

# Scientific Findings Support Replacing Poisons with Precaution

In the face of limitations in defining and regulating pesticide hazards, medical doctor and epidemiologist calls for avoiding pesticide use and adoption of the precautionary principle.



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**EDITOR'S NOTE:** Dean Baker, MD, MPH, is professor emeritus of medicine, epidemiology and public health in the School of Medicine, and former director for 23 years of the Center for Occupational and Environmental Health, University of California Irvine, California. This piece is taken from a talk, **Protecting Family Health and the Environment**, given by Dr. Baker to the 36th National Pesticide Forum, **Organic Neighborhoods: For healthy children, families, and ecology**. In this piece, Dr. Baker explains the complexities of studying the health effects of pesticides—from evaluating toxicity, exposure, and health outcomes. Because of the severe limitations in defining risk, he challenges us to embrace a prevention-oriented approach to chemical use under the precautionary principle, which states: When an activity raises threats of harm to the environment or human health, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. The context of his experience as a medical researcher and epidemiologist, as explained in this discussion, is central and important to his conclusion that we must urgently adopt alternatives to toxic pesticide use.

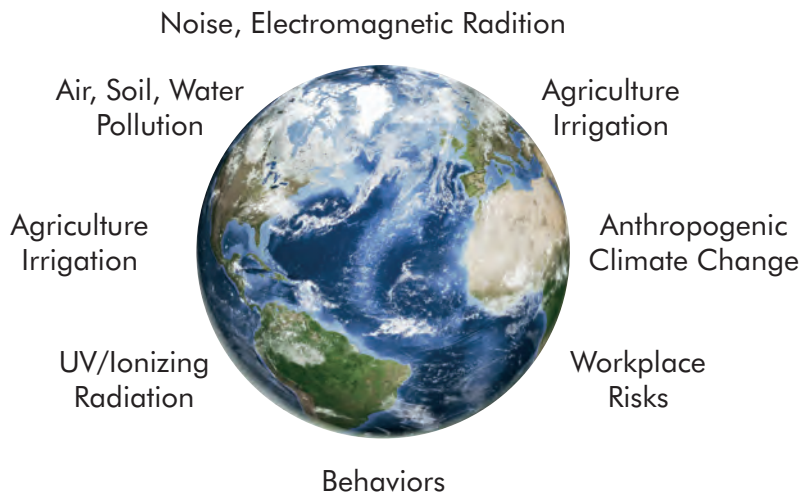
**DEAN BAKER, M.D.**

**T**he role of people like myself, academics who do research, is, one, to generate research and new knowledge, and, two, to be supportive of communities—because ultimately it takes community and people action to get things done.

The purpose of my talk is to give you an overview and introduction. The basic point to make is that, according to the World Health Organization (WHO), 22% of the global burden of disease is due to environmental factors. By that, beyond diet, they mean air, soil, water pollution, noise, the built environment, and agriculture. I am going to focus on pesticides, but there is a whole world of environmental factors. Collectively, environmental factors cause up to 22% of deaths in children less than five years of age. They list indoor and outdoor pollution. Indoor pollution includes mold, indoor combustion worldwide of organic materials (which is one of the major causes of death and disability among the women who are cooking and the children), lack of water, sanitation, disease vectors, inadequate food supply, and chemical hazards—with pesticides being one of the chemical hazards. We all know the story about lead, heavy metals, plasticizers, flame retardants. So there is a long list of chemical hazards.

FIGURE 1

## World Health Organization: Environmental Factors Responsible for 22 Percent of the Global Burden of Disease



**26% of deaths in children under five years due to modifiable environmental factors (WHO, 2016).**

Photo: © iStockphoto/Jimmy James Bond

## DEFICIENCIES IN ESTABLISHING SAFETY

Many of the themes we will talk about are the challenges in trying to get good regulation, regardless of the chemical, to reduce exposures. So, what are we facing? There are millions of chemicals registered in the chemical registry of the American Chemical Society, or the Chemical Abstracts Service (CAS) Registry. There are over 80,000 chemicals that are produced and used in the United States, most of them having been synthesized in the past 50 years. There are 2,000 new chemicals introduced into commerce every year. The majority have not been tested for other than acute toxicity. Over 95% have not been tested for their effects to children.

