

Environmental Health and the Precautionary Principle

By Devra Davis, Ph.D.*

Edited Keynote Talk

Massachusetts Precautionary Principle Project

Kickoff Meeting, May 15, 1999

Framingham State College, Framingham Massachusetts

Many of you are familiar with Plato's parable of a group of blind men in a cave trying to describe an elephant. Each of them confidently depicts a distinct part of the anatomy that they can grasp, but none of them understands how the whole thing fits together. The perception of each is accurate, but none can comprehend the totality. When it comes to figuring out how to make the Precautionary Principle work with respect to the environment, we need to realize that we are basically in a similar situation. We are required to build a coalition that recognizes that the different pieces of the puzzle are not all together and must be provided by persons working on distinct parts of the problem. The basic concept of the Precautionary Principle is simple. Where the state of the health of the people is at risk, preventing that risk from taking place and actually causing harm to human health or the environment is far better than seeking to cure harm after it occurs. What the major components of the Precautionary Principle are and how these fit together should be thought of as a work in progress, requiring independent input from all the relevant players who are trying to depict their part of a large, unknown elephant-like terrain.

Where the part of this puzzle that we can grasp indicates there are significant risks to public health, we should be prepared to take action, even when scientific knowledge is not conclusive and we do not have the complete picture at hand. We should use the best scientific information available, but this does not mean that we should sit back until we have certain evidence about everything. There are many instances of environmental harm that cannot be repaired once they have occurred. This is certainly the case with irreversible brain damage tied with lead poisoning, as well as with climatic changes. For these and a host of other environmental health problems, not only is prevention always cheaper than cure, often no cure exists, nor is one likely to emerge in the foreseeable future.

My remarks today are based in part on an article that my colleagues and I recently published on the Precautionary Principle as applied to breast cancer (Davis et al: 1998a). But what I am going to talk about now does not apply just to breast cancer. It really applies to environmental health, which is a big, complicated puzzle, as many of you know. The fundamental question is how do we change the burden of proof, while we continue to find all the right pieces of the puzzle. Nowadays, regulators often wait for sufficient numbers of complaints to amass from sick people before taking action. What can we do to move the

process so that proof of human harm does not remain the chief requirement for action? Well, I think that there are several components to that. The first is we need to be smarter about using experimental evidence. And whether that evidence comes from a cell culture or from experimental animals or whether it comes from global models of climate change, we must rely on the best models or evidence available. We must use this experimental information to predict and prevent harm, not to confirm it. That's a point to which I will return.

Modeling Our Risks

With respect to human health, we must use toxicology and modeling to predict the future. We need to change the burden of proof away from insisting on demonstration of human harm, because to do otherwise means treating humans as though their injuries or deaths are required before action can be taken. Literally, we become the bodies of evidence. And that is what has happened in the evolution of environmental law recently. In the 1970s, when environmental law was in its heyday, the Precautionary Principle was embedded in the law. The Clean Air Act, the Toxic Substances Control Act, the Clean Water Act, all embodied the notion that efforts to predict risk were essential to figuring out how to prevent those risks from happening.

Unfortunately, implementation of the preventive thrust of these statutes has proven to be problematic. And what is at risk now is nothing less than that fundamental tenet of environmental law. I really look forward to talking to environmental lawyers about how we have to shift back to the original intent of the 1970's legislation, which I am old enough to have helped write. The early laws intended to force the government and the private sector to come up with a way of defining what was an "unreasonable risk" in order to prevent that risk from happening, not to confirm that it did happen. We must understand that typical environmental epidemiology studies, along with the health assessments of the much beleaguered ATSDR [Agency for Toxic Substance and Disease Registry], have become focused on proving human harm has already occurred, rather than seeking to prevent it. As scientists and as people concerned with public policy, we need to say loudly and clearly, that proof of human harm should never be the litmus test against which regulatory or other action should be gauged. We are not going to allow ourselves to become the bodies of evidence.

Focus on the Possibilities of Prevention

"People should not be the laboratory rats of the earth."

That is a statement from a breast cancer survivor in the film, "Exposure." If we fail to use experimental evidence, we are effectively making people into laboratory rats. How much proof do we need? That is a question that cannot be answered by science alone. That is a question that requires coalition building, such as this Massachusetts Precautionary Principle Project. Health reflects

multiple factors, many of which cannot be easily changed. Chemical and physical factors you all know about. Diet is important, but so are the habits that people engage in.

