publ Health.* 1968; 69:508–511.
- 8 Katz, et al. Female laundry and dry cleaning workers in Wisconsin: A mortality analysis. *Am J Publ Health.* 1981; 71:305–307.
- 9 Duh, et al. Mortality among laundry and dry cleaning workers in Oklahoma. *Am J Publ Health.* 1984; 74:1278–1280.
- 10 Blair, et al. Cancer and other causes of death among a cohort of dry cleaners. *British J Indust Med.* 1990; 47:162–168.
- 11 Letter to EPA Administrator William K. Reilly from Dr. Raymond Loehr, chairman, EPA Science Advisory Board, and Dr. Bernard Weiss, acting chairman, Environmental Health Committee, August 16, 1991.
- 12 *Fact Sheet: Tetrachloroethylene (perc) in Indoor and Outdoor Air.* New York State Department of Health, October 1997.
- 13 Steinberg J. How a laundry in Harlem became a tainted school. *The New York Times.* October 11, 1997.
- 14 *The New York Times.* October 13, 1997.
- 15 Steinberg J. As Parents Scramble, Questions Swirl over a Closed School. *The New York Times.* October 8, 1997.

25. Not-Quite-Great Unfounded Health Scares

Introduction

As we worked to prepare the third edition of *Facts Versus Fears*, we asked all of ACSH's 250 scientific and policy advisors to nominate other scares to augment our original list of 20. The nominations were many and varied, but we had to rule out many of them because they did not completely adhere to our definition of a great unfounded health scare: a scare that received "great public attention in its day and followed its own course to closure in terms of public and regulatory response."

But even though these ruled-out scares did not produce the degree of furor or media hysteria characteristic of the "great unfounded scares," they still demand our consideration. These "not-quite-great" scares are important because they contribute to our understanding of why health scares, whether great or small, arise at all. In essence, these are not "great health scares" because

they did not build to a high pitch of hysteria—as happened, for example, in the Alar scare. Instead, these scares fade in and out of public consciousness, advancing and receding with little fanfare or rumbling along at a constant, mostly low, level, always viewed with discomfort and suspicion by segments of the public but seldom erupting into front-page news.

The not-quite-great scares we will briefly discuss in this section include community water fluoridation, food irradiation, and the use of bovine somatotropin (bST) in milk production.

Community Water Fluoridation

In 1945 Grand Rapids, Michigan, became the first city in the United States to fluoridate its public water supply. It had been discovered that fluoride—a mineral that occurs naturally in almost all foods and water supplies—helped teeth to resist decay and fostered repair of the early stages of tooth decay, before it becomes visible.

But even at this early date, several small but highly vocal national groups sprang up whose sole purpose was fighting fluoridation. Antifluoridationists claim that fluoridation is unsafe, ineffective, and/or costly. They assert that exposure to fluoridated water increases the public's risk of contracting AIDS, cancer, Down's syndrome, heart disease, kidney disease, osteoporosis, and many other health problems. Over the years the antifluoridationists' tactics have included attracting the media, holding demonstrations at the local government level, lobbying public health agencies and lobbying the United States Congress.

The amount of fluoride added to a municipal water supply is minuscule; fluoride levels in the United States are adjusted to about one part of fluoride per million parts of water. The overwhelming weight of scientific evidence in hundreds of peer-reviewed studies confirms fluoridation's safety and effectiveness.¹

In 1966 Dr. Luther L. Terry, then Surgeon General of the United States, described fluoridation as "one of the four great, mass preventive health measures of all times,"² the other three being the purification of water, milk pasteurization, and the development of vaccines for immunization against disease.

Today, despite these findings, and despite fluoridation's long history of safety, antifluoridationists continue to provoke the public's concern. Their efforts have diminished significantly in recent years, however. Effective antifluoridation lobbying is virtually nonexistent at either the state or the federal level, and most fluoridation initiatives are successful: In 1995, for example, despite antifluoridationist's efforts, a law mandat-

ing statewide fluoridation was passed in California.

The controversy over community fluoridation can be summed up by a 1978 quote from *Consumer Reports*: “The simple truth is that there’s no ‘scientific controversy’ over the safety of fluoridation. The practice is safe, economical, and beneficial. The survival of this fake controversy represents, in our opinion, one of the major triumphs of quackery over science in our generation.”

