

The Precautionary Principle (*Vorsorgeprinzip*): [*Vorsorge*: care ahead; fore-care]

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“When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.”

The ethical directive *primum non nocere* (first do no harm) has long been a guide for health care professionals weighing risks and benefits of medical recommendations. The precautionary principle (PP) extends that charge to others in positions to protect human health and the environment more generally. It calls for taking action to reduce threats of harm to what we love or depend upon. Preventive care. Not after-care. Not hospice care. Rather, forethought...fore-caring. Make sense?

No, say critics who find it un-American in a risk-taking nation. Are we to ban all activities that might be harmful? [Paralyzing](#) and a [mythical concept, perhaps like a unicorn](#) said two former directors of the Office of Information and Regulatory Affairs at the Federal Office of Management and Budget.

In a sense the PP is subversive. It challenges established ways of making decisions based on a clear-eyed assessment of their track records. In the US, advocates of the PP point to thousands of untested industrial chemicals and other pollutants released into the environment that have worked their way into people and wildlife, sometimes with disastrous effects that were only identified much later after the harm was done. Beyond that, dead and dying forests, crashing fish and other wildlife populations, loss of soil and biodiversity, climate change—the list goes on.

Much of this could have been avoided with different approaches to decision-making. But critics insist that implementing the PP would impede progress and economic growth, and only established proof of harm should be grounds for restricting a product or activity. That threshold leaves us with the question: When do we know enough to act?

In the late 1990s, concerned about the impacts of environmental chemicals on health outcomes other than cancer, several colleagues and I looked more closely at [reproductive and developmental effects](#) and [damage to the developing brains of children](#). Our interest was sparked by documented sperm count declines, increases in infertility, adverse pregnancy outcomes and various birth defects and developmental disorders. We extensively reviewed published laboratory and epidemiologic data, which were more complete for some toxicants than others.

There was little doubt, for example, that lead, alcohol, and mercury were harmful to the developing brain of a fetus but evidence of the impacts of certain kinds of pesticides was more limited. Even then it was clear from animal studies that organophosphate (OP) pesticides, including chlorpyrifos, disrupted brain development but human data were still limited. In our

book, we noted that the mechanisms by which OP pesticides impact the brain are shared by laboratory animals and humans and predicted similar impacts in the offspring of women exposed during pregnancy. It was a strong argument for replacing this class of pesticides with safer alternatives, even without compelling human evidence. But that did not happen, despite ongoing advocacy campaigns waged by a number of committed organizations. Pesticide manufacturers challenged the results of carefully designed studies at every opportunity and predicted soaring food prices and shortages if their OP pesticides were restricted.

While chlorpyrifos was finally banned from residential use in 2000, along with several other OP pesticides it remained in widespread use in agriculture. Farm workers, their families and rural communities continued to be exposed at higher levels than the general population. Now we have [compelling evidence](#) of damage to the developing brains of these children—harm that could have been avoided. It continues today because the administrator of the US EPA has [bowed](#) to the wishes of the pesticide industry, although many of the scientific uncertainties have been largely resolved.

This is not an isolated instance. The [record](#) is replete with examples of chemicals ultimately demonstrated conclusively to be reproductive or developmental toxicants, long after the first clues became apparent when they were already in widespread use.

A few years later, several of us explored data linking environmental factors to a few increasingly common disorders in later life with particular interest in cognitive decline and dementia. In addition to environmental chemicals and contaminants we considered interactions with genetic inheritance, diet and nutrition, physical activity, and social stressors, across the lifespan. Our publication, [Environmental Threats to Healthy Aging](#), used an [ecological model of health](#) to illustrate the complexity involved in both studying and responding to the many, multi-level factors and their interactions that influence health and the risk of disease. Similarly, in [The Ecology of Breast Cancer](#), I used an ecological model to help understand better the many variables that collectively influence the risk of this common cancer.

In each case, early clues and further studies began to identify risk factors, often in combination, more definitively linked to the outcomes—often decades later: Western-style diet and cognitive decline; lifetime lead exposure and cognitive decline; lifetime lead exposure and chronic stress even higher risk of cognitive decline; childhood lead exposure, dietary iron deficiency and poverty with more than additive damage to developing brains; higher early-life exposure to diethylstilbestrol (DES) or DDT and increased breast cancer risk decades later, and so on.

When is evidence sufficient to act? Who bears the burden of proof, responsibility, risks and benefits? What are the alternatives? These are the kinds of questions the PP poses that threaten established ways of decision-making.

What are some of the lessons learned from these diverse projects?

Single causes are primarily responsible for only a few common diseases—e.g. smoking and lung cancer—but these are the exception. Most common diseases or disorders are caused by combinations of multiple, multi-level risk factors.

Complex diseases are systems problems needing broad and diversified efforts to study and change dynamics of the system—not only in individuals but also in communities and populations. Single interventions are rarely as successful as combinations of strategic choices.

Individual behavioral change can help but is not enough for disease prevention in populations. Multi-level interventions—community and societal—are also essential. Some risk factors like polluted air and water, limited access to healthy food, or unsafe streets are designed into the fabric of a community or region and cannot be addressed by individual behavior change alone.

Understanding how to prevent and treat multifactorial diseases will always be fraught with uncertainties. Special economic interests routinely take advantage of uncertainties to protect their markets. Requiring absolute proof of harm before moving to safer alternatives is guaranteed to fail to protect public health and the environment when applied to multi-factorial diseases and environmental problems.

The precautionary principle uniquely combines science and ethics: What do we know? What are the uncertainties? What is at risk of harm if we act or fail to act? What should we do to protect health and the environment? Who gets to decide?

Established ways of making decisions with broad impacts on human health and the environment have not kept pace with current scientific understanding of systems problems, uncertainties and the undeniable reality of a rapidly-changing world of degraded ecosystems and growing population. In the second decade of the 21st century the precautionary principle forces us to confront vital questions and directs us to come to more health- and planetary-protective solutions with greater urgency than ever before.