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Environmental Studies Program

Pesticides and the West Nile Virus

An Examination of Environmental Claims

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Introduction

Ever since the mosquito-transmitted West Nile Virus appeared in New York City during 1999, environmental activists have been fighting efforts to keep the disease under control. Not only have they battled against any spraying for adult mosquitoes, they have also fought methods to manage mosquito larvae. These groups maintain that the control methods are more dangerous than the diseases they seek to control. In the case of spraying, activists say that the chemicals imperil public health and can kill or harm wildlife. These groups claim further that other methods, such as the use of biological agents to kill mosquito larvae, disrupt the balance of nature and thereby threaten non-target species. Finally, while maintaining that spraying can devastate non-target organisms such as butterflies and aquatic life, many groups claim that mosquito control efforts have little or no impact on mosquito populations.

Of course, mosquito control officials should, and most arguably do, make careful consideration of the public health implications of their actions as well as any environmental tradeoffs. After all, their programs are designed to protect the public from serious health risks; officials certainly have no interest in creating new, greater risks.

The good news is that historically, pesticides and mosquito-control efforts have provided important public health benefits. “Contrary to the environmentalist view, public health campaigns that use insecticides against diseases have a remarkable record of public safety and a remarkable record of protecting humans from insect-borne diseases,” says Dr. Donald Roberts, professor of tropical public health at the Uniformed Services University of the Health Sciences.¹ Other factors certainly also have played significant roles in reducing disease incidence in developed nations. In particular, improved living conditions associated with growing income levels — which produced amenities such as screens and air conditioning — greatly limit human exposure to insect-borne disease. It is difficult to quantify total benefits of vector-control programs, but there is evidence of important benefits.

The Institute for Medicine, an affiliate of the National Academy of Sciences, documented some of these successes in a 1992 report. For example, it notes that vector-control efforts helped mitigate an outbreak of St. Louis Encephalitis in 1966 during which a mosquito-transmitted form of viral encephalitis broke out in Dallas, Texas. Within a few weeks, there were 145 confirmed cases and 545 suspected cases. Dallas vector-control officials responded by spraying with the pesticide malathion over thousands of acres of land. The Institute reports that measurements of mosquito densities before and after spraying showed that spraying had significantly reduced the mosquito populations. The Institute concluded that: “The economic and public health consequences would certainly have been greater had pesticides not been available.”²

Similarly, U.S. vector-control efforts made possible the construction of the Panama Canal, which was impeded by yellow fever and malaria outbreaks. In particular, U.S. engineers controlled malaria and yellow fever through a combination of drainage and application of

¹ Personal communication on January 28, 2002.

² Joshua Lederberg, Robert E. Shope, and Stanley C. Oaks, Jr. eds., *Emerging Infections: Microbial Threats to Health in the United States* (Washington, D.C.: National Academy Press, 1992), 166.

larvicides, which reduced mosquito breeding.³ Similarly, governments have used the pesticide DDT to basically eradicate malaria-carrying vectors throughout much of the western world.⁴

Like anything, vector control hasn't proven problem free. Challenges associated with insect resistance, limitations of control methods, and impacts on wildlife continually emerge and must be managed. Environmental activists point to such issues — often exaggerating the extent of problems — to suggest that vector control, and pesticides in particular, should be eliminated. But just as we don't eliminate medical antibiotics because resistance presents challenges, we should not eliminate vector control. We should instead look for ways to manage and address such challenges.

Indeed, when governments followed anti-pesticide activists' advice in the past, it produced deadly results. The most egregious case involves the pesticide DDT. Some evidence indicates adverse impacts on wildlife from widespread use of DDT for agricultural purposes, harming the bald eagle and other birds of prey in particular. Others have suggested that DDT poses seriously adverse public health implications as well, but such claims are questionable.⁵ Environmental groups played an important role in raising concerns about DDT's impact on wildlife, but rather than providing constructive advice to manage risks, they decided that DDT should never be used. They have advanced bans around the world despite a resulting escalation of malaria deaths in developing nations. Use of limited quantities of DDT in and around residences can keep mosquitoes outside homes at night when the malaria-carrying insects feed. Such uses have negligible environmental impact, but can save millions of lives.

The adverse public health impacts of such extreme positions have been felt around the world. For example, South Africa nearly eradicated malaria-carrying mosquitoes when it used DDT, but cases soared again after the nation caved to environmental activists who pressed the country to switch to other pesticides. Cases rose from about 4,000 in 1995 to more than 27,000 by 1999 (or possibly as many as 120,000 if one considers pharmacy records).⁶ In response to this crisis, South Africa decided to resume DDT use. Similarly, tropical medicine specialist Dr. Donald Roberts and his colleagues explain in a research paper that “separate analyses of data from 1993 to 1995 showed that countries that have recently discontinued their spray programs are reporting large increases in malaria incidence. Ecuador, which has increased use of DDT since 1993, is the only country reporting a large reduction (61%) in malaria rates since 1993.”⁷ According to the World Health Organization, malaria alone infects 300 to 400 million people a year and kills 1 to 2 million.⁸ Most of its victims are children.

³ Ibid., p. 20.

⁴ For a discussion of the history of DDT use for malaria control see Richard Tren and Roger Bate, *When Politics Kills: Malaria and the DDT Story* (Washington, D.C.: Competitive Enterprise Institute, 2000), <http://www.cei.org/PDFs/malaria.pdf>.

⁵ See: A. G. Smith, “How Toxic is DDT?” *Lancet* 2000 356 (July 22, 2000): 267-268.

⁶ Amir Attaran and Rajendra Maharaj, “Doctoring Malaria, Badly: The Global Campaign to Ban DDT,” *British Medical Journal*, no. 321 (December 2, 2000): 1403-1405, <http://bmj.com/cgi/content/full/321/7273/1403#resp1>.

⁷ Donald R. Roberts et al., “DDT, Global Strategies, and a Malaria Control Crisis in South America,” *Emerging Infectious Diseases* 13, no. 3 (July-September 1997), <http://www.cdc.gov/ncidod/eid/vol13no3/roberts.htm>. See also Amir Attaran. *et al.*, “Balancing Risks on the Backs of the Poor,” *Nature Medicine* 6 (2000): 729-731.

⁸ World Health Organization, “Malaria: Fact Sheet,” 1998, <http://www.who.int/inf-fs/en/fact094.html>.

Because of the devastating death toll, public health officials including those at the World Health Organization took a stand against an unreserved international ban on DDT.⁹ These officials provided the support necessary to allow DDT use for malaria control as part of the Convention on Persistent Organic Pollutants. Nonetheless, environmental activists continue their crusade against DDT.

Environmental groups in the United States are posing similar public health challenges — although on a less devastating scale — by fighting any reasonable use of chemicals or other so-called “unnatural” means for vector control. Rather than offer sound advice on management of public exposure, insect resistance, or efficacy of the products and methods — all which present challenges for vector control — these groups downplay the public health risks of insect-borne disease and attempt to raise alarm about substances used for vector control. Anti-pesticide campaigns and subsequent government regulations have impacted the ability to control emerging diseases. “The practices of environmental advocacy groups are seriously degrading public health capabilities in the United States. Our public health threats are real, and growing,” says Dr. Roberts.¹⁰

Activists’ extreme views are coupled with alarmist rhetoric that is designed to scare public officials and others into opposing any chemical use. The following catalogs some of their claims and offers analysis as to where they contradict the facts.

The “Art” of Environmental Alarmism

The key strategy of anti-pesticide activists is to raise alarm about risks to humans and wildlife. That often means taking mundane and factual information and making it appear far more alarming than is reasonable. Other times, it involves making questionable claims that are not easily checked by the average person.

In addition, the appeal to the so-called “precautionary principle” is pervasive in environmentalist literature. This principle basically suggests that society should avoid any technologies — particularly chemicals — until they are proven safe. And many interpret that to mean ensuring absolute safety at all concentrations and all exposure levels and when combined with other agents. While such appeals sound reasonable, they ignore some important facts. There will always be scientific uncertainty about all technologies, as everything in life carries risks. And taking some risks in life is necessary and valuable. Individuals take reasonable risks because of the tremendous benefits we gain. As CEI’s Fred Smith notes: “Experience demonstrates that the risks of innovation, while real, are vastly less than risks of stagnation.”¹¹ Indeed, he asks, what would the world be like if we never introduced penicillin because we could not prove it’s 100 percent safe?

⁹ For example see “DDT-Malaria: Open Letter” supporting limited DDT use and opposing a worldwide ban, available at http://www.malaria.org/ddtcover_english.html and the list of signatures at http://www.malaria.org/DDT_signatures.html.

¹⁰ Personal communication on January 28, 2002.

¹¹ Fred L. Smith, “The Dangers of Precaution,” *Financial Times*, February 23, 2000.

Some products are beneficial because of their innately risky nature. Chemicals that are designed to kill insects and pathogens must carry some risk or they would not provide public health benefits. Drugs pose risks and often carry side effects, but we take them nonetheless to ward off more serious public health consequences. In a world laden with risks, so-called “precautionary” policies that prevent technologies actually represent the truly risky approach. Pleas for such policies should be viewed with great skepticism.

Unfortunately, the concept has even penetrated more mainstream scientific literature. In August 2003, *Science* magazine published an article suggesting that it was appropriate for researchers to raise alarm about risks and advance public expenditures to address potential crises, even when the probability of actual crises may be low. The author assumed it was safer to exercise “precaution” by responding to all potential calamities because one of the many alarm scenarios might actually come to pass. Yet this argument completely ignores the danger of diverting vast amounts of resources away from real problems to address highly improbable events.¹²

The Art of Rhetoric

Groups often make statements and use carefully chosen rhetoric that leads to alarming conclusions when in reality, the substance of their comments don’t mean much at all. As a result, you can’t accuse these individuals of lying, but the implications of what they say might suggest something that is not consistent with reality. In other cases, rhetoric is used to raise alarm by exaggeration of facts or by mischaracterization of what the facts mean. Often this entails leaving out key information, which otherwise would lead to a strikingly different conclusion. Consider some examples:

“Pesticides sprayed in response to West Nile Virus endanger our health, put our kids at risk, and poison our environment.” Why? The paper asserts: “Pesticides are toxic. In fact, it is illegal to call any pesticide ‘safe.’”¹³

“We’re not confident any level of use can be considered safe.”¹⁴

Malathion [one of the pesticides used for mosquito spraying] is as bad as it gets,” said Dr. Philip Landrigan, chairman of the department of community and preventive medicine at Mount Sinai School of Medicine in New York City. “There is no such thing as a safe pesticide. It’s a matter of looking at benefits.”¹⁵

¹² S.W. Pacala et al, “False Alarm over Environmental False Alarms,” *Science* 301, no. 5637 (August 29, 2003): 1187-1188.

¹³ “Say No to Pesticide Spraying to Control the West Nile Virus: Protect Our Health!” posted on the web page of the Neighborhood Pesticide Action Committee of Jamaica Plain, Massachusetts, http://www.orgsites.com/ma/npac/_pgg3.php3.

¹⁴ Carmela Fragomeni, “News,” *The Hamilton Spectator* (Canada), August 31, 2002 quoting Rich Waite of the Toronto Environmental Alliance.

¹⁵ Julie Deardorff and Karen Mellen, “Metro Spraying Targeting Hosts of Nile Virus,” *Chicago Tribune*, August 21, 2002.

Pesticides are indeed toxic because they are designed to be toxic — to bugs. That does not mean that low-level exposures to humans are much of a concern, despite suggestions to the contrary.