Finally, there is one other part that I think we all have to recognize – and there is a growing movement in the religious community to do so. Our health comes not only from things that happen to us externally and what we do to ourselves, it also reflects our spiritual identity and connections to something higher than ourselves, whether it is to a God in a church or synagogue or to the beautiful environment around us. This is a very important part of health. It's not a healthy environment if people can't have that connection to one another, whether it is in meditation, whether it's in a synagogue as I go to, or whether it's in other ways.

So many different parts of our lives affect our health. And many of them cannot be easily changed. That is why the environment becomes so important – because the environment is something you can do something about. It's one piece of the health puzzle that can best be changed by social policy. It is not possible to change your parents or alter what you ate 30 years ago. But, the environment can be controlled. We need to do a better job at bringing those pieces together by articulating those aspects that can be changed, and that's where the environment becomes critical.

Throughout our work on public policy, we need to look for opportunities to rely on prevention and precaution. I am going to talk about three quite distinct and important areas where the Precautionary Principle is relevant. Some of you have worked on breast cancer. Some of you have worked on reproductive health. Some of you have worked on climate. I'm going to try to talk about all those things and, finally, I am going to talk briefly about Donora, Pennsylvania, a small steel town of the Monongahela Valley where my family first lived.

Burden of Proof: The Evidence

Let's consider the three types of evidence that are usually available to those trying to assess public health. First there is laboratory or experimental evidence, which comes from controlled studies done under carefully simulated conditions or modeled conditions. It can involve studies in a petrie dish, studies in lab rats, computer analyses of climate, or ice cores, anything that is experimental. We must accept the fact that this evidence is absolutely critical to implementing precautionary policies. Second, for some instances, we also can draw upon wildlife or other natural studies. I think most of you are well familiar with the endocrine disrupter story with respect to wildlife. How many of you have heard of the alligators of Lake Apopca? Basically, in the years following a massive pesticide spill most of the alligators in this lake appeared to be female. Finally, we have human evidence, which can be the most difficult to obtain, and, if we are smart, it will remain so.

We must understand that there has been a general movement in the courts, regulatory agencies and in other public discussions to ask when it comes to controlling environmental pollution: Can we prove that this pollution causes

harm? I think we should no longer ask that question. The way a question is asked determines its answer. Consider this question instead: Is there exposure to materials that are likely to cause harm? That changes the question from “Do we have evidence that human harm has occurred,” to “Do we have evidence of human exposure to conditions that are likely to be harmful?” In essence, the Precautionary Principle argues that we should shift the calculus away from illness to exposure. I'm going to start my talk in breast cancer about evidence on illness and then end it with some evidence on exposures that should be controlled, while evidence continues to mount.

Breast Cancer Evidence

Fewer than one in ten cases of breast cancer arises in women born with genetic defects, as I think many here know. This means that nine out of ten women who get breast cancer are born with healthy genes and something happens to them in the course of their lifetime to give them the disease. That's the challenge that we face. Why do the nine out of ten women born with healthy genes get the disease? I'm not going to go into detail, except to say that most cases of breast cancer occur in women with few of the known risk factors. As a consequence, we have to get beyond continuing to confirm the old risk factors because the risk factors we have identified are often not causes of the disease.

Because most cases of breast cancer occur in women with so few risk factors, the search for environmental causes and other avoidable factors is getting increased attention from those who are wrestling with the disease. In fact, in less than half of all cases have any known risk factors been identified; among these are: menses beginning before age 12 and ending after age 55, having no children or late childbearing, not having nursed children, early or repeated exposures to radiation, higher socio-economic status, lack of regular exercise and vitamin D and fiber, obesity after menopause, and possibly higher levels of melatonin. Excepting radiation and early family history of the disease, these risk factors can all be tied with greater lifetime exposure to estrogen and other hormones and higher exposures early in life. Throughout life, the breast continues to develop, responding to a complex, shifting exchange of estrogen, progesterone and other growth factors. Even prenatal exposures can affect the chances that a woman will develop the disease. Girls born as fraternal twins have greater risks of breast cancer, possibly due to their greater overall exposure to hormones in utero. Thus, the earlier in life that regular menses begins and the later that it ceases, the longer that exposure to hormones extends that affect breast growth.

Efforts to identify new explanations for breast cancer rank among the most important that can be conceived at this time. The sad fact is that science has not resolved some of the most troubling questions about this common disease. Except for damaging radiation, most of the traditional risk factors, such as early onset of menstruation and late onset of menopause, are not direct causes of the disease; rather they are associated with it because they are markers for something else. For example, total lifetime exposure to certain metabolites of

estrogen will be higher in girls who start to menstruate earlier and women who enter menopause later. What are the things that cause that? Could it be contaminants in animal fat? Could it be fat itself? Those things are going to be the direct causes. These other risk factors are simply going to be things associated with it. So we have a puzzle.