Antifluoridation efforts have not been stamped out completely, but higher education levels among voters and over 50 years of positive consumer experiences with fluoridated water (and fluoridated toothpaste) should continue to help deflate this scare.

Food Irradiation

The United States Food and Drug Administration (FDA) first approved food irradiation in 1963 for use in controlling insects in wheat and flour. In 1983 the FDA extended its approval to the use of irradiation to control insects and microorganisms in spices, herbs, and plant-derived dehydrated foods. The process was further approved for use to control the parasite *Trichinella* in pork, in 1985; to prevent post-harvest sprouting by stored potatoes and to control insects and maturation in fruits and vegetables, in 1986; and to destroy *Salmonella* bacteria in poultry, in 1992. Finally, in 1997, the FDA approved using irradiation to control toxic *E. coli* bacteria and other infectious agents in beef, veal, and other red meats.

Food irradiation—a process by which foods are treated with ionizing radiation—can improve both the variety and the safety of the foods we eat and can help reduce the incidence of foodborne illnesses. Additionally, irradiation can be used at doses lower than those used to sterilize foods to pasteurize food products. Irradiation can delay the spoilage of highly perishable fresh fish and shellfish, destroy or greatly reduce the number of microorganisms in spices, destroy or greatly reduce the number of disease-causing bacteria and parasites in meats and poultry, prevent sprouting in potatoes and onions, and extend the shelf life of fruits such as strawberries and mangoes. The irradiation of foods at pasteurization doses has little or no effect on flavor.

More than 50 years of scientific research have established that the irradiation of foods to minimize foodborne illness and decrease waste is both safe and effective. Forty countries around the world have already approved some applications of irradiation, and irradiated foods are now marketed in 27 countries. In Japan 15,000 to 20,000 tons of potatoes are irradiated each year to prevent spoilage due to sprouting. Worldwide,

about 20,000 tons of irradiated spices and dry vegetable seasonings were used in 1992.

Both the World Health Organization³ and the Food and Agriculture Organization of the United Nations⁴ have approved the irradiation of many foods. In the United States the American Medical Association,⁵ the American Dietetic Association,⁶ and the Institute of Food Technologists⁷ have all endorsed the use of irradiation to supplement other methods of safeguarding the American food supply.

Despite this widespread approval of irradiation by governmental and health agencies, the FDA’s gradual approval of irradiation was won only after a series of hard-fought battles against antinuclear activists. One of irradiation’s most outspoken opponents has been Michael Jacobson, executive director of the Center for Science in the Public Interest (CSPI). Jacobson has declared, “While irradiation does kill bacteria, it involves the use of inherently dangerous materials and poses its own risks to workers, the environment and consumers.”

Contrary to such claims, however, properly irradiated foods are neither radioactive nor toxic. No by-products unique to the irradiation process have been identified in foods irradiated under FDA-approved conditions. The by-products produced by irradiation are the same as those found in foods processed by other means, such as canning and cooking. The safety of irradiation has been studied more extensively than that of any other food-preservation process.⁸

Furthermore, the process of irradiation does not pose a risk to workers in irradiation plants or to residents in the communities in which irradiation plants are located. For more than 30 years medical supplies and drugs have been sterilized by irradiation in some 40 to 50 radiation plants around the United States. This sterilization program has been most successful, and there have been no accidents due to the radiation process.

Despite vigorous resistance from anti-irradiation activist groups, the last hurdle to irradiation’s acceptance was cleared in 1997 when the FDA extended approval for its use on red meat. The activists may have alarmed consumers in the past, but the growing acceptance of irradiation by so many health and governmental agencies has served to reassure today’s public. With this last barrier behind us, it is now highly likely that the clamor against the irradiation of foods will gradually dissipate.

The Use of Bovine Somatotropin (bST) in Milk Production

Bovine somatotropin (bST) is a natural hormone

that stimulates milk production. Biotechnology companies began manufacturing a genetically engineered version of bST in the early 1990s.⁹

On November 5, 1993, the FDA approved genetically engineered bST for commercial use in the United States. Treating dairy cows with this hormone increases milk production by as much as 20 percent, and no detectable difference has been found between milk from treated cows and milk from untreated cows. The hormone bST has no adverse effects on the health of treated cows, and milk and meat from bST-treated cows are both safe for human consumption.