In addition to using the “toxic” claim, activists regularly point out that it is not legal or appropriate to call a pesticide “safe” — creating the impression that, even when used appropriately, these products should be feared. But the word “safe” can be used in both absolute and relative terms. Most people use the word in relative terms. For example, most of us think that eating healthy food or taking a shower is safe. We know that we could choke or slip, but we deem such things “safe.” But anti-chemical activists’ use the word “safe” in absolute terms, saying pesticides are not safe simply because no one can prove anything 100 percent safe. Since everything carries risks, decisions regarding pesticide use should focus on whether risks warrant great concern and if inaction poses greater risks. As the following will detail, the data on public health uses of pesticides indicates that risks are negligible, and that risks associated with vector-borne illnesses have proven significant throughout human history.

Moreover, the quote stating that it is illegal to call a pesticide safe implies that it is illegal for *anyone* to make that statement. Actually, the law applies to manufacturers in regard to labels and advertising.¹⁶ Since they cannot prove the chemicals are absolutely safe, those selling the product cannot make that claim. Such labeling regulations apply to commercial producers of a variety of products for a range of reasons, some to demand precision and some for political reasons. Pesticide safety label mandates are not much different from FDA labeling regulations that prevent manufacturers from guaranteeing certain drug benefits and that require disclosure of risks. The Federal Trade Commission also applies labeling restrictions to prevent obviously fraudulent claims.

In this case, Dr. Landrigan isn’t saying very much at all, yet it sounds ominous. What does “as bad as it gets” really mean? It sounds like he’s saying that malathion is a terrible poison. But if malathion doesn’t pose much of a risk and it’s the worst risk out there among pesticides, then this comment indicates that most pesticides pose less of a risk than this relatively innocuous product. However, Dr. Landrigan is well known for his anti-pesticide claims and is probably well aware of the implications of the message delivered.

Creative “Science”

“New York City’s indiscriminate spraying ... has put the public’s health and natural environment in great danger. ... As a direct effect of the pesticide exposure, thousands of people suffered impaired respiratory and neurological health, including many of the workers who were temporarily hired by the City to do the actual spraying. Thousands more are expected to experience long term health problems which may not manifest as symptoms for many years including cancer, hormonal imbalances, neurological damage and possible genetic mutations.”¹⁷

¹⁶ The law prevents “misbranding” of pesticides at 7 USC § 136j(a)(1)(E) and 7 USC §136(q)(1)(A). Environmental Protection Agency regulations prohibit claims related to safety at 40 CFR 156.10(a)(5), http://www.access.gpo.gov/nara/cfr/waisidx_03/40cfr156_03.html.

¹⁷ Posted on the Web page of Hudson River Sloop Clearwater, Inc. Poughkeepsie, New York, <http://www.clearwater.org/news/nospray.html>, also posted on www.nospray.org.

“In 1999 and 2000, New Yorkers were exposed to massive amounts of toxic pesticides ... Despite overwhelming evidence of the hazards of indiscriminate pesticide use ... Malathion (Fyfanon ULV), is described by the NYC Mayor’s Chem-Bio Handbook — the City’s official guide to handling chemical and biological emergencies which is distributed to every police precinct, ambulance and fire truck — as a toxic nerve gas directly related to those used in WWII. Anvil ... the synthetic pyrethroid nerve gas sprayed from trucks in 2000, is known to cause asthma, disruption of sexual hormones and various other health disorders, and has been linked to breast cancer in women and diminished sperm counts in men.”¹⁸

The rhetoric here is colorful and scary, but not reflective of reality. What constitutes “massive” amounts may perhaps be a personal view, but it’s arguable that the amounts were quite small and not applied “indiscriminately.” Sprayers employ “ultra-low volume” spraying methods, which release very small amounts of pesticides. For example, spray applications for a category of pesticides called pyrethroids typically amount to two to three-and-a-half ounces per acre.¹⁹

Perhaps more alarming are claims that these substances are “known” to cause problems or that they are “linked” to certain ailments. Some activists make such claims in a way that appears more credible by citing select (and often numerous) scientific studies that “suggest” a possible link or report a weak association between a chemical and a health ailment. But the fact that one study might report a link to problems does not mean that it actually poses a significant risk.

Environmental activists often make such claims based on studies that find statistical associations. Statistical analysis can find associations among many things, but association does not prove cause-and-effect. The National Cancer Institute notes that there is much more that goes into determining whether a link is probable.²⁰ Of note are:

- Strength of the association: Is the relationship strong or weak? For example, did a high percentage of subjects exposed to a chemical develop cancer or was the percentage so low that the number could be attributed to mere chance.
- The consistency of the association: Do many studies find this association or is the finding an aberration?
- The dose-response relationship: Was the dose large enough to pose a serious risk?
- The order of events: The association must be temporally correct; the alleged cause must precede the effect. For example, if an illness occurs before exposure to a chemical, you can’t blame the chemical.
- Biologically plausibility: The finding is stronger if there is a good biological explanation as to the link.

¹⁸ Robert Lederman, “Why is the Media Hiding The Truth About the NYC ‘Epidemic’ and the Deliberate Misuse of Malathion?” August 24, 2000, posted on the No Spray website at http://www.nospray.org/aL_824truth.html.

¹⁹ U.S. Environmental Protection Agency, “Synthetic Pyrethroids For Mosquito Control,” Updated April 17, 2002, <http://www.epa.gov/pesticides/factsheets/pyrethroids4mosquitos.htm>.

²⁰ National Cancer Statistics Branch, National Cancer Institute, *Cancer Incidence and Survival among Children and Adolescents: United States SEER Program 1975-1995* (Washington, D.C.: National Institutes of Health, 1999), <http://seer.cancer.gov/publications/childhood>.

Researchers must also consider whether there are likely confounding factors — things that might have affected the study findings. For example, a statistical analysis might find that people who spend a great deal of time in bars have a higher cancer rate. Does that mean that going to a bar can give you cancer? Probably not. Instead, it might be that smoking rates are higher among those who spend time in bars. Hence, smoking, not going to bars, would be the problem.

In addition, studies can produce associations by accident. Chance can explain many studies, many of which we see quoted in the press, that report all kinds of crazy associations. Because of such challenges, studies emerge with all sorts of findings, reporting potential links where none may actually exist and failing to find a link when one does exist. For these reasons, rarely is a single study all that conclusive. If a number of studies of substantial size meet the standards outlined by the National Cancer Institute in the list above, then it is fairer to make the case for a probable link.

Anti-chemical activists also make such claims based on studies conducted on rodents. While such studies can improve understanding of how chemicals react on living beings, these too have their limitations. Many of these entail administering massive amounts of chemicals to rodents bred to be highly susceptible to cancer. Then researchers extrapolate the possible effects of such chemicals on humans who may be exposed to small amounts of the same chemical over their lifetime.

First, one should ask: Are the impacts on rodents relevant to humans? Researchers Sir Richard Doll and Richard Peto note that some chemicals found to be carcinogenic in humans have not produced cancerous tumors in rodent experiments.²¹ In fact, for many years, cigarette smoke failed to produce malignant tumors in laboratory animals despite the fact that tobacco is perhaps the leading cause of cancer in the United States. These discordant effects of chemicals in animals and humans underline the difficulty of relying on animal results to estimate human risks.

Second, one should consider whether the extremely high doses administered in the lab are relevant to low-level exposures in the real world. Researchers have discovered that it may be the dose that is causing tumor formation. At such high levels, the rodent's body responds to the overload by cell proliferation, increasing possibility of accidental mutations, which result in cancer.²² Hence, many substances may be labeled carcinogens when in fact, cell proliferation produced by the test — not the chemical — caused accidental cell mutation.

Some chemicals act directly on the cells in a way that produces mutations. However, there are still reasons why we need not fear these substances at very low levels. Some don't cause cancer until the exposure level reaches a certain threshold and when exposure is prolonged over many years, often decades. In many cases, some substances have beneficial effects at low levels — such as the essential mineral magnesium — while they are dangerous at high levels.

²¹ Richard Doll and Richard Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," *Journal of the National Cancer Institute* 66, no. 6 (June 1981).

²² Bruce Ames and Lois Swirsky Gold, "Too many Rodent Carcinogens: Mitogenesis Increases Mutagenesis," *Science* 249 (August 31, 1990): 970.

Low-level exposures need not raise alarm because the body employs repair mechanisms, enabling it to correct many cell mutations. In fact, both man-made and naturally occurring chemicals bombard the human body on a regular basis, causing cell damage. Because of repair mechanisms, the body is able to address low-level exposures and any resulting damage. But when exposed to some substances at higher levels for long periods of time, the body is less able to repair all damage, and cancer can result.

Hence, a big part of the equation involves the dose and term of exposure. Rodent studies can reveal information about risks of substances at high levels, but often tell us little about low-level exposures. It is fair to assume that low-level exposures even of substances that cause cancer in lab rats are not a grave concern, despite what environmental activists often suggest. Scientists Bruce Ames and Lois Swirsky Gold have found that we are exposed to thousands of naturally occurring carcinogens in our diet on a daily basis without adverse effects.²³ Rodent carcinogens are found in apples, broccoli, lettuce, coffee, and dozens of other healthy foods. In addition, plants we eat have created their own natural pesticides, which we consume daily at levels that are far higher than that of man-made pesticides. Ames and Gold estimate that 99.99 percent (by weight) of the pesticides humans consume are natural pesticides.²⁴

Proper use of science considers the limitations of both rodent studies and statistical associations. When drawing conclusions, one must consider the full body of evidence, such as the criteria noted earlier regarding statistical association as well as the importance of dose-response relationships.

However, environmental activists often don't follow this advice. Instead, they take liberties with the limited abilities of science. Often they raise alarm based on select citation of studies in which there are associations that are not well established throughout the literature, or they cite studies that are merely "suggestive" that a risk might exist. "Suggestive" findings represent nothing much at all. Many times, when researchers have found no link but note that the possibility of a link remains, activists will even cite those studies as evidence of a link. In addition, they will often say that a pesticide applied at a very low level in the environment is a public health threat based on rodent studies that find cancer at levels that are tens of thousands of times higher.

Often times, activists will lump together a group of studies that report weak associations or have "suggestive" findings to suggest that while no single study proves their point, the body of research indicates a problem. But something that proves nothing much alone does not become stronger when added to a list of other studies that don't prove much at all. Instead, conclusions are drawn when many substantial studies have strong associations. Other tactics involve citing unpublished, non-peer reviewed "studies" and data or producing non-peer reviewed studies that allege a host of claims. The best science survives the scrutiny not only of peer reviews but of others who can access the data in an attempt to replicate conclusions.

²³ Ibid.

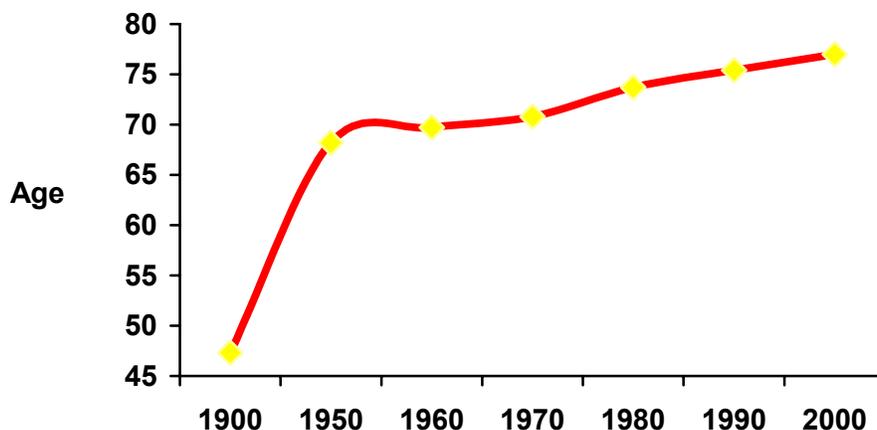
²⁴ Ibid.