In biological surveys, like the National Health and Examination Survey (NHANES), many of these chemicals in trace amounts are found in most Americans. In some cases, like DDE (the breakdown product of DDT), even though it has not been allowed in the U.S. for decades, 99% of the population has at least trace levels in their blood. Because of the poor testing and the challenges in conducting research, we really have the classic toxic iceberg of what is known, what is partially

### BOX 1

## In Response to a Pesticide Tragedy

The Center Occupational and Environmental Health was created at University of California Irvine, along with centers at UCLA, UC San Francisco, UC Berkeley and UC Davis, by state law in 1978. The law provided permanent funding for research, training, and service in occupational and environmental health. We at Irvine have programs in occupational and environmental medicine. We train specialist doctors in the field. We also have graduate programs in exposure, toxicology and epidemiology. I think the poignancy of the centers is that they were actually funded by the state in response to an episode in the mid-1970s where workers at a plant in northern California were exposed to 1,2-Dibromo-3-chloropropane (DBCP), which is a fumigant

The story basically unfolded as workers at this plant socialized together. They had a baseball team, and while they were out there playing baseball, a lot of their spouses would be sitting in the stands. They got to chatting with each other and realized that none of them had kids for a while. They thought this was unusual and they talked with some public health officials. So, they started to do an investigation and they tested the men and they found out that almost everybody who had worked at the plant for a year or more either had no sperm or a very low sperm count, and were virtually sterile. They did some

research on it and found that DBCP was produced in this plant.

DOW (and Shell) had done research, which was published in obscure journals in the mid-1950s, 25 years earlier, that showed that in animal toxicology studies there were atrophied testicles and sterility in male animals that had been exposed to this pesticide. But, it was industry-sponsored research and never made it into the broader literature. This led to, as you might imagine, outrage in the state and it eventually led to the state legislature deciding that public funding was needed for more research and the training of more professionals in the field.

Our center, which has existed since the late 70s, really came about because of a pesticide and an unfortunate history about its use and in hiding the pesticide's adverse effects from the public. It is quite appropriate that the Center would be a cosponsor of the National Pesticide Forum. The research did lead to the banning of the use of DBCP in the U.S., at least, by 1979. It was allowed for several more years in Hawai'i before it was banned there. And, it was allowed on banana plantations in Central America, where there have been lawsuits going on until quite recently on worker exposure to the chemical. So, it can be banned for use in the U.S., but it can still be manufactured and exported.

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## Environmental Risks Identified by the World Health Organization

- Outdoor air pollution
  - Indoor air pollution
  - Lack of water and inadequate sanitation
  - Disease vectors, such as mosquitoes
  - Inadequate food supply—malnutrition
  - Chemical hazards and hazardous waste
  - Injuries
  - The built environment
  - Disasters and conflicts
  - Global environmental change
- 

proven, and what is not yet recognized—and perhaps will be forever unrecognized. Even with all the bad news, there is probably worse news out there when we eventually find out about the toxic extent of our problems.

### WHAT DO WE KNOW?

We know that children are more vulnerable than adults. They are not just little adults. They have developmental toxicity. From the time of gestation, in development, which does not end at birth, the nervous system is still developing. That is why children do not get up and walk right away. Their nervous system has not completed the myelination of the long nerves. This cell differentiation, organ development, and growth continue through early life and adolescence. Children are rapidly growing and developing. They have less developed natural defenses. For example, lead that gets into the gut is more easily absorbed. Lead that is absorbed more easily crosses the blood-brain barrier to the brain, which is a target organ. Even when exposed to the same amount of lead as an adult, children actually get more exposure dose at the target organ. In addition, the developing brain is more vulnerable—more exposed, more absorption, more to the target organ, and more vulnerable target organ. They have more skin per pound of body weight and eat more per pound.