Scientists throughout the world—researchers working in academia, in government, and in the dairy industry—conducted more than 2,000 scientific studies of bST. The studies show clearly the efficacy, the safety, and the benefits that can be realized by integrating bST into dairy production technology. To stem the tide of misinformation about bST, the FDA itself—in an unprecedented move—sponsored a 1990 article in *Science* magazine stating that bST was perfectly safe.¹⁰

But despite the scientific data and the proved efficacy of bST, opposition arose. One day before U.S. sales of milk from treated cows began, consumer activists dressed up in cow suits and dumped milk to protest the use of bST. Jeremy Rifkin, the president of the Foundation for Economic Trends, raised particularly vigorous objections to the introduction of bST. Because Rifkin could not present a convincing case to the FDA, the EPA, or other scientific groups, he decided to take his case directly to the people. Rifkin and others used the popular press to make unsubstantiated claims that the use of bST would increase the incidence of antibiotic-resistant infections and increase milk drinkers' risk of developing allergies. Neither of these claims is true, however.

Like all other plant and animal proteins in the human diet, bST is destroyed during the digestion process. It therefore has no effect on people who consume it. Furthermore, bST is inactive in humans even when injected: The makeup of bovine somatotropin is significantly different from that of human somatotropin, and human cells can neither identify nor react to the bovine hormone.¹¹

Recently, the activists' attention has turned to Insulin-like Growth Factor (IGF-I), a protein hormone. This hormone, which is stimulated by naturally occurring bST, converts nutrients into milk. Both humans and cows possess IGF-I. Although supplemental bST does increase IGF-I levels in the milk of treated cows, treating cows with the hormone increases the level of IGF-I in their milk to only two to five parts per billion more

than the levels that occur naturally in untreated cows.

The fear of increased IGF-I levels in milk has, indeed, led to a scare, because IGF-I, estrogen, and organochlorines in milk have all been linked to breast cancer.¹² The FDA has dismissed this scare, however, and has concluded that the claim that IGF-I milk promotes breast cancer is scientifically unfounded.¹⁰

Despite the body of scientific evidence and bST's approval by the FDA, scares centering on the hormone's use in milk production are likely to continue because of the public's apprehension about the use of biotechnology to enhance the food supply. This continuing uneasiness is evidenced by a label displayed on the carton of every Ben & Jerry's ice cream product—a label stating the company's commitment to the use of “natural ingredients” and expressing disapproval of the use of bST in cow's milk.

Conclusion

Public concern over these three “not-quite-great” scares—fluoridation, irradiation, and bST—has not mounted to a high pitch of anxiety. But the existence of these “lesser” scares does point up the American public's generalized fear of the unfamiliar—a fear not easy to dispel. And scaremongers habitually try to exploit this uneasiness—the vague feeling of misgiving that people commonly display in response to unfamiliar technologies and scientific innovations.

Unfortunately, the consequence of these scare tactics is twofold: Much time, effort, and money are spent refuting the scaremongers' false claims; and the activists' playing of the scare card delays the benefits these new technologies and processes have to offer. The public's anxiety about irradiation, for example, delayed its approval for the pasteurization of meat products in the U.S.—despite the fact that the process can kill *E. coli* and so might have halted the foodborne illnesses and deaths that preceded Hudson Food's recall of 25 million pounds of beef in the summer of 1997.

Thus, even as the activists are mounting scare campaigns to try to convince people that the increased use of chemicals and new technologies are increasing their health risks, the scientific evidence is demonstrating that technology is, in fact, helping to make the world a better—and safer—place.

¹ *Bulletin of the World Health Organization*. 1983; 61(5):871–883.

² Terry LL. *The Fourth Great Preventive Measure*. Public Health Service Publication No. 1552, Washington, DC: Government Printing Office; 1966:4.