Public Health Claims verses Realities

In the end it is very difficult for anyone, particularly the average member of the public, to address every claim and every source of environmentalist hype. The best approach for addressing such claims is to look not only at what the body of scientific literature states regarding the specific claims, but to examine what public health record reveals. As chemical and pesticide use has increased throughout decades, are people getting sicker and experiencing greater cancer rates as environmentalist rhetoric suggests? The public health record indicates the opposite. According to the World Health Organization, the average worldwide human life span has increased from 45 years in 1950 to about 66 in 2000 and will most likely continue to increase to 77 years by 2050.²⁵ In the United States, it has reached 76 according to a recent estimate.²⁶ In 1915, the infant mortality rate was about 10 percent of all live births.²⁷ Today, the infant mortality rate is less than 1 percent, and we continue to make progress.²⁸

Life Expectancy

Source: National Center for Health Statistics



Yet activists claim that cancer risks have mounted with the use of chemicals. The Environmental Working Group notes, for example:

“Cancer incidence in the American population has skyrocketed — up 48% from 1950 through 1990, according to National Cancer Institute statistics. These statistics are adjusted for an aging

²⁵ International Agency for Research on Cancer, World Health Organization, *World Cancer Report* (Lyon: IARC Press, 2003), 320.

²⁶ U.S. Centers for Disease Control and Prevention (CDC), Bureau of the Census, *Health, United States, 2000* (Washington, D.C.: CDC, 2000), 7, <http://www.cdc.gov/nchs/data/hus00.pdf>.

²⁷ U.S. Centers for Disease Control and Prevention, *CDC Fact Book 2000/2001* (Washington, D.C.: USCDC, 2001), 11, <http://www.cdc.gov/maso/factbook/Fact%20Book.pdf>.

²⁸ *Ibid.*

population and exclude lung and stomach cancers where the causes are generally well-understood.”²⁹

A cursory review of the data on cancer might draw one to conclude that a rise in cancer rates do in fact coincide with increased chemical use during the 20th Century. Raw data appear to show a rising cancer rate, but such conclusions arise from the failure to account for changes in population and lifestyle decisions. For example, one researcher failed to account for population increases in a chart plotting cancer incidence between 1900 and 1990. By not accounting for the fact that population increased from 77 million to 250 million, this researcher created the impression that cancer rates skyrocketed.³⁰ Instead cancer rates should be measured in units of population, such as the number of cancers per 100,000 people.

In addition, analysis of cancer trends should consider the age of various segments of the population. Since cancer is a disease related to aging, older segments of the population inevitably have higher cancer rates. Hence, if the segment of senior citizens in a population grows more quickly compared to the others, we should expect more cancers per 100,000. Hence as the baby-boomer generation ages, total cancer numbers increase. Moreover, as life expectancy increases, so do cancer rates. Not surprisingly, the World Health Organization reports that cancer deaths and incidence grew 22 percent between 1990 and 2000. These trends are expected to continue regardless of chemical use because, as the World Health Organization reports, the number of individuals over 60 is expected to triple by 2050.

When factors like smoking and an aging population are considered, cancer trends are far less ominous (although the Environmental Working Group somehow claims they considered such factors). According to a 1981 landmark study on cancer trends conducted by scientists Sir Richard Doll and Richard Peto, cancer rates have remained nearly constant in the United States during the 20th Century except for cancer rate increases due to smoking. Improvements in medical technology, more accurate identification and reporting of cancer cases, and, most importantly, increasing life expectancies which result in more people reaching the old ages at which cancer is more likely to occur only make it appear as if rates increased.³¹

In 2001, researchers from the University of Alabama analyzed data from the Centers for Disease Control and Prevention (CDC), cataloging cancer rates from 1950 to 1990. When all smoking-related cancers were removed, they found that cancer had declined continuously since 1950 for a total drop of 25 percent. They pointed out that “a typical commentary ‘blamed’ increasing cancer rates on ‘exposure to industrial chemicals and run-away modern technologies whose explosive growth had clearly outpaced the ability of society to control them.’” Yet the researchers explained: “There is no denying the existence of environmental problems, but the present data show that they produced no striking increase in cancer mortality.”³²

²⁹ Environmental Working Group, “Pesticide Industry Propaganda: Myth #3: Cancer Rates Are Decreasing, Or: ‘We’re Winning The War On Cancer,’” posted online at: <http://www.ewg.org/pub/home/Reports/Myths/Myth3.html>.

³⁰ Charles B. Simone, *Cancer & Nutrition* (New York: McGraw-Hill, 1983), as noted in William M. London and John W. Morgan in “Living Long Enough to Die of Cancer,” *Priorities* 7, no. 4 (December 31, 1995).

³¹ Richard Doll and Richard Peto, “The Causes of Cancer.”

³² Brad Rodu and Philip Cole, “The Fifty Year Decline of Cancer in America,” *Journal of Clinical Oncology* 19, no. 1 (January 1, 2001): 240-41.

Researchers at the National Cancer Institute, in collaboration with other cancer experts around the world, annually produce one of the world's best reports on cancer trends. In recent years, they have reported that total cancer rates — including smoking related cancers — declined in the 1990s.³³ In their 2001 report, the National Cancer Institute researchers noted: “Cancer incidence for all sites combined decreased from 1992 through 1998 among all persons in the United States, primarily because of a decline of 2.9 percent per year in white males and 3.1 percent per year in black males. Among females, cancer incidence rates increased 0.3 percent per year. Overall, cancer death rates declined 1.1 percent per year.”³⁴ There is some incidence increase among women, but such increases are related to such things as better breast cancer detection and the fact that women as a group have taken longer to reduce smoking rates than have men. The National Cancer Institute reported in 2003 that cancer rates stabilized in recent years, but it notes that further reductions are possible given efforts to disseminate cancer-control information.³⁵

Environmental activists continually hype that certain cancer risks are on the rise because of chemical use. In particular, they have maintained that breast cancer rates have risen because pesticides have somehow “disrupted” the human endocrine system.

“The Breast Cancer Prevention Coalition (BCPC) believes that continuing escalation in cancer incidence is due in no small part to our exposure to thousands of synthetic chemicals and radioactive pollutants released continuously into our workplaces and the environment at large, as well as in the products we use on a day-to-day basis.”³⁶

“CCE [Citizens Campaign for the Environment] has actively supported efforts to reduce pesticides in food and environmental exposure, including sustaining strong organic farm standards. Pesticide exposure may be a risk factor in breast cancer.”³⁷

However, the National Cancer Institute notes that breast cancer rates only appear to be higher because better screening and increased detection are finding more cancers.³⁸ Given the increased number of women receiving mammograms, this is a highly likely scenario. The percent of women aged 40 to 49 who obtained mammograms doubled between 1987 and 1998 from 32 percent to 63 percent. The percent of women aged 50 to 64 who received a

³³ The National Cancer Institute reports: “The American Cancer Society, the National Cancer Institute, the North American Association of Central Cancer Registries, and the Centers for Disease Control and Prevention, including the National Center for Health Statistics and the Center for Chronic Disease Prevention and Health Promotion collaborate to produce an annual report on the current burden of Cancer in the United States; Holly L. Howe et al., “Annual Report to the Nation on the Status of Cancer (1973 Through 1998), Featuring Cancers with Recent Increasing Trends,” *Journal of the National Cancer Institute* 93 (June 6, 2001): 824-42.

³⁴ Holly L. Howe, et al., “Annual Report to the Nation on the Status of Cancer.”

³⁵ Hannah K. Weir et al., “Annual Report to the Nation on the Status of Cancer, 1975-2000, Featuring Uses of Surveillance Data for Cancer Prevention and Control,” *Journal of the National Cancer Institute* 95, no. 17 (September 3, 2003): 1276-1297.

³⁶ Breast Cancer Prevention Coalition, based in Ontario, Canada, “Philosophy,” posted on website: <http://www.stopcancer.org/join/philosophy.html>.

³⁷ Citizens Campaign for the Environment, “Public Health and Toxic Chemical Contamination,” posted online at: http://www.citizenscampaign.org/programs/public_health.html.

³⁸ Howe et al., “Annual Report to the Nation on the Status of Cancer.”

mammogram increased from 31 to 73 percent in that same time period.³⁹ In addition, breast cancer risks increase when children delay child bearing. Women who delay child bearing until after 30 or don't have children have a two-to-threefold increased risk for breast cancer. According to the National Cancer Institute, "the greater number of women who are delaying childbirth or remaining childless may explain some of the recent increased incidence of breast cancer."⁴⁰

Activists have also suggested that pesticides have provided elevated cancer rates in various parts of the country, such as in Long Island, New York.⁴¹ There have been many studies on breast cancer and pesticides — most of which focused on DDT and PCBs. One study claimed to find a link in 1993,⁴² but it was not deemed definitive in part because of its relatively small sample size. Subsequent studies of greater scope could not find a link.⁴³ The National Research Council concluded in 1999, in a report reviewing the "endocrine disrupter" issue, that the original breast cancer study and all the ones published before 1995 "do not support an association between DDT metabolites or PCBs and the risk of breast cancer."⁴⁴

More recently, U.S. researchers produced one of the largest and most comprehensive studies ever on the topic, assessing the impact of pesticides on breast cancers among women in Long Island, New York. These researchers could not find a link between the breast cancers and the chemicals most often cited as the problem (DDT and other pesticides as well as PCBs).⁴⁵

Not emphasized by environmental activists is that modern medicine is saving women from breast cancer. The National Cancer Institute report notes that death rates from breast cancer decreased by 1.6 percent for all races combined from 1989 through 1995. Between 1995 and 1998, the death rate declined even faster at a rate of 3.4 percent.⁴⁶

Along with breast cancer, groups say that "estrogenic" effects of pesticides cause prostate cancer. The National Cancer Institute reports that prostate cancer incidence increased after 1973 at a rate of 2.9 percent annually and then at a steeper rate when improved screening methods identified more cases. But then rates declined by 11 percent annually between 1992 and 1995, and have since leveled off. Mortality follows a similar trend, with mortality declining between 1995 and 1998 at a rate of 4.7 percent for white males and 3 percent for African American males.

³⁹ U.S. Centers for Disease Control and Prevention, *CDC Fact Book 2000/2001*, 46, <http://www.cdc.gov/maso/factbook/Fact%20Book.pdf>.

⁴⁰ Celia Byrne, National Cancer Institute, "Risk Factors: Breast," *Cancer Rates and Risks* (Washington, D.C.: National Cancer Institute), online publication: <http://seer.cancer.gov/publications/raterisk/index.html>.

⁴¹ For example, see Hudson River Sloop Clearwater, Inc., "Breast Cancer Risk: The Environmental Factors, Fact Sheet 7," *Clear Water News & Bulletin*, April 17, 1997, <http://www.clearwater.org/news/fs7.html>.

⁴² Mary S. Wolff et al., "Blood Residues of Organochlorine Residues and Risk of Breast Cancer," *Journal of the National Cancer Institute* 85 (April 21, 1993): 648-652.

⁴³ For an overview of this debate and subsequent studies, see Angela Logomasini, "Endocrine Disrupters," *The Environmental Source* (Washington, D.C.: Competitive Enterprise Institute, 2001), 31-36, <http://www.cei.org/gencon/026.01623.cfm>.

⁴⁴ National Research Council, *Hormonally Active Agents in the Environment* (Washington, D.C.: National Academy Press, 1999), 250.

⁴⁵ Marilie D Gammon Ph.D. et al., *Cancer Epidemiology Biomarkers Prevention* 11, no. 8 (August 2002): 677-85.

⁴⁶ Holly L. Howe, "Annual Report to the Nation on the Status of Cancer," 824-842.