We also know that children are natural explorers. They spend more time on the ground or floor. They have mouthing behavior that creates another pathway—ingestion.

### FAMILY HEALTH

In looking at all family members, puberty can be a vulnerable period. Exposure at puberty can have effects on sex differentiation and growth of reproductive organs, pregnancy, reproduction, and aging. We are now starting to focus on the environmental factors that influence age of menarche (the first occurrence of menstruation), menopause in women, and premature ovarian

failure. There is growing research evidence of environmental factors, studied by neurotoxicologists, associated with Alzheimer's and Parkinson's disease, and the quality of aging and cognitive decline, again associated with environmental factors that can lead to oxidative stress (the imbalance of unnatural sources of oxidizing substances, including toxics, and antioxidants in the body). There are occupational and community exposures that often cause the highest exposure in economically disadvantaged populations, like farmworker communities.

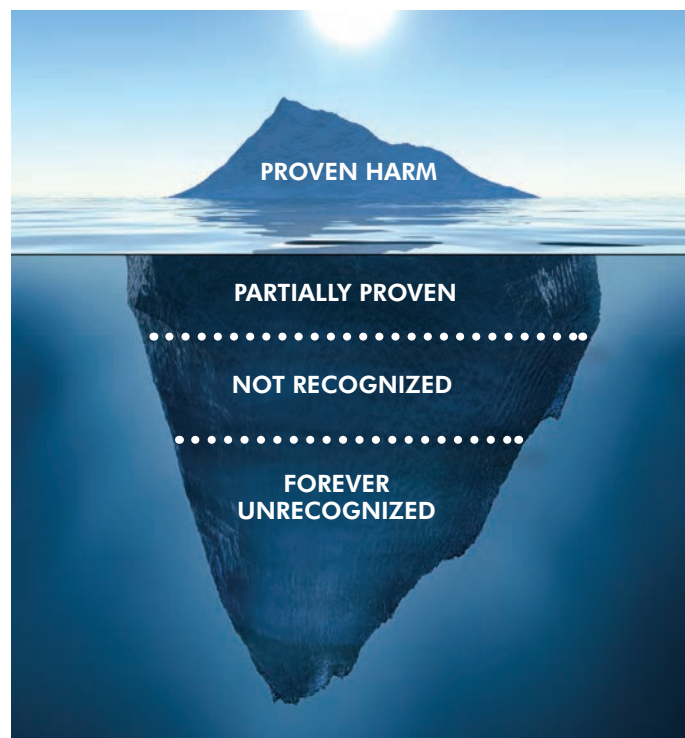
### HOW DO WE STUDY ENVIRONMENTAL FACTORS?

There are the exposure sciences that measure the pathways of exposure, the settings and the media, to try to understand whether there is a completed pathway of exposure from where a pesticide is being sprayed. With aerial spraying and drift, it is inhalation exposure, but we have to understand that putting a school next a field that is being sprayed is not very smart.

In toxicology and basic sciences, we hear a lot about animal studies and the research on mechanisms. It is probably the area that is given the most weight by the regulatory agencies. But it is limited because, while acute toxicity can be studied, for chronic toxicity—transgenerational, reproductive, and neurodevelopmental effects—there are not great assays and they are not really supported. So, there is a lot that we do not know. slide

Epidemiology, what I do, studies the patterns of effects in human populations, in the workplace, home, and community. Risk assessment tries to put all this information together and

FIGURE 2  
The Toxic Iceberg



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## Children's Health

**W**hat do we know about children's health and development? The patterns of illness of children in developed countries have changed substantially in the past hundred years, particularly with nutrition and control of infectious diseases. We still have a lot of premature births and birth defects, asthma, injuries, childhood cancers, neurodevelopmental disorders, obesity, and diabetes. There are adverse trends for a lot of these.