Most cancer is not born, but results from exposures and activities that take place throughout life, including those that occur prenatally. Because women living with breast cancer are determined to keep their daughters and sisters from developing the disease, activists are turning attention to what kinds of exposures can be avoided or controlled that lead to these acquired malignancies. Research into the avoidable causes of breast cancer has yet to receive the same impetus as that on genes, with less than \$5 of every \$100 dollars spent being devoted to such matters. But even as we speak, that is changing.

Today in San Francisco, Cape Cod, and London, researchers are trying to tackle one of the most baffling and challenging puzzles of all. Why do women in some areas have higher rates of breast cancer? Organizations such as One in Nine and the Long Island Breast Cancer Coalition conducted public hearings and a major campaign that compelled the federal government to launch a multi-year, multi-million dollar project. This study asks why women born and living on Long Island for 40 or more years have four times more breast cancer than women who have resided there less than five years? Why do women who live near two or more chemical facilities in the region have between 60% to two times more of the disease compared with women who do not live in such areas? Funds from the State of Massachusetts and other private donors are supporting the Silent Spring Institute of Newton as it devises innovative studies to fathom why women who live on Cape Cod acquire breast cancer at rates that are from 20% to three times higher than those of women living elsewhere in Massachusetts. The Breast Cancer Fund, Breast Cancer Action and other California groups are urging study of the higher rates of breast cancer in San Francisco overall, especially in young black women in the Bay View Hunters Point area, a region notorious for chemical contamination and spills. In London, the National Federation of Women's Institutes and the Women's Environmental Network, with funds from the National Lottery Charities Board, has launched a nation wide, grassroots effort to construct maps of the distribution of the disease and link these patterns with pollution through state of the art, computer-modelled geographic information system analyses.

Out of this unprecedented public demand, something different is emerging. Scientists are being dragged by public concerns to explore the causes of breast cancer in ways not previously imagined. The safe course of science is to consider single factors under controlled conditions. This is how drugs are tested and developed and how elegant science works. When it comes to parsing through the complex causes of breast cancer, women cannot live through controlled experiments. Those with the disease do not have the luxury of waiting for such plodding puzzle-solving work to conclude. They need answers and directions about things that can be done now to reduce their risk. This has

led to a remarkable, sometimes forced collaboration, which finds breast cancer activists and basic researchers sitting at the same table. For instance, the Department of Defense includes a half billion dollars a year in research on breast cancer, which is doled out by collaborative review of activists and scientists.

Prediction of Harm to Avoid Harm

Why do we test animals? To identify new risk factors, to set standards, to set priorities, and to take emergency actions. That's why we test animals. We are blessed, some may say we are cursed, with talented toxicological modelers today who are adept at discerning the myriad ways in which animal metabolism differs from human's. While this is inherently fascinating work, it sometimes has the effect of obscuring the basic reason why animals are studied. Moreover, it has led to a circumstance where for some high profile compounds, such as dioxin or more recently phthalates, hundreds of studies are underway. In contrast, other high volume compounds, such as the artificial sweetener aspartame, nearly inescapable in today's diet food drenched world, or MTBE, one of the most common ground water contaminants used as an additive to reduce greenhouse gas emissions from gasoline, have undergone little toxicological testing and epidemiological study. The latter are not even planned to gauge these substances' impact on human health.

With all its limits and variations, this experimental and epidemiological evidence is a key part of building better models with which we can predict risk. It is critical to realize the simple reason why we use models. We do this in order to avoid experimenting on people. We have to keep that in mind. In an effort to protect human health, we have to rely more on the experimental models that we have and be smarter at using them, again, to predict harm, and to prevent that harm from occurring.

In our lifetimes, with the adjunct of computerized information technology, we will be able to use experimental information as a way to try to figure out the things we can do better, the things we can do smarter, the things we can do without. That is why redesigning our products becomes one of the fundamental challenges. Some of my architect and engineer friends say that environmental pollution is really nothing but a design failure. Those of us who were weaned on toxicology and epidemiology learned to deal usually with the end of the pipe, or what comes out of it. We need to create students who are going to redesign these products, who are going to create shoes that spontaneously degrade into the environment, who are going to create cars that are completely recycled and don't use any fossil fuels. Those are going to be the solutions to what we have done with respect to the pollution burdens that we have created.