Recent increases in prostate cancer should be of no surprise given that people are living longer. Most prostate cancers occur after age 55 and most are not detected until age 70.⁴⁷

However, the National Cancer Institute researchers contend that the rate of increase is not fully explained by better detection. Environmental factors may be causing some additional cancers, but they do not cite environmental exposures of chemicals as a likely cause. Instead, dietary factors, such as increased intake of animal fats among the population, or greater sexual activity are more likely sources. Occupational exposure to pesticides (which is far higher than public exposure) is noted as a possibility, but not a strong one since “it is unclear if this finding is the result of occupational factors or to concomitant lifestyle factors.” Occupational exposures to other chemicals show only “weak associations” and are far from conclusive.⁴⁸ In any case, such occupation exposures are not particularly relevant to public exposures to much lower levels of substances.

Activists also have suggested that pesticides and chemicals in general have contributed to growing rates of brain and other cancers among children. One group called the Center for Children’s Health and the Environment has run an advertising campaign against chemicals. In one advertisement, they proclaim:

“More children are getting brain cancer. Why? Toxic chemicals appear to be linked to rising cancer rates.”⁴⁹

But according to the National Cancer Institute, the trends are anything but alarming. Cancer incidence among children is stable and we are experiencing “dramatic declines” in childhood cancer mortality overall. In 1999, the Institute concluded:

“There was no substantial change in incidence for major pediatric cancers, and rates have remained relatively stable since the mid-1980s. The modest increases that were observed for brain/CNS [central nervous system] cancers, leukemia, and infant neuroblastoma [cancer of the sympathetic nervous system] were confined to the mid-1980s. The patterns suggest that the increases likely reflected diagnostic improvements or reporting changes. Dramatic declines in childhood cancer mortality represent treatment-related improvement in survival ... recent media reports suggest that incidence is increasing and that the increases may be due to environmental exposures. However, these reports have not generally taken into consideration the timing of changes in childhood cancer rates, or important development in the diagnosis classifications of childhood cancers.”⁵⁰

Anti-chemical activists nonetheless point out that most cancer is caused by environmental factors, and that alone, should warrant reduction and elimination of pesticides:

⁴⁷ “Stat Bite: Incidence Rates by Age at Diagnosis for Breast and Prostate Cancers,” *Journal of the National Cancer Institute* 93 (March 21, 2001): 425.

⁴⁸ Richard B. Hayes, “Risk Factors: Prostate,” *Cancer Rates and Risks* (Washington, D.C.: National Cancer Institute), online publication, <http://seer.cancer.gov/publications/raterisk/risks185.html>.

⁴⁹ Center for Children’s Health and the Environment; The advertisement is available online: <http://www.childenvironment.org/images/ad2big.pdf>.

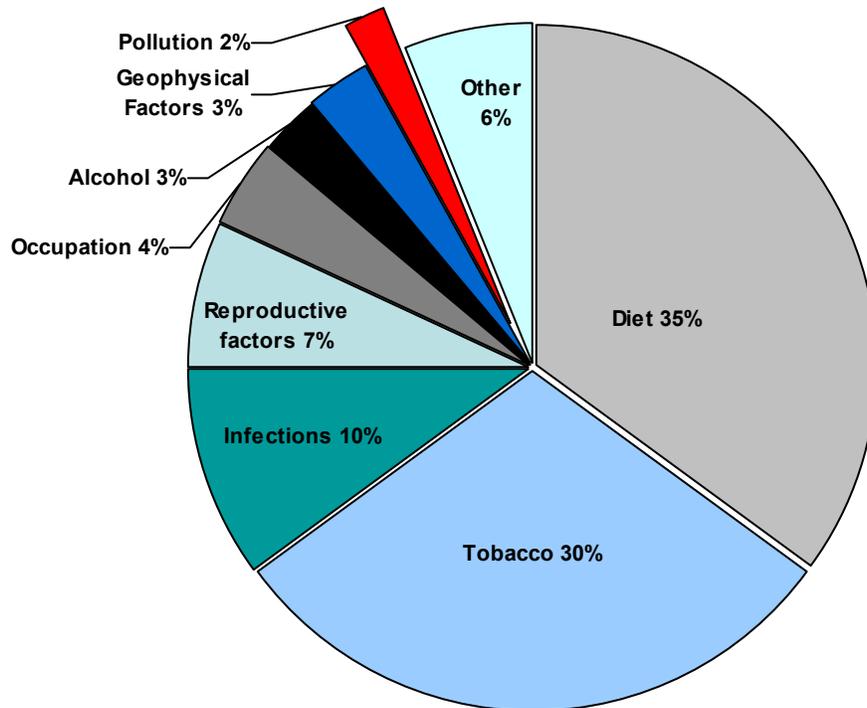
⁵⁰ Martha S. Linet et al, “Cancer Surveillance Services: Recent Trends in Childhood Cancer Incidence and Mortality in the United States,” *Journal of the National Cancer Institute* 91 (June 16, 1999): 1051-1058.

“Many researchers now believe that environmental factors may together account for a large proportion of breast cancer risk — from 50% to 70%. In the Hudson Valley we have three environmental factors working against us: Pesticides ... PCBs ... Radiation.”⁵¹

When anti-chemical activists make such claims, it appears that they are suggesting that environmental pollution and man-made chemicals causes 50-70 percent of cancer. The World Health Organization cites research of Doll and Peto on this regard, which does indeed note that 80 to 90 percent of all cancers are caused by “environmental factors.” Yet this phrase encompasses anything other than genetics. It does not include pollution alone. Environmental causes of cancer include smoking, diet, occupational exposure to chemicals, “geophysical factors” (such as naturally occurring radiation) man-made radiation, medical drugs and radiation, and pollution. According to Doll and Peto’s report, pollution accounts for only 2 percent of all cancers. Doll and Peto report that tobacco use accounts for about 30 percent of all annual cancer deaths, and dietary choices (excluding chemical additives and pesticide residues, which are considered as part of the 2 percent for pollution) account for 35 percent of annual cancer deaths.⁵²

Environmental Causes of Cancer

Source: Poll and Peto



Nonetheless, the developed world’s aging population does indeed present new health challenges that are important to address. The World Health Organization suggests that cancer

⁵¹ Hudson River Sloop Clearwater, “Breast Cancer Risk: The Environmental Factors,” <http://www.clearwater.org/news/fs7.html>.

⁵² Doll and Peto, “The Causes of Cancer,” For a summary of causes, see chart on page 1256.

prevention efforts should focus on three factors: tobacco use, diet, and infections, which together, the World Health Organization says account for 75 percent of cancer cases worldwide.⁵³ Encouraging people to change personal habits is deemed most effective cancer prevention policy. Elimination of public health use of pesticides is not even on this list.

Federal Safety Standards

Environmental activists downplay government safety standards, stating that they don't demonstrate safety. It's true that nothing is 100 percent safe. But unlike environmental groups, government agencies, such as the Environmental Protection Agency (EPA) and the U.S. Agency for Toxic Substances and Disease Registry (ATSDR), are required to review the body of scientific literature and to develop conclusions about risk, which offers a far clearer picture of the situation. Despite activist claims, the findings of government agencies tend to be excessively cautious; if anything they overstate risks, sometimes by factors in the tens of thousands.

EPA traditionally sets standards that only allow exposure levels that are 100 times safer than the exposure level regulators consider safe. Under the 1994 pesticide law, EPA is now applying a standard that is 1,000 times safer than a "no adverse effect level" for many pesticides. In addition, EPA exposure assumptions often mean that the agency estimates risks that are tens of — and sometimes *hundreds* of — thousands of times higher than actual risks.⁵⁴

Consider the substance that Dr. Philip Landrigan of the Mount Sinai School of Medicine in New York says is "as bad as it gets" in terms of health risks. EPA employs absurdly excessive exposure assumptions when setting a standard for malathion. According to the EPA risk assessment on malathion, regulatory standards ensure that a three-year-old toddler could stand for 20 minutes in a cloud of malathion that remains at the full, legally allowed concentration level as released from a fogger truck.⁵⁵ No one expects such high exposure levels to occur in real life situations, but regulators assume them when setting standards to keep risks extremely low. Because of such overly cautious assumptions about exposure and built in safety factors, low-level exposures of registered pesticides don't reach anywhere near levels of concern when used properly.

As a result, malathion is hardly the environmental enemy that activists make it out to be. Keith Solomon, a toxicology expert at the University of Guelph in Canada, says that malathion is not particularly toxic to humans because our bodies can break it down easily. "It's less toxic than table salt," he told a Canadian publication. Solomon notes that malathion also breaks down quickly in the environment. "It doesn't turn into anything nasty,"⁵⁶ he says.

According to the ATSDR:

⁵³ International Agency for Research on Cancer, *World Cancer Report*, 321.

⁵⁴ For some examples see: Frank Cross, "Dangerous Compromises of the Food Quality Protection Act," *Washington University Law Quarterly* 75, no. 1155 (1997).

⁵⁵ U.S. Environmental Protection Agency, Malathion: Revised Risk Assessments (released 11/9/00), Occupational and Residential Exposure and Risk Assessment for the Reregistration and Eligibility Document (RED), September 14, 2000, 74, <http://www.epa.gov/pesticides/op/malathion.htm>.

⁵⁶ Carmela Fragomeni, "The Buzz about West Nile," *The Hamilton Spectator*, August 31, 2002, A10.

“There is no conclusive proof that malathion causes cancer in humans, although some studies have found increased incidence of some cancers in people who are regularly exposed to pesticides, such as farmers and pesticide applicators. Animal studies also fail to provide conclusive evidence of carcinogenicity. The International Agency for Research on Cancer (IARC) has determined that malathion is unclassifiable as to carcinogenicity to humans.”⁵⁷

The most damning information on malathion is that it causes cancer and other problems in rodents exposed to very high levels. ATSDR says that “birth defects have not been observed in humans exposed to malathion,” but that animal studies find “developmental effects” in the offspring of animals that ingested enough malathion while pregnant to cause health effects in the mother.” As noted earlier, the relevance of such studies to humans is highly tenuous, particularly considering exposure levels. It’s the dose that makes the poison. The same type of experiments also cause tumor formation in rodents when they are given massive amounts of the chemicals found in coffee, broccoli, carrots, lettuce, and numerous other safe and healthy substances.⁵⁸ Human exposure to malathion during spraying is too minimal to be dangerous as mosquito-control applicators typically only release two to three and a half ounces of the substance per acre.⁵⁹

Another category of pesticides approved for public health purposes by EPA are pyrethroids. Again, activists claim that EPA standards aren’t enough. Yet after extensive testing and an approval process that can take decades, the EPA and the ATSDR find that this group of chemicals is not particularly toxic to humans.⁶⁰ According to these agencies, these chemicals are not human carcinogens. The ASDR notes:

“There is no evidence that pyrethrins or pyrethroids cause birth defects in humans or affect the ability of humans to produce children. There is no proof that pyrethrins or pyrethroids cause cancer in people.”⁶¹

Problems only result from misuse. Large amounts can cause sickness, dizziness, and even in severe cases, coma. However, adverse reactions are possible from overexposure to a variety of products found in our homes, ranging from bleach to natural gas to aspirin. We know from experience with these products that proper use is relatively safe and risks increase with exposure. Like malathion, the sprayers typically only apply two to three-and-a-half ounces of pyrethroids per acre,⁶² and the substance quickly breaks down and basically disappears within a few days.

⁵⁷ Agency for Toxic Substances and Disease Registry, “ToxFAQs: Malathion,” September 2001, <http://www.atsdr.cdc.gov/tfacts154.html>.

⁵⁸ Committee on Comparative Toxicity of Naturally Occurring Carcinogens, National Research Council, *Carcinogens and Anticarcinogens in the Human Diet: A Comparison of Naturally Occurring and Synthetic Substances* (Washington, D.C.: National Academy Press, 1999).

⁵⁹ U.S. Environmental Protection Agency, “For Your Information: Synthetic Pyrethroids For Mosquito Control.