Asthma prevalence went up dramatically from the 1980s to the mid-1990s and has leveled off since then. Over 30% of children are either overweight or obese. Children diagnosed with Attention Deficit/Hyperactivity Disorder (ADHD) and the percent of children with autism spectrum disorders has gone up. While it was thought that there could be a diagnostic or recognition bias, the Centers for Disease Control and Prevention (CDC) funded in several different states a study with an evaluation, examination, and diagnostic protocol and five years later, using the identical protocol and criteria, showed that there was a real increase in autism spectrum disorders not due to study bias. ADHD is clearly increasing.

FIGURE 3

### Trend in Overweight ( $\geq 30$ kg/m<sup>2</sup>), Children 12–17

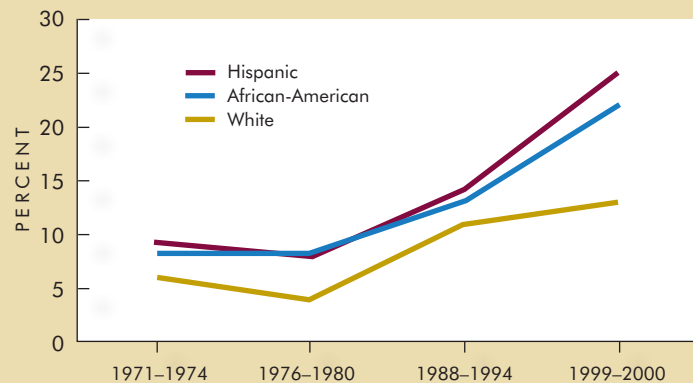


FIGURE 4

### Percentage of Children Ages 3–17 with Autism Spectrum Disorders, 2007 and 2011/12



Source: National Survey of Children, Health Resources and Services Administration

make some estimates about what amount of exposure or dose might be acceptable in the population.

The Centers for Disease Control and Prevention (CDC), the Agency for Toxic Substances and Disease Registry (ATSDR), created by the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA—the Superfund law), conducts work that shows there are multiple pathways of exposure through inhalation and ingestion from an industry, the pollution of rivers, the soil, and the food supply. In order to understand, from the toxicology to the translation into epidemiology and human health effects, we have to be able to measure exposures—which is quite complicated. So, there is a whole field of exposure science.

### EPIDEMIOLOGY: MEASURING REAL WORLD EFFECTS

In epidemiology, we have to keep in mind that there are many domains of exposure. Some of them can have similar effects,

synergistic effects, and additive effects. Therefore, we have to look at chemical, physical, psychosocial, and biological exposures. Of course, genetics is a risk factor, although not an exposure. We know about gene expressions. Although we have set genes, through epigenetics and other mechanisms some can get turned on and some get tuned off. This can all be modified by health care, education, and home and community environment. A range of outcomes can be caused, affecting pregnancy, neurodevelopment and behavior, asthma, obesity and growth, child health and development, injury, and reproductive development.

We have the additional complication, which I refer to as “life course epidemiology,” since we have multiple exposure, mediators, and outcomes. All of this occurs over time. Again, some exposures are high enough and cause acute effects, but many exposure do not cause acute effects, but go on for a long time and cause delayed or chronic effects. In order to really study these, we have to look at the exposures in the many

domains, combined with the genetics and the epigenetics, and look over the life course from pregnancy, infancy to childhood—follow people and look for the outcome. That is the challenge that we face in epidemiology and generally in environmental research.

### CASE STUDY: HEPTACHLOR

A retrospective cohort study looked at the use of heptachlor [an organochlorine pesticide in the DDT family], which was banned from use on the continental U.S., but in the early 80s it was still allowed on the pineapple plants in Hawai'i because they said there was no alternative. So, what happened? In 1980 to 1982, heptachlor was sprayed on the pineapple plants to control ants and mealybugs, but after harvest it got into the milk supply and pregnant women drank it. Many years later, we were asked to go back and we showed that the mothers drinking milk was associated with higher blood levels of the chemical in the women. Then we were asked 15 years later to do a neurobehavioral study. What we found, when we studied high school students who were randomly selected, was that the mothers' reported milk consumption was associated with worse neurobehavioral performance and more behavior problems reported by the teachers—who clearly did not know anything about the mothers' exposure to the milk. We then did a neurobehavioral study in which we gave the adolescents pneumococcal vaccinations and we went back six weeks later and got titers [measured antibodies]. What we found is that among the Oahu born children there was an inverse dose response—the lower the titers the more the mother drank milk. In a non-Oahu born cohort, we did not see that effect.