Experimental Evidence

Now, I want to just show you a few slides from some experimental work as an example of some of the things we have to be smarter about using. This is

what happens when you take a petrie dish of breast cancer cells from a human being and let them alone for two days. They grow; because they are cancer cells, they will grow. If you give them these increasing doses of DDT, you see that up to 150 micrograms per ml accelerates the growth of these breast cancer cells more than eight-fold. [Figure 1] This is what happens with red dye number 3: this is currently a legally-used dye in junk food. We published this in 1997 in a monograph of the National Institutes of Health, Environmental Health Perspectives (Volume 108, April), which I was an editor of. As far as I know, nothing has happened to take this dye out of circulation. It is, in fact, on the GRAS (Generally Regarded As Safe) list. [Figure 2]

Now, I am not suggesting that this is the most important public health problem that we face, but simply that we have to be smarter about using this information to avoid exposure to things that are of some probable harm. I think if something takes breast cancer cells and increases the growth of them this much, we ought not to have it in our food supply.

Now, just to show you another example of experimental evidence we have to be smarter about using. This is a beautiful fluorescent stain of healthy breast cells communicating. And this shows, in the cancer process, cross-talk between cells that is critical to preventing cancer from developing. [Figure 3] Cells can tell one another to kill themselves, rather than to become cancer. Apoptosis. They can repair themselves. They can go into rest. They can go into various states of enzymatic repair. If you add a little bit of DDT, this is what happens to these cells. If you add a mixture of DDT and chlordane, these cells lose their ability to communicate. Literally there are gaps in the junctions between them, which impairs cell life and may lead to malignant growth. [Figure 4] This work was published by Kang et al in 1996. Now, this is experimental work. This is reason enough, it seems to me, to say that these materials ought not to be in widespread use. And, again, we now have to change the calculus, away from proving harm to showing exposure. And I think that that would be a fundamental advance; that we should not make ourselves into the lab animals of the world.

Reproductive Health Evidence

Now let me go over a little bit of human evidence that we have. I want to talk about what is happening in men in industrial countries. We need to create a "save the males" campaign. There are increases in testicular cancer in every industrial country. There are increases in defects of the penis of baby boys, hypospadias, increases in undescended testes of baby boys, and infertility, possibly reduced sex-ratios [reduced numbers of male to female births]. These pieces don't really all fit together except that they are all examples of things that reflect some disturbance in the hypothalamo-gonado-pituitary axis, in the way the body regulates itself hormonally. And many of them have got to come from prenatal factors, or things that happen even before conception to the mother or father. The father's role in producing healthy babies and defects in babies has been underestimated. Ted Schettler and Gina Solomon have produced a number of very important analyses that show that this has repeatedly been

under-appreciated. There shouldn't be any surprises here. People have been reluctant to accept the reality: it takes two parents to make a baby. And, therefore, some defects in that baby are going to be contributed from both parents equally and certain defects are only conveyed by the paternal gene element and not by the maternal gene element. So we need to pay more attention to that.

Just to tell you the hypospadias trends in the United States, CDC data from 1970 to 1993 shows a 50% increase. There have been significant declines in the ratio of males to females born in the U.S. and Canada (1970-1990: 38,000 less U.S. male births and 8600 less Canadian male births). In an article in JAMA, [Davis DL et al: 1998b] April 1, 1998, we discussed POSSIBLE causes of the decline of the sex ratio. While we say we don't know WHY it is declining, there are a number of studies that show that workers exposed to high levels of pesticides and solvents are unable to produce very many boy babies. In the people exposed to the highest levels of dioxin following an explosion of a chemical plant in Northern Italy, ten years afterwards in the town of Seveso, of the 20 people that produced a baby in the areas of highest level of exposure, guess how many produced a boy baby? Twenty babies born, all girls. Now it is a small sample, but it is, I think, something we should be concerned about. If the sex ratio in the United States and Canada had not dropped from 1970-90, as we calculated in our JAMA paper, there would have been 38,000 more baby boys born in the United States and 8,600 more in Canada. And leaving aside jokes about whether we would have cleaner homes or less war or not, this is just not a good thing biologically.

Now, let me add some recent findings from Tokyo that I am trying to get translated into English. The Tokyo medical examiner has conducted studies of the autopsies of Japanese young men killed in accidents from 1970 to 1998. Over the years the men's bodies became taller and heavier. Researchers measured their height, their weight, the weight of their brain, the weight of their liver, and the weight of their testes. And in proportion to the weight increase of their other organs and total body mass, their testes did not continue to grow after 1980. I think this is pretty important. I don't know why Japanese men now have smaller testicles, and I think this may be of interest to more than just Japanese women. But I think it is pretty important that we find out.