⁶⁰ Ibid.

⁶¹ Agency for Toxic Substances and Disease Registry, “ToxFAQs: Pyrethrins and Pyrethroids,” September 2001, <http://www.atsdr.cdc.gov/tfacts155.html>.

⁶² U.S. Environmental Protection Agency, “For Your Information: Synthetic Pyrethroids For Mosquito Control.”

Mosquito Control Worse than West Nile?

West Nile's Toll

While exaggerating public health risks of pesticides, environmental activists also claim that vector control should stop because West Nile is not a serious risk. One has even suggested that government reporting of West Nile cases reflected some form of conspiracy to make things appear worse than reality. Others have made comments that appear insensitive to those who have suffered from the disease's debilitating and sometimes deadly effects. For example:

"You have a 1 in 300,000 chance of getting sick, according to the sprayers, if you are bitten by a West Nile Virus-Carrying Mosquito!"⁶³

"A person has a much better chance of winning the lottery" than of getting West Nile.⁶⁴

"These diseases only kill the old and people whose health is already poor."⁶⁵

West Nile is not serious because it only killed seven people in 1999 one activist told the Ottawa Citizen in 2000. More people die of the flu, he says.⁶⁶

"Seven elderly people supposedly died from the virus; yet to date none of the names or medical histories of the deceased have been released nor have any human or bird blood and tissue samples been made available to independent scientists for testing and confirmation ... Officials are afraid to tell the truth because the possibility exists that there were no WNV deaths."⁶⁷

"...And the news is — pesticides pose a much greater health hazard than the West Nile virus ... Since 1999 only a handful of deaths per year have been associated with West Nile, even though the virus has been found in 33 states ... The fact that this 'health crisis' has been exaggerated, and that chemical spraying is usually the least effective yet most toxic way to control mosquitoes, has deterred some state officials, but not others."⁶⁸

These groups are correct when they point out that more people die of the flu. They seem to be suggesting that the flu isn't so bad, and hence West Nile should be of little concern because its death toll is lower. But most people do not realize that the flu is a serious problem, killing up to 36,000 Americans a year.⁶⁹ No one would suggest that we don't try to manage that risk by

⁶³ Posted on the NoSpray.org website: <http://www.nospray.org/WNVodds.html>.

⁶⁴ William Stoner, "Education, Not Spray Will Defeat Virus" in Letters to the Editor, *Buffalo News*, August 12, 2000. Mr. Stoner is listed as program coordinator for Citizens Campaign for the Environment.

⁶⁵ New York City Green Party, "Stop Spraying NY City With Pesticides." This quote was available on the web on September 12, 2000 at <http://www.greens.org/ny/tohtml.cgi?stop-spraying/home.htm>. It is no longer available at that address; a hard copy is available by contacting the author of this paper.

⁶⁶ Paraphrased by Laura Landonl, "Virus Fears Grip New York," *Ottawa Citizen*, August 7, 2000.

⁶⁷ Attributed to the No Spray Coalition, "Stop the Spraying! A Letter from the No Spray Coalition," posted at <http://www.garynull.com/issues/Pesticides/No%20SprayLawsuit.htm>.

⁶⁸ Lynn Landes, "Blowing the Whistle on West Nile — Shades of 1950's and DDT," August 12, 2002, CommonDreams.org, <http://www.commondreams.org/views02/0812-06.htm>.

⁶⁹ Centers for Disease Control and Prevention, "Influenza: The Disease," CDC notation: "This page last reviewed January 21, 2003," <http://www.cdc.gov/ncidod/diseases/flu/fluinfo.htm>

encouraging flu shots and/or taking other precautions. But the seriousness of the flu does not mean we should fail to manage other risks.

Radical environmentalist comments on this topic may have played better before 2002. When they began their crusade against vector control in 1999, there were 62 cases of West Nile in New York State and 7 deaths.⁷⁰ During 2000 (21 illnesses, two deaths)⁷¹ and 2001 (9 deaths and 66 cases)⁷² the impact remained modest.

However, the numbers alone were not the only reason for concern. The unpredictable nature of such viruses raised concerns about future impacts. Jorge Benach of the Center of Infectious Diseases at the State University of New York at Stonybrook noted in a September 2000 op-ed article that “West Nile virus is already a major health threat on Long Island with scores of virus samples taken from mosquitoes and dead birds.”⁷³ Benach highlighted that the “dynamics of this epidemic remain a mystery.” Indeed, 1999 was the first time this virus crossed our borders. We did not know exactly what to expect given that there are various strains of the virus, and the impact varies by region and population. Hence, the risk level was not as clear as environmental activists claimed, which is why Benach noted that cautious use of pesticides was wise. Benach noted further: “The epidemic of the West Nile virus illustrates the extreme vulnerability of our region to acquire and maintain new infectious diseases.” Long Island, he notes, has problems with Lyme disease, Rocky Mountain spotted fever, babesiosis, and ehrlichiosis. Cautious use of pesticides and increased research in these areas will help reduce risks.

Benach’s point about Long Island gets to the heart of what concerns most experts in the field of medical entomology. The emergence of the West Nile virus in the United States is part of a larger phenomenon related to the growth of infectious diseases and increasing difficulties controlling them. “There are many other similar mosquito-borne viruses which could just as easily be introduced into the USA, and some are a lot worse than West Nile,” explained World Health Organization advisor Dr. Norman Gratz at a meeting in Fairfax, Virginia in January 2002.⁷⁴ Indeed, insect borne diseases have presented some of the greatest public health threats known to man and continue to kill millions every year around the world. U.S. citizens are the beneficiaries of vector-control efforts, which help protect us from such diseases.

A New England Journal of Medicine article recently noted such realities:

“Insect-transmitted disease remains a major source of illness and death worldwide. Mosquitoes alone transmit disease to more than 700 million persons annually. Malaria kills 3 million persons each year, including 1 child every 30 seconds. Although insect-borne diseases currently represent a greater health problem in tropical and subtropical climates, no part of the world is immune to their

⁷⁰ “Human West Nile Virus Surveillance — Connecticut, New Jersey, and New York 2000,” *Morbidity and Mortality Weekly Report* 20, no. 4 (April 13, 2001): 265-268, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5014a3.htm>

⁷¹ *Ibid.*

⁷² “West Nile Activity — United States, 2001,” *Morbidity and Mortality Weekly Report* 51 no. 23 (June 14, 2002): 497-501.

⁷³ Jorge Benach, “LI Is a Mystery Magnet for Exotic Diseases,” *Newsday*, September 9, 2000, A43.

⁷⁴ Anita Manning, “West Nile Virus Could Reach Rockies This Year,” *USA Today*, January 8, 2002, <http://www.usatoday.com/news/health/westnile/2002-01-08-usat-rockies.htm>

risks. In the United States, arboviruses transmitted by mosquitoes continue to cause sporadic outbreaks of eastern equine encephalitis, western equine encephalitis, St. Louis encephalitis, and La Crosse encephalitis.”⁷⁵

Starting in 2002, West Nile took a disturbing turn. The CDC reported that 4,156 people became ill and 300 died.⁷⁶ The CDC’s tally for cases in 2003 thus far reports 8,694 cases and 206 deaths.⁷⁷ More than 2,200 of these were reported to be West Nile meningitis or encephalitis, which is a particularly painful and potentially debilitating form of the disease. In addition, this strain of West Nile has been transferred in unexpected ways: through blood and organ donations,⁷⁸ in laboratories during tests on birds,⁷⁹ possibly through breast milk,⁸⁰ and to a baby during pregnancy.⁸¹ And in some cases, victims come down with debilitating illnesses that resemble polio.⁸²

While it is true that the most vulnerable victims of West Nile are the elderly and infirm, younger individuals have suffered as well. During 2001, the median age was 68 with an age range of 9 to 90 and a median age among fatal cases of 70 years.⁸³ During 2002, the median age for the illness was 48, with a range of 1 to 93.⁸⁴ For 2003, the median age is currently 47 years with a range of 1 month to 99 years old.⁸⁵

These cases can hardly be considered trivial, particularly given that the extent of the outbreak has exceeded expectations. All other recorded West Nile outbreaks around the world

⁷⁵ M.S. Fradin J. F. Day, “Comparative Efficacy of Insect Repellents Against Mosquito Bites,” *New England Journal of Medicine* 347, no. 1 (August 5 2002): 13-8.

⁷⁶ U.S. Centers for Disease Control and Prevention, “West Nile Virus 2002 Case Count,” posted online, <http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount02.htm>.

⁷⁷ U.S. Centers for Disease Control and Prevention, “West Nile Virus 2003 Human Cases as of December 3, 2003,” posted online, <http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount03.htm>.

⁷⁸ “Detection of West Nile Virus in Blood Donations — United States, 2003” *Morbidity and Mortality Weekly Report* 52, no. 32 (August 15, 2003): 769, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5232a3.htm>; “Public Health Dispatch: Investigations of West Nile Virus Infections in Recipients of Blood Transfusions,” *Morbidity and Mortality Weekly Report* 51, no. 43 (November 1, 2002): 973-974,

<http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5143a5.htm>; Martha Iwamoto, M.D., M.P.H et al., “Transmission of West Nile Virus from an Organ Donor to Four Transplant Recipients,” *The New England Journal of Medicine* 348, no. 22 (May 29, 2003): 2196-2203, <http://content.nejm.org/cgi/content/short/348/22/2196>.

⁷⁹ “Laboratory-Acquired West Nile Virus Infections — United States, 2002,” *Morbidity and Mortality Weekly Report*, 51, no. 50, (December 20, 2002): 1133-1135, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5150a2.htm>.

⁸⁰ “Possible West Nile Virus Transmission to an Infant Through Breast-Feeding — Michigan, 2002,” *Morbidity and Mortality Weekly Report* 51, no. 39, (October 4, 2002): 877-878, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5139a1.htm>.

⁸¹ “Intrauterine West Nile Virus Infection — New York, 2002,” *Morbidity and Mortality Weekly Report* 51, no. 52 (December 20, 2002): 1135-1136, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5150a3.htm>.

⁸² “Acute Flaccid Paralysis Syndrome Associated with West Nile Virus Infection — Mississippi and Louisiana, July-August 2002,” *Morbidity and Mortality Weekly Report* 51, no. 37, September 20, 2002): 825-828.

⁸³ “West Nile Activity — United States, 2001,” *Morbidity and Mortality Weekly Report* 51, no. 23 (June 14, 2002): 497-501.

⁸⁴ “Provisional Surveillance Summary of the West Nile Virus Epidemic — United States, January-November 2002,” *Morbidity and Mortality Weekly Report* 50, no. 51 (December 20, 2002): 1129-1133, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5150a1.htm>

⁸⁵ “West Nile Virus Activity — United States, October 30-November 5, 2003,” *Morbidity and Mortality Weekly Report* 52, no. 44 (November 7, 2003): 1080, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5244a7.htm>.

have been substantially smaller: e.g., Romania in 1996 reported 393 illnesses and 17 deaths⁸⁶ and Russia in 1999 reported 500 cases and 40 deaths.⁸⁷ The unexpected high rates of West Nile in the United States show that it was reasonable for medical entomologists to be concerned as far back as 1999 about the unpredictable nature of such diseases.

Documented Cases of Pesticide Health Effects

As a point of comparison, we can consider the CDC data on documented cases of health problems related to pesticide exposures from spraying during 1999-2002. If spraying-related health problems are as rampant as environmental activists suggest, we should expect some significant documentation of cases. But the CDC data indicate that the number of cases were very small and the impact only temporary. According to the CDC report, there were two cases of definite health impacts, 25 probable cases, and 106 possible cases. No deaths were reported. That's a total of 133 potential cases of temporary illness over four years among a population that CDC estimates was 118 million in 2000. Despite what environmental activists might claim, that's a pretty impressive record of success.