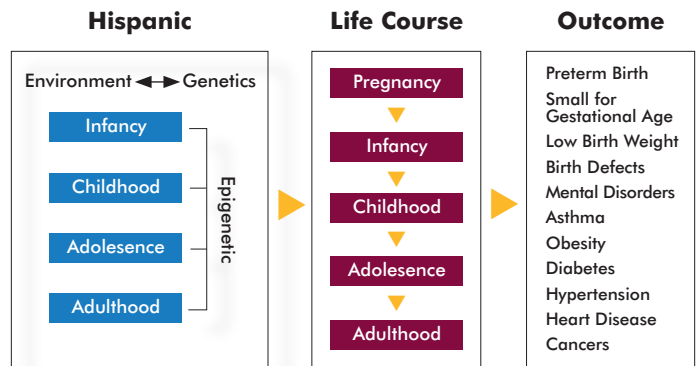
### THE CHALLENGES OF ASSESSING HAZARDS

So, there are challenges: Exposure Assessment—multiple exposures, transient exposure; Toxicology—few chemicals are fully tested for toxicity. Most are tested for only acute effects, and; Epidemiology—there is a long latency. Additionally, there are windows of vulnerability, so it is not just the pure dose and exposure, it is when the dose and exposure occurs. There are vulnerable subpopulations, gene-environment interactions, and lots of technical issues related to how we select populations, how to make sure your comparison populations are actually comparable, and how to measure exposure and outcomes without errors, or biases.

### SUPPORTING RESEARCH

Research takes time and requires funding, while hazards continue. We cannot just wait for the research to be done. Now, the funding for environmental and occupational health research, particularly at the federal level, is abysmal. Actually, the White House budget proposed a zero extramural funding budget for the National Institute for Occupational Safety and Health this year. Do they not want research because they do not want to have those answers out there?

FIGURE 5  
Life Course Epidemiology



We know a lot about pesticides and their effects. But, there is a lot we still must learn. I look at it as the challenge of new pesticide production and whack-a-mole research. We had organochlorines. Then they were replaced by organophosphates and carbamates. Then it is on to new classes of chemicals. We do research and show that is harmful and it is on to new classes of chemicals. It is whack-a-mole. They keep producing and we keep showing there is harm because pesticides are meant to cause harm. Continuing research needs on health risks require that we look at multiple pesticides, combined effects, effects of adjuvants [chemical added to boost performance], effects in vulnerable populations, and the development of appropriate risk assessment strategies to lead to efficient and quick regulation to protect, rather than waiting years and years for research to take place.

### PREVENTION AND THE PRECAUTIONARY PRINCIPLE

The fundamental approach requires a prevention orientation toward diseases caused by toxic chemicals that are preventable. We need to think about prevention in the context of the home, school, community, clinical practices of physicians, research, advocacy, and legislation. slide

This leads to the precautionary principle—when an activity raises threats of harm to the environment or human health, precautionary measures should be taken, even if some cause and effect relationships are not fully established scientifically. We cannot wait for the final answers. Research just takes too long. In this context, the proponents of this activity, rather than the public, should bear the burden of proof. This is an approach that is being taken more in Europe and it needs to be taken more in the U.S. In the meantime, we cannot rely on regulations. We have to use local and personal actions to take precaution.

*To view a video of Dr. Baker's talk, please go to [bp-dc.org/protectinghealth](http://bp-dc.org/protectinghealth).*