Negative Evidence: What does it mean?

When it comes to human evidence, things get a bit more complicated, because we can't really experiment directly on people. Instead, we can use epidemiology to follow groups of people to see what happens, or has happened to them and to try and associate that with some sort of possible cause. Now, let's be aware that epidemiology is a very blunt instrument. There is some confusion about how to interpret negative findings, which do not find any tie between environmental exposures and disease. As to negative results, the fact that we lack statistical significance in many circumstances is due to the small size of the samples we look at or the relatively rare nature of what we are studying.

Negative studies also tend to occur because of limitations in the ability to measure exposure accurately. Because we all must breathe air and drink water, there is basically no control group of persons who have not breathed or drank some of the pollutants with which we are concerned today. This tendency to misclassify exposure tends to bias all epidemiological study results in a systematic way. As my colleagues at Johns Hopkins University, Professors Jonathan Samet and Scott Zeger, have elegantly shown in a series of innovative modeling analyses, positive results in environmental epidemiology studies of large-scale data sets usually understate risk. Statistical significance should not be confused with public health importance. By the time we have certain scientific evidence that achieves statistical significance it is often too late. As Professor David Ozonoff of Boston University once quipped, a public health disaster is something so obvious that even an epidemiologist study can detect it.

As to the conflicting literature on breast cancer and pesticides, this provides an excellent case of how flawed negative studies can arise that make the interpretation of findings very confusing to non-experts. For many chronic diseases, including breast cancer, there is a long latency between exposure and effect, with prenatal exposures being of unspecified importance. This means that we would have to follow a group of people for their entire lifetime to see what chemicals or habits might lead to certain health problems. However, because of the amount of time and resources this type of study requires, no such studies have ever been conducted.

With respect to problems of exposure classification, breast cancer studies also provide an excellent illustration. Breast cancer can result from exposures that take place early on in life, as well as those that occur at different ages. The environment is messy. It involves lots of components that are not well characterized and can be difficult to measure. People seldom know what they have been exposed to throughout their lives. Key exposures can occur at three critical periods of life – prenatally, during adolescence, or later in life. Studies that assess current patterns of the disease in adults and contemporaneous exposures to chemicals are incapable of addressing the role of early life exposures. Looking at prevailing patterns of pollution, diet or other suspected behavioral risk factors to explain cancer rates today is akin to expecting the present to account for the past. Of course, efforts to characterize pollution in the environment are welcome in their own right, as they afford the public critical information about hazards in their communities. Still such analyses remain limited in their potential to resolve many key questions about breast cancer.

Inconsistent results

Because of negative studies, results concerning the role of synthetic materials, such as some lipophilic organochlorine pesticides, in breast cancer have been inconsistent. These pesticides contain chemicals which are known to act like hormones, such as estrogen, in the body. Some early studies on organochlorine residues tended to be positive, with higher levels of PCB and

DDE, either in the blood or fat, found among women with breast cancer than in those without cancer [Dewailly, 1994; Glass, 1990]. However, in some studies, negative results were described for the total cancer study population, or for specific subgroups of breast cancer subjects, such as Asian women [Lopez-Carrillo, 1997]. In several recent studies, women with breast cancer were reported to have lower levels of organochlorine residues in their bodies compared with women without the disease.

These studies, however, fail to look at the larger picture; at the environmental and socio-cultural factors which also impact health. For example, in Asia, people tend to eat foods high in possibly protective substances, such as soy and fish, and also have high fiber diets. In the three studies where breast cancer victims had lower levels of organochlorine residues, the role of exposures during critical windows earlier in life were not examined, only the residues remaining after the disease was expressed. In two of these studies, the cases were already in the middle stages of breast cancer. In these circumstances, preclinical disease may have altered lipid, or fat, metabolism, which could explain these lower levels of compounds.

Studies such as these, which consider current levels of pesticide metabolites (the substances which these chemicals produce in the body), have been described as analogous to “looking under the nearest lamppost for lost keys because that is where there is light.” They do not ask two critical questions: what were exposures to hormone-like chemicals during critical windows of development, including before birth and pre-pubescent times; and what was the lifetime exposure to these chemicals?

So what about all these human studies that are underway today on breast cancer and the environment? This work – epidemiologic research on the environment – needs to be understood as a limited, though critical tool. Such research can sometimes confirm the importance of historical exposures for chronic diseases, such as cancer, but can rarely be used to predict future outcomes. Further complicating this is the fact that key relevant exposures cannot be divined through the usual instrument of human studies, a questionnaire. It makes little sense to ask people what chemicals they have been exposed to or what foods they typically ate three decades ago, when most folks are blissfully unaware of such matters or cannot accurately recall those things of which they were informed.