CDC points out:

“The findings in this report indicate that serious adverse outcomes potentially related to public health insecticide application were uncommon. When administered properly, in a mosquito-control program, insecticides pose a low risk for acute, temporary health effects.”⁸⁸

It is true that CDC data doesn't include potential cancers — which in theory would occur decades from now. But as documented earlier, exposures are too short and too low to produce any significant risks in that category. Activists might also point out that the number of cases documented doesn't represent all such cases. That is correct, but it is also true for both West Nile cases and pesticide exposures. Each sample provides some indication of the scope of each problem — and the data show a much more significant effect from West Nile despite activist claims to the contrary.

Of course, short-term health effects should be controlled and long-term risks kept minimal. The fact that CDC could find relatively few cases of pesticide exposure related problems indicates that vector-control operations have done a remarkable job keeping risks low. Consider the breakdown and circumstances of the pesticide-exposure cases.

There were 133 cases of potential “illnesses,”⁸⁹ but actually, some of these are not what most people would classify as illnesses. Instead, some (15) were individuals who simply called the poison control center for advice and then pursued no additional assistance. Eighty five of the

⁸⁶ New York Academy of Sciences, “International Cases of West Nile Virus Provide Blueprint for Understanding U.S. Outbreak,” Media Alert, August 13, 2002, http://www.nyas.org/press/pr_020813.html.

⁸⁷ Ibid.

⁸⁸ “Surveillance for Acute Insecticide-Related Illness Associated with Mosquito-Control Efforts — Nine States, 1999-2002,” *Morbidity and Mortality Weekly Report* 52, no. 27 (July 11, 2003): 629, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5227a1.htm>.

⁸⁹ Ibid.

people sought medical advice, and one was hospitalized. Of note, a significant number (36 or 27 percent) were work-related exposures. Hence only 96 cases involved public exposures.

Twenty-nine (about 30 percent) of the public exposures resulted from a single event when a spray truck accidentally sprayed near a park where kids were playing baseball. None of those cases appear to have proven significant, and it may well be that fear of the pesticides (not surprising given the hype by activists) was the more serious contributor to health concerns. CDC notes that while the pesticides can cause temporary respiratory symptoms (such as aggravation of asthma) or neurological impairment (causing dizziness), “anxiety about insecticide use for mosquito control also might have been responsible for symptoms in some persons.”⁹⁰

In four years of spraying, there is only one case that could be considered severe, and that case was related to asthma and chronic obstructive pulmonary disease (COPD). CDC explained the circumstances as: “When her neighborhood was sprayed, a woman aged 54 years was exposed to sumithrin, which passed through operating window fans and a window air conditioner. She had exacerbation of her asthma and chronic obstructive pulmonary disease.” Window fans suck in air from the outdoors, which is something that a COPD patient probably should avoid. Individuals with COPD generally have to be very careful because even minute amounts of substances can create problems. Dust, pollen, and air fresheners can produce breathing problems. Fortunately, this individual recovered. Her case highlights special precautions that COPD patients must take to reduce exposure to a host of substances, both natural and man-made, but it does not justify inadequate protection of the public from vector-related risks. In fact, COPD patients would be particularly vulnerable should they be struck with West Nile, which could easily kill someone with a compromised respiratory system.

Nonetheless, environmental activists may use this case to suggest that many people, particularly children with asthma, are vulnerable to pesticide spraying. As we use more chemicals and produce more pollution, they note, asthma cases have grown. But the rise in asthma is inversely related to pollutants in the air. Cases have risen as pollutants in the air have declined. There can be no cause and effect. There are other more likely explanations. According to one study: “Allergens associated with dust mites (DM) and cockroaches (CR) are probably important in both onset and worsening of asthma symptoms for children who are chronically exposed to these agents. Young children spend a great deal of time on or near the floor where these allergens are concentrated in dust.”⁹¹ In that case, use of in-home pesticides — not reduced pesticide use — may be a big part of the solution. In addition, as fewer and fewer buildings have windows that open, indoor air quality declines.⁹² Better ventilation may be in order.

⁹⁰ Ibid.

⁹¹ Floyd J. Malveaux and Sheryl A. Fletcher-Vincent, “Environmental Factors of Childhood Asthma in Urban Centers,” *Environmental Health Perspectives* 103, Supplement 6 (September 1995): 59; D. L. Rosenstreich et al., “The Role of Cockroach Allergy and Exposure to Cockroach Allergen in Causing Morbidity Among Inner-City Children with Asthma,” *New England Journal of Medicine* 336, no. 19 (May 8, 1997): 1356-1363.

⁹² Thomas A.E. Platts-Mills et al., “Asthma And Indoor Exposure To Allergens,” *The New England Journal of Medicine* 336, no. 19 (May 8, 1997): 1384.

Wildlife Impact Claims

Environmental activists have also made a host of claims about the impact of vector-control efforts on wildlife. Spraying and use of chemicals can have impacts, but environmental organizations have tended to grossly exaggerate such impacts, and in some cases they seem to wrongly attribute wildlife deaths and disease to chemicals. In the past, it appears that some chemicals have had adverse impacts on wildlife, such as in the DDT case noted earlier. Fortunately, changes have greatly minimized impacts, and there are few impacts observed from pesticide spraying in particular.

Various states have programs to track wildlife diseases and pesticide-caused health effects. Since the West Nile virus emerged, states have stepped up screening of birds in particular to track the progression of disease and risks to communities. These efforts have increased knowledge of wildlife disease, but the impact of pesticides is less clear.

Despite the lack of evidence of widespread pesticide problems, environmental groups have claimed that unequivocal evidence exists that spraying and other pesticide use is harming wildlife. In particular, they have focused on impacts to birds and aquatic life (mostly pointing to a lobster die-off in the Long Island Sound). In addition, they make general, yet unsupported, statements that pesticides “devastate” non-target species such as butterflies and bees, while failing to kill mosquitoes. However, spraying mainly kills bugs that are in flight within the area sprayed, making the most likely targets the mosquitoes and other insects that are active at night when spraying occurs. Exposure is limited in regard to butterflies, bees, and other insects that are primarily out during the day.

Birds

Environmental activists claim there is clear evidence that pesticides are a far greater risk to birds than West Nile. They claimed back in 2001 that data from New York State showed that more birds were dying from “toxins” like pesticides than from West Nile. But science writer Steven Milloy obtained New York State data in 2001 that showed the toxins that affected the birds in this sample were mostly naturally occurring.

According to Milloy, the New York State data that he obtained on 3,216 dead birds found that natural diseases and toxins caused the majority of the bird deaths (1,263 from West Nile virus and 1,100 from botulinum). Meanwhile, the data included 219 pesticide-related bird deaths, of which 30 were from intentional poisonings of pest birds and 100 were from illegal use of pesticides for intentional killing of birds. Twenty-seven bird deaths resulted from lawn care products.⁹³

More recently, the Audubon Society says that data collected by New York State in subsequent years from a sample of 80,000 dead birds shows that pesticides, primarily lawn

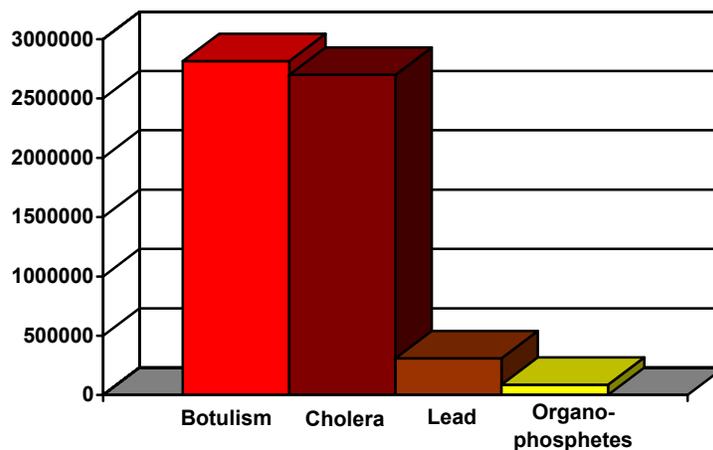
⁹³ Steve Milloy, “Audubon’s Fly-By-Night Pesticide Campaign,” Fox News, July 8, 2001.

products, are killing the majority of birds.⁹⁴ If true, that would be a very significant finding, which should be documented in some peer-reviewed format.

Yet New York State has not released the data in any report, nor has anything been peer reviewed. The “majority” of such toxin-related deaths may again include natural toxins, like botulinum. In addition, it is unlikely that New York State conducted studies on all 80,000 birds collected; the number actually analyzed was likely much smaller. In fact, New York State’s pathologist in charge of this research, Dr. Ward Stone, is reported as stating that only a few thousand of the sample collected were studied one year.⁹⁵ Press quotes of Dr. Stone directly indicate that he is concerned about pesticide risks to wildlife, but he has not offered any official quantification of pesticide impacts.

The U.S. Geological Survey (USGS), a division of the Department of Interior, which conducts extensive research on wildlife disease, could not confirm the New York State data.⁹⁶ USGS has been collecting data and studying wildlife diseases for decades.⁹⁷ USGS researchers study pesticide-related deaths among wildlife, focusing on pesticides only when an illegal use or over application is reported. USGS does an impressive job reporting extensive deaths related to natural diseases, but reports seem to cover birds found mostly on public lands. It shows that many mass die-offs of species occur from natural causes. Far fewer incidents are related to illegal and/or excessive use of pesticides. In particular botulism epidemics kill thousands of birds, as does West Nile. Unfortunately, they have not collected data on dead birds found in residential areas, which would help determine whether residential exposure to pesticides has a significant impact on wildlife.

25 Years of USGS Data on Bird Mortality — Incidents/Epidemics



⁹⁴Audubon, New York, “Lawn Pesticides Implicated In Bird Deaths,” Press Release, January 16, 2002, <http://ny.audubon.org/news/020116.htm>; see also: “Pesticide Is Leading Cause of Bird Deaths in New York, Not West Nile Virus: Audubon Calls on Other States to Test for Pesticides, Release Data,” PR Newswire, June 21, 2001.

⁹⁵ For example, Stone noted that in 2002 the state conducted studies on only 5,000 out of 7,000 of the birds collected that year.

⁹⁶ Personal communications on July 28, 2003.

⁹⁷ USGS has posted 25 years of their data online at <http://www.nationalatlas.gov/wildmortfrm.html>.

In addition, other states also test birds for disease and pesticide poisoning, and they have not reported findings similar to those that Audubon claims for New York. Michigan's *Wildlife Disease Manual*,⁹⁸ has two entries on pesticides. Both note that state officials rarely find pesticide poisonings. In contrast, they provide a lengthy list of mortalities from natural diseases. The pesticide entries state:

“Occasional organophosphate poisonings are seen in Michigan wildlife following exposure to recently treated areas. Diazinon intoxication in Canada geese, mallard ducks and wild turkeys is the most common organophosphate poisoning seen. Parathion poisoning in ring-billed gulls and disulfoton intoxication in a mallard, sevin poisoning in bees, and chlorpyrifos poisoning in a mallard have occurred as the use of organophosphates has increased. These deaths are usually sporadic and infrequent in occurrence.”⁹⁹

“Mortality in wildlife due to chlorinated hydrocarbon poisoning is seldom observed in Michigan anymore. Due to the banning of many of the highly toxic chlorinated hydrocarbon compounds in the 1970's, the possibility of exposure today is rare. The importance of these compounds to humans is comparable.”¹⁰⁰

Despite Audubon's suggestions to the contrary, the data on bird deaths from all sources isn't particularly clear. Dr. Stone (who reportedly gave the data to the Audubon Society) has told the press that he doubts spraying will do much harm to birds — at least not as much as does the virus.¹⁰¹ The EPA asserts that spraying poses a negligible risk to birds.