- 3 World Health Organization. *Safety and Nutritional Adequacy of Irradiated Food*. Geneva: World Health Organization; 1994.
- 4 Joint FAO/WHO/IAEA Expert Committee. *Wholesomeness of Irradiated Food*. Report No. 659, Geneva: World Health Organization; 1981.
- 5 Lee P. Irradiation to prevent foodborne illness. *JAMA*. 1994; 272(4):261.
- 6 Bruhn CM, Wood OB. Position of the American Dietetic Association: food irradiation. *JADA*. 1996; 96(1):69–72.
- 7 *Radiation Preservation of Foods*. Chicago IL: Institute of Food Technologists; 1983.
- 8 World Health Organization. *Food Irradiation—Sky's the Limit*. Geneva: WHO Press Release. September 19, 1997.
- 9 Grossman, CH. Genetic engineering and the use of bovine somatotropin. *JAMA*. 1990; 264(8):1028.
- 10 Juskevich JC, Guyer CG. Bovine growth hormone: human food safety evaluation. *Science*. 1990; 249:875–884.
- 11 National Institutes of Health. Office of Medical Applications of Research. *Bovine Somatotropin: Technology Assessment Conference Statement*. Office of Medical Applications of Research; 1991:16.
- 12 National Milk Producer's Federation. *Breast Cancer Scare*. June 24, 1997.

Conclusions

What lessons can we learn from the above incidents? First, that many scares are fueled by the “precautionary principle”: “Where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent degradation.”¹ Thus, even in the face of incomplete or negative scientific information, action is taken to be on the “safe side”—the cellular phone issue is a good example of this.

But the problem with applying the “precautionary principle” is that—as in the cases of many of the scares discussed in this report—next to no evidence can be considered “lack of scientific certainty.”

Furthermore, many of these scares involved the extrapolation of effects in laboratory animals exposed to extremely high doses of the agent in question, or the mere detection of minuscule amounts of contaminants in the environment at levels that pose no human health threat; the scares tended to focus on man-made chemi-

cals rather than on naturally occurring substances; and the actions taken in response to the scares were driven not by the relative magnitude of the alleged risk, but rather by public perceptions of the importance of the questioned product in consumers' daily lives. (Hence the difference in the public responses to the ban on cyclamate and the ban on saccharin.)

We must not let the distraction of purely hypothetical threats make us lose sight of known or highly probable dangers. In a rational, technologically advanced society such as ours, we should be making decisions on the basis of what we know—not on the basis of what we fear.

Finally, there is little consideration given to whether the measures taken to address a given scare are truly cost effective; by focusing money and effort on a trivial or even nonexistent problem, we run the risk of diverting finite resources away from problems that pose real, significant risks to public health. Thus it is pertinent that we do not act immediately and reflexively on every hypothetical scare that comes along.

In closing, consider the following newspaper items, which were cited in a statement given by Dr. Elizabeth Whelan at the National Press Club on February 26, 1992 (the third anniversary of the Alar scare):

“Fruit contaminated, poses risk [of] cancer [the message conveyed in the headlines]. “Grocers removed fruit products from their shelves, restaurants dropped the allegedly tainted produce from their menus.”²

“[Officials in Ohio, Chicago and San Francisco] banned the sale of the suspect fruits.”³

Environmental groups and “pure food” advocates (which included a famous movie star) “maintain that chemical residues on agricultural products pose a threat to health”⁴

All these quotes appeared in *The New York Times* between November 11 and 22, 1959. The fruit in question was not apples, but cranberries. The worry-provoking chemical was not the growth regulator Alar, but the weed killer aminotriazole. The movie star was not Meryl Streep, but Gloria Swanson.

So must we merely sigh, shrug, and mutter, *Plus ça change, plus c'est la même chose* (the more things change, the more they remain the same)?

Let's hope not.

Let's hope that today's American public is more aware of the hallmarks of hypothetical “scares”; that people are capable of considering the facts rather than falling for media hype; and that they will place the facts—and the hype—into proper perspective the next time a scare appears on the horizon.

¹ United Nations Conference on Environment and Development (UNCED). Declaration of Rio, Rio de Janeiro, Brazil: UN 1992 (Principle 15).

² Cranberry crop facing huge loss. *The New York Times*. November 11, 1959:1.

³ Cranberry crop.

⁴ Pesticide scare worries manufacturers. *The New York Times*. November 22, 1959:1.

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