Epidemiology confirms the past. Prevention is better than treatment. That should be a mantra. That doesn't mean we do not need epidemiology. Of course we do. But we should not make positive epidemiology studies the requirement for regulatory action.

Children's Risks in a Changing Climate

Now, I am going to quickly tell you about a few studies we've done on children because they indicate that globally, children are being exposed to levels of air pollution known to be harmful. This work is being developed in the context of general studies on climate and human health. Climate change entails

a complex process, ranging from temperature shifts at the local and global level, to sea level and weather instability.

A number of unusual events have been reported recently. Whether these are merely normal variations or constitute some shift in overall patterns remains to be seen. For instance, tropical forests aren't supposed to burn. Following the drying affects of El Nino in some regions, areas that normally are resistant to fire can become quite vulnerable to it. When they do, it upsets an entire ecosystem for many years. For instance tigers are endangered by the burning forest. Also, other bizarre things are happening to weather all over the world. One picture from the newspaper in Los Angeles last January showed snow on the palm trees. Recent data from NASA show an increase in global mean temperature, with 1998 being the hottest year on record and 1999 being the sixth hottest ever recorded.

Fossil fuels create both local air pollution and also contribute to the buildup of greenhouse gases. We know that children are being exposed to this air pollution in cities around the world today. The sources that we have identified are important because particulate air pollution, no matter whether it comes from forest fires or cigarette smoke or fossil fuels, gets deep into the lungs. Some of these particles are highly reactive. Some of them may function almost as gases because if they are very fine, they can get into the bloodstream.

I'm going to talk to you just briefly about a study that we've been doing with the World Health Organization, looking at where the most children are at the greatest risk from air pollution globally on an annual basis. Eighty-five percent of the world's children live in the developing world and half of them live in cities. And in those cities, they are breathing air that is equivalent to inhaling side stream smoke from two packs of cigarettes each day. Now, this fact I dug out from a 1991 publication. It made it around the world on a slow news day because I presented it like this to the AAAS [American Association for the Advancement of Science] meetings.

The lesson for you here is that if you can talk about children's exposure, for a variety of complex reasons, you can push buttons and trigger interest that you don't get when you talk about someone like me, now, who is a 52-year-old mountain climber hit with asthma for the first time in her life. Nobody cares about us old people dealing with asthma, but when I was a child growing up in Donora, Pennsylvania, people might have cared if they understood that I was breathing air as dirty as side stream smoke from cigarettes every day.

What we did in this study that we released officially this summer (Davis DL et al: 1999, available at www.wri.org and www.climate.org) is we calculated where the most children are with the greatest risk in the mega cities of the world, and Mexico tops the list. We need to pursue aggressive policies with respect to climate because we know that what we will be doing by reducing inefficient combustion of fossil fuels is promoting public health and reducing the eventual buildup of greenhouse gases by increasing energy efficiency and assuring sustainable development.

Back to the Future – An Historical Air Pollution Episode

Now what I am going to do at this time is move to something that's a bit personal. I have decided that there is a reason why I have been affected with asthma at this time. It is a manageable disease, but I want to share with you something about a work in progress about my hometown, which I call, "Killer Smogs: Back to the Future."

The 50th anniversary of the first acknowledged killer smog to hit America passed this fall [1998], unmarked by parades or misty-eyed, quivering-lipped Presidential speeches. A half century ago, the small working class town of Donora, in the Monongahela River Valley of southwestern Pennsylvania, drowned in a blanket of lethal sooty air. Within 12 hours on Saturday, October 29, 1948, 17 people dropped dead, and the local funeral homes ran out of coffins.

Donora was the kind of town where when a three-year-old ran five miles away from home, across the Webster Monongahela Bridge, it made headlines, each of the two times he did it. "Runaway Marty Does It Again." That was my brother. Everybody in Donora either worked in the local steel mills or worked to feed, clothe, fuel or take care of those who did. Like many towns in what is now the rust belt of the Monongahela and Ohio valleys, Donora sprang up around its metal works and foundries. Nearly a century ago, William Donner began building an iron mill at a horseshoe bend in the fast moving river. Enough immigrants showed up for jobs that a year later the town was officially incorporated. At the end of October, 1948, the homes and streets of Donora were clotted with a lethal fog of coal, coke and metal fumes. Cooler, heavier air sat on top of lighter, warm zinc and sulphur-laden smoky poisonous gases, fluoride and sulphur, from the local mills, and coal from the furnaces and stoves at homes of the residents in town. A black fog floating up from the river turned days into nights. That was 1948. This is 1999. The only thing left standing from my grandfather's scrap yard is a remnant of the steel wire fence and some rotting wooden posts that formerly marked the front gate. A rusted old oil drum sits right behind the fence, a solitary residue of what had been heaps of colorful, forbidding junk spread over a few acres that seemed to me, as a child, to go on forever.