While claims about rampant pesticide deaths are not supported, the impact of the West Nile virus is well recognized and documented as a serious threat to wildlife. “That's [West Nile virus] a huge concern” Emi Saito of the USGS told *USA Today*. “If it's attacking our endangered species, is it going to lead to their extinction?”¹⁰²

Because of such concerns, the USGS, the U.S. Department of Agriculture, and the CDC, have stepped up efforts to study the impact of the West Nile virus on birds. A press release explains their concerns:

“Surveillance activities, public observations, and preliminary analysis of population survey data suggest that WNV has caused extensive mortality in many avian species, particularly corvids (crows) and raptors (hawks). Scientists are worried the virus could devastate flocks of threatened and endangered species, migratory birds, and other wildlife. Additionally, wild horse and burro populations are at high risk.”¹⁰³

⁹⁸ State of Michigan, Department of Natural Resources, “Organophosphate Toxicity,” *Michigan Wildlife Disease Manual*, online at: http://www.michigan.gov/dnr/1,1607,7-153-10370_12150_12220---,00.html.

⁹⁹ State of Michigan, Department of Natural Resources, “Organophosphate Toxicity,” *Michigan Wildlife Disease Manual*, online at: http://www.michigan.gov/dnr/1,1607,7-153-10370_12150_12220---,00.html.

¹⁰⁰ *Ibid.*

¹⁰¹ Lucy Chubb, “Northeast States Spray Toxic Pesticide to Rid Areas of West Nile Virus,” *Knight-Ridder Tribune Business News*, August 17, 2000; Mark Weiner, “Some Fear Spraying More Than West Nile Not All Counties Use Pesticides,” *Herald American*, August 20, 2000, A1.

¹⁰² “West Nile Virus Striking Down Bird Population Across the USA,” *USA Today*, September 9, 2002.

¹⁰³ USGS, Federal Scientists Refocus on West Nile Virus Impacts on Wildlife, Press Release, February 4, 2003, http://www.usgs.gov/public/press/public_affairs/press_releases/pr1720m.html.

USGS reports that West Nile has killed birds from at least 160 bird species, including some endangered species.¹⁰⁴ Last year, the Milwaukee Zoo lost a dozen penguins because of West Nile. “In the State of Michigan last year [in a] twelve to fourteen day period in September and October, every snowy owl in captivity died from West Nile,” Lake Superior Zoo Director Mike Janis told Minnesota Public Radio.¹⁰⁵ Pat Redig, Director of the University of Minnesota’s Raptor Center, noted on the Minnesota Public Radio report: “We had about 59 confirmed cases of it that came through our center here ... Most of them in Great Horned owls. But as we look around this year, this spring, we can’t detect that there’s been a measurable impact on populations of birds at this stage of the game.”¹⁰⁶

Mitchell Byrd, of the Center for Conservation Biology at the College of William & Mary, told the Associated Press that West Nile “has the potential to be disastrous” for birds. Wildlife officials report that they are getting increasing reports of sick birds, with birds of prey being affected in increasing numbers. In Virginia, three peregrine falcons, which are listed as endangered species, were recently reported dead from West Nile. “We’ve only got 19 pairs in Virginia. We can’t afford to lose very many of them,” Byrd told the Associated Press.¹⁰⁷

The *Bird Watcher’s Digest* reported in 2002 that 400 great horned owls were found dead in the Midwest from West Nile during 2002.¹⁰⁸ Researchers estimate that for each dead bird reported, there are probably 100 to 1,000 unreported cases, which means there could have been as many as 40,000 to 400,000 great horned owl deaths from West Nile last year alone. Also at risk are red tailed hawks. According to the *Digest*, research centers that collect and rehabilitate injured birds are finding that, at some facilities, as many as two-thirds of the birds collected die from West Nile. Victims include bald eagles, golden eagles, snowy owls, and gyrfalcons.

Zoos have begun using a new vaccine that they hope will help. It was approved recently to protect horses. Horses have been hard hit, with 15,000 cases of equine West Nile virus in 2002 alone.¹⁰⁹ Unfortunately, a vaccine won’t be helpful for protecting wildlife.

Lobsters

In addition to birds, activists also say that aquatic life is at grave risk. When a massive lobster die-off occurred in the Long Island Sound during 1999, lobstermen also cried foul, claiming that New York City’s malathion spraying reached the waters and caused the die-off. “It’s not a coincidence,” said one lobsterman in a *Discover* story on the topic.¹¹⁰ Environmentalists have bolstered the lobstermen’s position by repeatedly asserting that spraying is responsible:

¹⁰⁴ U.S. Geological Survey, National Wildlife Health Center, “West Nile Virus, Frequently Asked Questions,” http://www.nwhc.usgs.gov/research/west_nile/WNV_FAQ.html.

¹⁰⁵ Bob Kelleher, “West Nile at the Zoo,” Minnesota Public Radio, June 24, 2003, http://news.mpr.org/features/2003/06/24_kelleherb_westnilezoos.

¹⁰⁶ Ibid.

¹⁰⁷ “West Nile Hits Birds Hard This Year Across Virginia,” *Washington Times*, September 2, 2003.

¹⁰⁸ Eirik A. T. Bom, “Special Topic: West Nile Virus,” *Bird Watcher’s Digest*, December 2002.

¹⁰⁹ Animal Plant Health Inspection Service, U.S. Department of Agriculture, “Questions and Answers about the West Nile Virus,” March 2003, http://www.aphis.usda.gov/lpa/pubs/fsheet_faq_notice/faq_ahwnv.html.

¹¹⁰ Gene Santoro, “Silent Summer,” *Discover*, July 2000, 76.

“... these pesticides are known to severely impact many aquatic species and nontarget insects. There is a pending lawsuit regarding the impact of these pesticides on the widespread die-off of crabs and lobsters in Long Island Sound ... Thousands of fish, lobsters, birds and beneficial insects like butterflies and bees were killed by the spraying. Our waterways were polluted. Even the Connecticut Sea Grant (Sea Grant is a Federal Agency which sponsors regional projects on coastal marine problems usually tied with industry) notes with alarm that pesticide spraying is implicated in the lobster die-off. Repeated spraying has severely impacted vital ecosystems, and the offspring of mosquitoes that survived the spray are likely to now be growing increasingly resistant to the pesticides applied.”¹¹¹

In addition to spraying on Long Island, others have picked up on the issue. A group called “Save the Bay” in Rhode Island suggested that the state should stop using larvicides because they feared they might be contributing to a shell disease among lobsters in that state. One state official responded that the claim amounted to “the most wild and speculative association that you could come up with,”¹¹² but that doesn’t prevent environmental groups from using such tactics.

Lobstermen clearly have a stake in pinning the issue on pesticides. So far, they have been able to leverage their hard times to get \$7.3 million dollars in federal relief funds. In addition, they are trying to collect on lawsuits filed against pesticide manufacturers. They have even filed a class action lawsuit.

The federal government responded to the problem by funding lobster research at universities in Connecticut and New York as part of the Long Island Lobster Research Initiative.¹¹³ Early on, the research pointed more strongly to Mother Nature as the cause than to the spraying. The University of Connecticut’s Dr. Richard French explained in 2000: “There is no definitive evidence that pesticides are causing a problem ... The pathology is showing evidence of a parasite.”¹¹⁴ In 2000, he offered additional findings at a symposium on the issue on Long Island. French and his colleagues found: “There is no quantitative evidence of pesticide toxicity ... All the indications based on pathological evaluation of the American Lobster in LIS [Long Island Sound], suggest that the mass mortality of lobsters in 1999 was the effect of a natural disease.”¹¹⁵

Research has continued to show that a combination of natural factors likely played important roles in the die-off. In particular, Long Island represents some of the farthest southern reaches for these lobsters, and unusually warm waters in years leading up to and beyond 1999 seem to have created natural environmental stresses that made the shellfish more susceptible to the parasites. Alistair Dove, of the New York University at Stonybrook, says that his research

¹¹¹ Posted on the Web page of Hudson River Sloop Clearwater, Inc. Poughkeepsie, New York, <http://www.clearwater.org/news/nospray.html>, also posted on www.nospray.org.

¹¹² Peter B. Lord, “Public Health vs. Environmental Concern — War on West Nile at Issue,” *Providence Journal*, May 17, 2001.

¹¹³ See: <http://www.seagrant.sunysb.edu/LILobsters/default.htm>.

¹¹⁴ Telephone conversation on September 6, 2000.

¹¹⁵ Richard French et al, “Long Island Sound Lobster Health Symposium,” *Second Annual Long Island Sound Lobster Health Symposium*, Background Material, November 2001.

strongly indicates that warm water was indeed the problem. “Climate is the killer here,” he said.¹¹⁶

More recently, the Research Initiative reported last spring:

“The year 1999 was characterized by very warm winter and fall temperatures and above average temperatures in July and August. Strong winds forced mixing of the water column in late August 1999, causing unusually warm surface waters to be mixed to the bottom, further elevating the bottom water temperature. Historical records of bottom water temperatures indicate that temperature in 2001 were within the historical averages. However, the bottom water temperatures in 2002 were higher than historical averages.”¹¹⁷

In addition to warm water, oxygen levels near the sea floor have been unusually low and other naturally occurring chemicals were high:

“More sensitive measurements show that hypoxic events [low oxygen] persisted in western LIS [Long Island Sound] in summer 2000 for much longer periods than believed. There were increases in the amounts of sulfide and ammonia during August to November 2000. This indicates that anaerobic processes (such as decomposition) were dominating over normally aerobic processes in the near-surface sediments. Research is ongoing to study the effects on lobster health of elevated ammonia, hydrogen sulfide and chlorinated hydrocarbons in water and sediment.”¹¹⁸

Since lobsters spend most of their lives in the bottom of water bodies, these changes were likely to create serious stresses and make them more sensitive to natural diseases. In addition to the original parasite that was discovered by Dr. French and his colleagues, some lobsters have also developed a shell disease in recent years.¹¹⁹

Hence, the research provides considerable evidence that lobster deaths are attributable to natural pathogens and some unfortunate climatic circumstances. The only issue is whether pesticides were yet another environmental factor that contributed to weakening of lobster immune systems. Laboratory evidence does indicate that lobsters are highly sensitive to a wide variety of agents, including pesticides. Research shows that malathion proved toxic to lobsters at levels as low as 33 parts per billion when the lobsters were exposed for 96 hours under laboratory conditions. Yet researchers admit that laboratory conditions are not analogous to exposure in the moving waters of the Long Island Sound. In addition, malathion was found to degrade very rapidly in both the environment and within the lobsters in the laboratory experiments. Studies show that 65 to 77 percent of the pesticide degraded within the first 24 hours and 98 percent within three days or 36 hours — a threshold lower than the 96 exposure

¹¹⁶ “Scientist Link Dying Lobsters to Warmer Long Island Waters,” Associated Press, November 9, 2002.

¹¹⁷ “Bottom Water Conditions Can Create Problems for Lobsters in Long Island Sound,” *Long Island Sound Lobster Health News* 1, no. 1, (Spring 2003), <http://www.seagrant.sunysb.edu/LILobsters/LHN-Spr03.pdf>.

¹¹⁸ Ibid.

¹¹⁹ “Disease Pathogens in Long Island Sound’s Lobster Populations,” *Long Island Sound Lobster Health News* 1, no. 1, (Spring 2003), <http://www.seagrant.sunysb.edu/LILobsters/LHN-Spr03.pdf>.

hours in the study.¹²⁰ Studies also examined the impacts of a larvicide, suggesting that developing lobsters are sensitive at “various levels.”¹²¹

The question remains whether exposures in the environment reach lobsters at levels that matter. The probability of any significant impact appears remote for a number of reasons: Rapid degradation of these pesticides limits exposure, particularly given that spraying is not that frequent and not directed near waterways. In addition, adult lobsters live mostly near the bottom of the sea, where pesticides are unlikely to reach.