As to the mill, for years nothing marked the fact that so many people had died in what became one of the first publicly-understood episodes of deadly air pollution. An earnest high school student brought that to an end on the 50th anniversary of the smog this past October. The state historical and museum commissioner erected a bronze plaque near the mill's former center as a memorial to those who died from air pollution in 1948. To mark the occasion, residents and local and state officials held a memorial service at the only remaining church in the town. No plaques could be mounted for those who did not succumb at the time of this pollution but went on to earlier deaths than they would have, or just became a bit sicker for the rest of their lives. Whether plane crashes, hurricanes or deadly air pollution, disasters where many people die at once command public notice. If 20 people are killed in a commuter airline crash, or from crippling air pollution in a few days, this makes a front page story. If over

the course of several months, 2,000 individuals are killed while driving to get to different flights at different airports, or die early deaths from air pollution, this goes unnoticed. No tally is routinely made or reported.

Today nobody knows what happened to most of us who grew up in Donora; most are long gone. Some of the records of the health department surveys, conducted right after the fog, burned in a fire several decades ago. A 1994 doctoral dissertation from the University of Pennsylvania was unable to turn up the expected kinds of papers. No correspondence files, no site visit records, no maps, no rough drafts of reports could be unearthed from the Public Health Service files on what turned out to be the start of the modern environmental movement in this country.

You have to suspect the worst. Not only of U.S. Steel, but of the Public Health Service. Someone may have decided the data gathered by the investigators were too hot to handle and got rid of them. Council leader Burgess August Chandler ended a heated public session in Donora in early 1949 with this plea: "No one should go away from here with a chip on his shoulder. I'm ashamed to make this statement, but it is a blessing that we lost these lives to bring this thing to light, in the hope that we can save thousands of more lives."

So here I am, 51 years later, and I think there's something that we all have to do. If people had listened to the warnings of Donora, the world today would be very different. At this point, it is wonderful that you are all here, that we are all here, on the Sabbath day to see that there are no more Donoras. But I can tell you from the work I am doing in China and Mexico and Brazil, they are there. And that is why I am going to spend the next year trying to write more about the Donora Smog and related conditions around the world today, and digging for the evidence on what really happened there. I hope that through the stories I will be able to move people to take the kinds of precautions that stop making ourselves the bodies of evidence.

References

Davis DL, Axelrod D, Bailey L, Gaynor M, Sasco AJ. Rethinking Breast Cancer Risk and the Environment. The Case for the Precautionary Principle. *Environmental Health Perspectives*. 1998a; 106(9): 523-529.

Davis DL, Gottleib MB; Stampinsky JR. Reduced Ratio of Male of Female Births in Several Industrial Countries: A Sentinel Health Indicator? *JAMA*.1998b; 279(13):1018-023.

Davis DL, Saldiva PHN, Ahmed KA et al. Urban Air Pollution Risks to Children: A global Environmental Health Indicator. *Environmental Health Notes*. World Resources Institute, Washington D.C. 1999.

Dewailly MS, Dodin S, Verrault R, Ayotte P, Sauve L, Brisson J. High organochlorine body burden in women with estrogen receptor-positive breast cancer. *J Natl Cancer Inst* 86(3):232-234 (1994).

Glass R, Hoover RN. Rising incidence of breast cancer relationship to stage and receptor status. *J Natl Cancer Inst* 82(8):693-696 (1990).

Kang KS, Wilson MR, Hayashi T, Chang CC, Trosko JE. Inhibition of Gap Junctional ntercellular Communication in Normal human breast epithelial cells after treatment with pesticides, PCBs, PBBs, Alone or in Mixtures. *Environmental Health Perspectives*. 1996; 104(2): 192-200.

Lopez-Carillo L, Blair A, Lopez-Cervantes M, Cebrian M, Rueda C, Reyes R, Mohar A, Bravo J. Dichlorodiphenyl trichloroethane serum levels and breast cancer risk: a case-control study from Mexico. *Cancer Res* 57:3728-3732 (1997).

Madigan MP, Ziegler RG, Benichou J, Byrne C, Hoover RN. Proportion of breast cancer cases in the United States explained by well-established risk factors. *J Natl Cancer Inst*; 87(22):1681-1685 (1995).

Schettler T and Solomon G. *Generations at Risk: Reproductive Health and the Environment*. MIT Press, Cambridge, MA.1999

Acknowledgement

This work was supported in part by grants from the Susan G. Komen Foundation, the Breast Cancer Fund, and private donors. Barbara Ley, Miranda Loh, and Margaret Powell provided valuable research assistance and critical editorial review. For a slide show on this topic search through www.wri.org or www.breastcancerfund.org.

Biography

Devra Lee Davis, Ph.D.
email: ddavis@andrew.cmu.edu

After five years with the World Resources Institute, Devra Lee Davis, a leading epidemiologist and researcher on environmental health and chronic disease has been appointed Visiting Professor at Carnegie Mellon University's Heinz School, where she is working on a book, *No Body Counts: How the Environment Shapes, Life, Death and Sex*.

President Clinton appointed the Honorable Dr. Davis to the newly established Chemical Safety and Hazard Investigation Board, (1994-99) an independent executive branch agency that investigates, prevents, and mitigates chemical accidents. As the former Senior Advisor to the Assistant Secretary for Health in the Department of Health and Human Services, she has counseled leading officials in the U.S., United Nations, World Health Organization and World Bank. She also was a Distinguished Visiting Professor at The Yeshiva University and Stern College for 1996-97 and Scholar in Residence and Executive Director of the Board on Environmental Studies and Toxicology at the U.S. National Research Council, of the National Academy of Science, 1983-93.

Dr. Davis holds a B.S. in physiological psychology and a M.A. in sociology from the University of Pittsburgh. She completed a Ph.D. in science studies at the University of Chicago, as a Danforth Foundation Graduate Fellow, and an M.P.H. in epidemiology at the Johns Hopkins University as a Senior National Cancer Institute Post-Doctoral Fellow.

She has authored more than 160 publications, in books and journals ranging from *Scientific American* to the *Journal of the American Medical Association* and the *Lancet*, and the *Annals of the New York Academy of Sciences*, and has also written for *the New York Times*, the *Los Angeles Times*, and other mass media outlets.

A member of both the American Colleges of Toxicology and of Epidemiology, Dr. Davis is also Visiting Professor in the Department of Environmental and Occupational Medicine at Mt. Sinai Medical Center in New York City. In addition, she is a Visiting Scientist of the Strang Cornell Cancer Prevention Center of the Rockefeller University and Scientific Advisor to the Women's Environment and Development Organization. She also founded the International Breast Cancer Prevention Collaborative Research Group, an organization dedicated to exploring the causes of breast cancer. She currently serves on the Board of the Breast Cancer Fund and the Mickey Leland National Urban Air Toxics Research Center, the Climate Institute, and the Coalition of Organizations on the Environment and Jewish Life.

Figure 1: Breast Cancer Cell Growth and DDT
(Source: Dees et al., 1997)

Figure 2: Red Dye Effects on Breast Cancer Cells
(Source: Dees et al., 1997)

Figure 3: Normal Cell Communication

(Source: Kang, et al., 1995)

**Figure 4: Abnormal Cell Communication after
DDT exposure**

(Source: Kang, et al., 1995)

Massachusetts Precautionary Principle Partners

Contact information

Clean Water Fund
36 Bromfield Street #204
Boston, MA 02108
Tel. 617-338-8131 Fax 617-338-6449
Email: bostoncwa@cleanwater.org

Lowell Center for Sustainable Production
University of Massachusetts Lowell
One University Avenue
Lowell, MA 01854
Tel. 978-934-2981 Fax 978-4522-5711
Email: joel_tickner@uml.edu

Massachusetts Breast Cancer Coalition
Contact: Sharon Koshar
51 Diauto Drive, Suite B
Randolph, MA 02638
Tel. 1-800-649-6222
Email: 1in8@mbcc.org

Please give credit to the Massachusetts Precautionary Principle Project when making reprints.
Originals printed on 100% recycled, post-consumer, process chlorine-free paper.

* Visiting Professor, Heinz School for Public Policy and Management, Carnegie Mellon University. Pittsburgh, PA

PAGE

PAGE 19

PRECAUTIONARY PRINCIPLE PROJECT

CLEAN WATER FUND ♦ LOWELL CENTER FOR SUSTAINABLE PRODUCTION ♦ MASSACHUSETTS
BREAST CANCER COALITION ♦ SCIENCE & ENVIRONMENTAL HEALTH NETWORK

EMBED MSGraph.Chart.8 \s

EMBED MSGraph.Chart.8 \s