The researchers working on the link themselves note the limitations of their research. Sylvain De Guise, of the University of Connecticut, notes that while his research shows lobsters are very susceptible to pesticides, it cannot show pesticides had an impact in a real-life scenario. He noted to the press: “We looked for pesticides in the water and couldn’t find it. We looked for pesticides in the lobsters and couldn’t find it.”¹²² Moreover, pesticides or no pesticides, it seems very probable that a substantial lobster die-off would have resulted anyway given all the climatic conditions present during these past several years. In fact, the lobster die-off began before pesticides were applied in 1999.

In addition to disease, there may be other factors that have contributed to problems for the lobster industry. Data on lobster landings show unusually high record landings throughout the 1990s. Record catches during those years might reflect robust lobster population or potential overfishing. Some have pointed to overfishing as a probable cause of a weakened industry throughout the region. Rhode Island is experiencing a reduced population of legal-sized lobsters that might be the result of overfishing. “Despite all the warnings that the harvests couldn’t last, everybody just kept on fishing. And now we are in a downward spiral with no end in sight,” said Mark Gibson, marine administrator for the Rhode Island Division of Fish and Wildlife.¹²³ Similar concerns have been raised about lobster fishing in the Long Island Sound during the past decade. The data show that the lobster industry during the 1990s in New York State did indeed catch an unusually large number of lobsters.¹²⁴

In any case, regional lobster problems do not signal the end for the lobster industry at large as environmental activists and lobstermen seem to suggest. New York’s regional problem has not stopped the industry from growing nationally just about every year for decades. According to figures from the National Marine Fisheries Service, the highest national lobster catch occurred in 1999 — the year New York suffered its massive die-off.¹²⁵ The data show that since 1950, the lobster catch has increased through the decades, with some years dipping only to be followed by the continued march upward. Measured on a decade-by-decade basis, the catch for each decade exceeded the last.

¹²⁰ “Toxic Effects of Pesticides on Lobster’s Health in Long Island Sound,” *Long Island Sound Lobster Health News* 1, no. 1, Spring 2003, <http://www.seagrant.sunysb.edu/LILobsters/LHN-Spr03.pdf>

¹²¹ Ibid.

¹²² Louis Porter, “Four Years After Die-Off, Lobsters not Rebounded,” *Stamford Advocate*, September 23, 2003.

¹²³ David Arnold, “Overuse, Disease Hurt N.E. Lobster Industry,” *Boston Globe*, March 11, 2003.

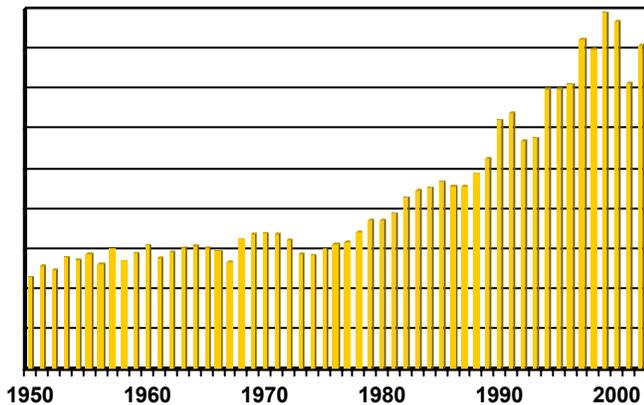
¹²⁴ This data was collected by the National Marine Fisheries Service and is available at: <http://www.st.nmfs.gov/commercial/index.html>.

¹²⁵ Data available at: <http://www.st.nmfs.gov/commercial/index.html>.

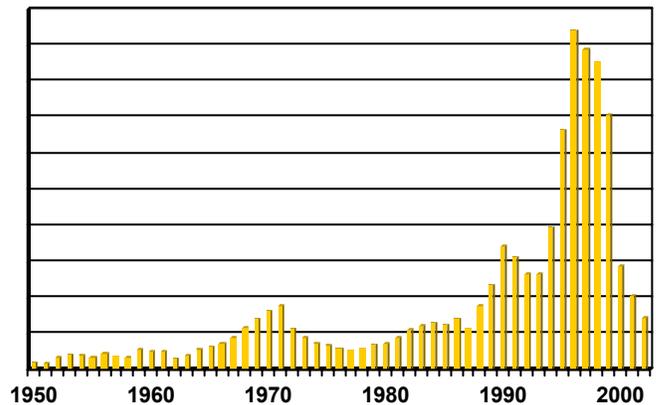
Interestingly, New York’s data has more variability than national data, with a peak catch in 1971 of just fewer than 2 million pounds. The number of lobsters caught dipped in the late 1970s into the 1980s, only to balloon in the 1990s, up to a record of more than 3 million pounds in 1992 and then to its pinnacle of nearly 9.5 million pounds in 1996. Before the 1990s, such high figures must have been unimaginable to New Yorkers given that the average catch for all the years between 1950 to 1989 totals less than a million pounds a year.

Hence, 1999 ended a decade that was largely an aberration for New York. Interestingly, the yields for 1999 (7 million pounds) and 2000 (3 million) are still higher than any year before 1990. That does not minimize the economic pains felt by the industry, but the national data indicate that that lobster fishing will survive, even amidst regional problems.

National Lobster Landings 1950-2002
(millions of pounds)



New York State Lobster Landings 1950-2002
(millions of pounds)



Claims About Insect Repellants

In addition to unsupported claims about pesticide spraying, environmental activists have even attacked one of the few things that individuals can do to protect themselves: application of chemical insect repellants, particularly those that contain DEET. In particular, environmental activists say children could suffer from DEET exposure:

“The use of DEET in mosquito repellents is extremely troubling. DEET has been associated with seizures and several cases of toxic encephalopathy (encephalitis) in children, including three deaths, according to the Extension Toxicology Network at Cornell University.”¹²⁶

However, medical literature questions these claims. The several case reports of children suffering seizures or death actually are not conclusive. Researchers recently published a review of these case studies in the *Canadian Medical Association Journal*. They could only find ten case reports of children suffering from seizure possibly related to DEET exposures. They report

¹²⁶ Lynn Landes, “Blowing the Whistle on West Nile — Shades of 1950’s and DDT,” posed at www.nospray.org; The No Spray website notes that this article was also published on August 12, 2002 by CommonDreams.org.

that none of these studies were conclusive that DEET was in fact the cause of seizures. Given that 3 to 5 percent of children suffer from such seizures for a variety of reasons and that 23 to 29 percent of children are exposed to DEET, it is possible that the cases were incorrectly attributed to DEET. “Nonetheless, these case reports have been widely quoted and have led regulatory agencies and pediatric societies to limit use of DEET in young children,” the researchers note.¹²⁷

New England Journal of Medicine published a similar study that addressed the relative effectiveness of various repellants. They found: “DEET-based products provided complete protection for the longest duration. Higher concentrations of DEET provided longer-lasting protection.”¹²⁸ DEET lasted for 301.5 minutes, while a soybean-oil based repellant lasted not even one third as long (94.6 minutes). Another repellant lasted just under 23 minutes (22.9 minutes), and botanical repellants lasted less than 20 minutes. And wristbands containing repellants were ineffective. They also noted that DEET has been in use for decades with few health repercussions. Problems only emerge in cases of in which parents apply massive amounts of DEET on children.

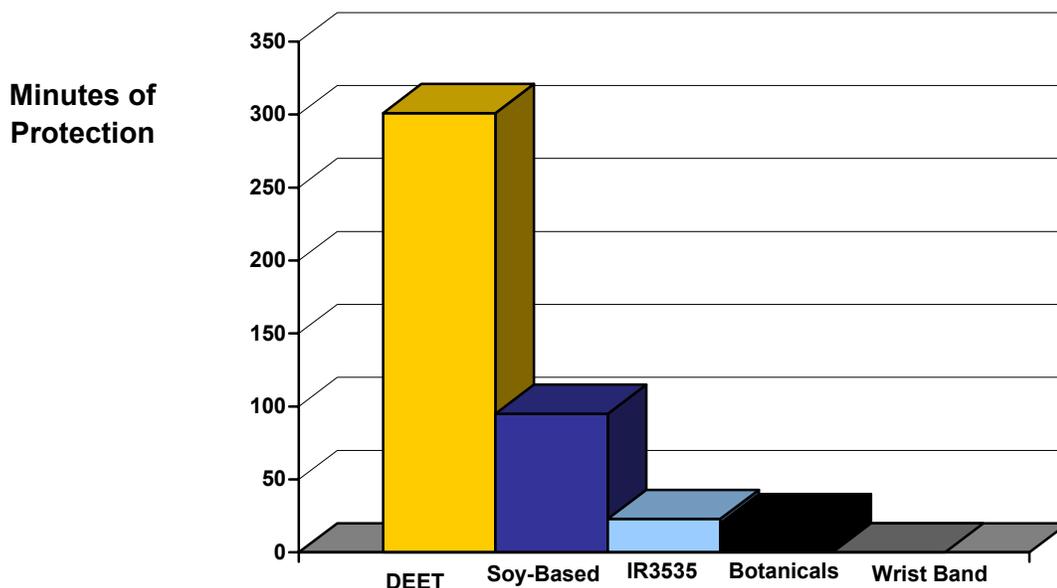
Given an impressive record of safety and effectiveness, these researchers went as far as to label DEET the “gold standard for protection” against insect-borne disease:

“Despite the substantial attention paid by the lay press every year to the safety of DEET, this repellent has been subjected to more scientific and toxicologic scrutiny than any other repellent substance. The extensive accumulated toxicologic data on DEET have been reviewed elsewhere. DEET has a remarkable safety profile after 40 years of use and nearly 8 billion human applications. Fewer than 50 cases of serious toxic effects have been documented in the medical literature since 1960, and three quarters of them resolved without sequelae. Many of these cases of toxic effects involved long-term, heavy, frequent, or whole-body application of DEET. No correlation has been found between the concentration of DEET used and the risk of toxic effects. As part of the Reregistration Eligibility Decision on DEET, released in 1998, the Environmental Protection Agency reviewed the accumulated data on the toxicity of DEET and concluded that ‘normal use of DEET does not present a health concern to the general U.S. population.’ When applied with common sense, DEET-based repellents can be expected to provide a safe as well as a long-lasting repellent effect. Until a better repellent becomes available, DEET-based repellents remain the gold standard of protection under circumstances in which it is crucial to be protected against arthropod bites that might transmit disease.”¹²⁹

¹²⁷ Gideon Koren, Doreen Matsui, and Benoit Bailey, “DEET-Based Insect Repellants: Safety Implications for Children and Pregnant and Lactating Women,” *Canadian Medical Association Journal* 169, no. 3 (August 5, 2003): 209-212.

¹²⁸ M.S. Fradin, and J. F. Day, “Comparative Efficacy of Insect Repellents Against Mosquito Bites,” *New England Journal of Medicine* 347, no. 1 (July 4, 2002): 13-8.

¹²⁹ *Ibid.*



Yet again, environmentalist overstatements and mischaracterization of reality threatens public health. Given DEET's impressive record in protecting against insect-borne disease, efforts to discourage use or suggest that alternatives are sufficient could contribute to increased disease incidence.

Conclusion

The preceding material documents the extensive record of radical environmentalists producing misleading facts about chemical use and pesticides. Unfortunately, this paper only offers a small sampling of the numerous claims they make. The number of claims is beyond the ability of anyone to respond specifically to each of them. However, the simple fact that public health has greatly advanced from, and along with, growing chemical use disputes a vast array of activist claims. Public health officials and citizens would be wise to view any claims from these groups with great skepticism.