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Four Suggestions for Improving Environmental Health Policy

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by Kenneth W. Chilton

Concern about the effects of environmental contaminants on Americans' health appears to be increasing even though pollution levels are decreasing. As one who has an occupation that places me in the role of a "consumer," or interpreter, of environmental health research, I have some thoughts about why this paradox persists. The four suggestions that I offer relate to the information provided by environmental health scientists and researchers to policy makers.

The four suggestions (or issues) discussed are:

- When analyzing environmental health risks from a particular "contaminant," the complete picture of risks and benefits of the agent should be considered.
- *All* elements of an environmental health risk assessment need to be grounded in solid science.
- To foster accurate communication of risks, the environmental health sciences community should respond to high profile statements that are incomplete or inaccurate.
- Environmental health risks should be placed in the context of more commonly experienced health risks.

These four proposals are hardly exhaustive of the set of suggestions that one might raise about important aspects of environmental health research that impacts public policy formation. Hopefully, they can stimulate discussion among environmental health scientists and policy makers, leading to improved communication and, ultimately, better public policy.

• When Analyzing Environmental Health Risks from a Particular Contaminant, the Complete Picture of Risks and Benefits of the Agent Should Be Considered.

Let me illustrate what I mean by the need for a more complete picture of risks and benefits of an environmental agent by using the example of risks to children's health posed by pesticides. While several reports from environmental groups played a role in changing policies controlling pesticide use, the seminal study was published by a highly respected body within the National Academy of Sciences.

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The 1993 report by the National Research Council (NRC), *Pesticides in the Diets of Infants and Children*, was a valuable analysis of the possible effects of pesticide residues on America's children.[1] The report examined quantitative and qualitative differences between adults and children with regard to toxicity of pesticides. Quantitative age-related differences in absorption, metabolism, detoxification, and excretion of xenobiotic compounds as well as physical and biological differences—body size and the maturity of body systems—were considered. Qualitative differences due to brief periods in early development of an organ system were also analyzed.

In addition, the NRC study examined differing exposures to pesticide residues between children and adults. Children one to five years old eat three to four times more food than adults in proportion to their mass. These young children also have less variety in their diets. A one-year-old drinks 21 times more apple juice and 11 times more grape juice and eats two to seven times more grapes, bananas, pears, carrots, and broccoli per pound of body weight than the average adult.[2] According to the NRC study, most of the differences in pesticide-related health risks result from this second factor—exposure—not physical differences between adults and children.

The report led to a chain of events that has placed children's health in the forefront of concerns about environmental contaminants. It had a major impact on the provisions of the Food Quality Protection Act (FQPA) of 1996.[3] FQPA requires that pesticides be tested for their effects on children and that pesticide residues be limited to levels that pose a "reasonable certainty of no harm." It then applies a tenfold added safety factor in cases where data about effects on children are unknown or uncertain. FQPA further requires that the cumulative risk posed by exposures to similar classes of pesticides be considered in setting residue levels.

But what about the *benefits* to children's health resulting from judicious use of pesticides? The NRC was not charged with examining this side of the equation and its report clearly acknowledges this fact. No explicit evaluation of benefits is called for when considering pesticide residues under provisions of FQPA.

Diet is reported to be one of the largest controllable risk factors for cancer. The rate of most types of cancer (lung, larynx, oral cavity, esophageal, stomach, colorectal, bladder, pancreatic, cervical, and ovarian) is roughly *twice* as high in the quarter of the population with the lowest intake

of fruits and vegetables as in the quarter with the highest.[4]

Synthetic pesticides are one of the key factors in producing an abundant supply of high-quality, low-cost fruits and vegetables. A Texas A&M study suggests that a 50 percent reduction in pesticide use on crops of nine fruits and vegetables (apples, grapes, lettuce, onions, oranges, peaches, potatoes, sweet corn, and tomatoes) would reduce yields by nearly 40 percent.[5] Former Food and Drug Administration Commissioner, pediatrician David Kessler, remarked on the occasion of the Clinton Administration's 1993 plan to reduce pesticide use:

> We are not saying that food is unsafe.…There is no reason for a scare.…There is no doubt that the benefits of fruits, vegetables and grains far outweigh the risks of residues of pesticides in these products.[6]

Public policies that restrict judicious use of these pesticides or potentially ban them based on the very tight standards on pesticide residues spelled out in the Food Quality Protection Act could actually *harm* children's health. As this example illustrates, there are public policy consequences to looking only at the harm that environmental agents might cause without also considering their benefits.

- ***All* Elements of an Environmental Health Risk Assessment Need to Be Grounded in Solid Science.**

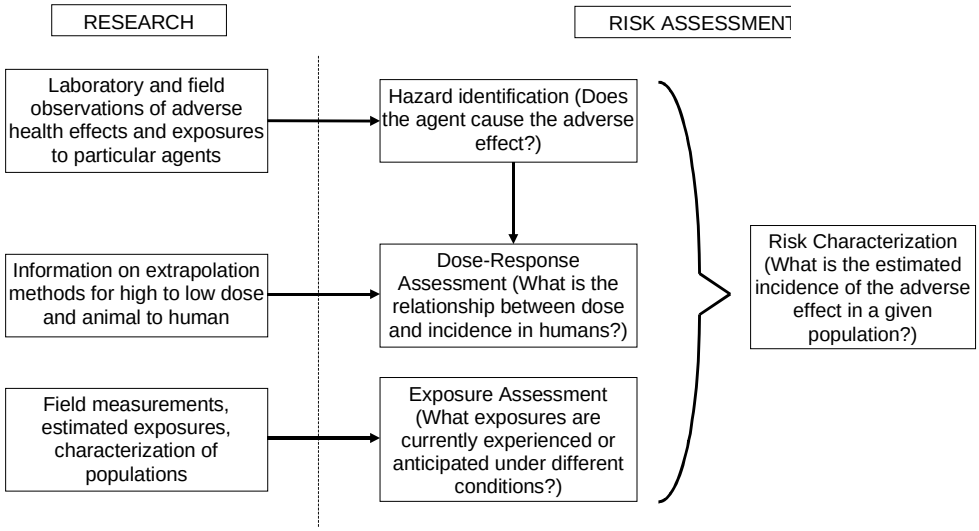
Figure 1 is a somewhat simplistic and dated, yet still serviceable, illustration of the risk assessment process. Of course, the process does not stop with risk assessment but proceeds to risk management. But if key elements of the assessment process are uncertain, the prescriptions to manage the risk may be overly stringent or, conversely, too lenient.

Hazard Identification

Each element of the risk assessment process has its own difficulties. Hazard identification typically relies either on animal studies using maximum tolerated dose results or epidemiological studies with many potentially confounding factors. The tendency by policymakers (risk managers) is to use epidemiological data when the results show an adverse effect and ignore them when they do not. There is also a bias toward findings of *harm*, discounting or ignoring findings that are inconclusive or don't show adverse effects.

Figure 1

Elements of Risk Assessment



Source: *Risk Assessment in the Federal Government: Managing the Process* (Washington, D.C.: National Academy Press).

The National Ambient Air Quality Standard for fine particles adopted in July of 1997 is an example of problematic hazard identification that, nonetheless, resulted in regulations to manage the risk. The epidemiological research pointing the finger at fine particles as a cause of premature death was quite recent. Raw data from the two key studies used to support a new standard for fine particles (2.5 microns in diameter) was not made available to other researchers to verify the results. Little distinction was made between premature deaths of days or weeks versus years or decades. The epidemiological research was not supported by toxicological findings—the biological mechanism for how fine particles cause deaths was unknown at the time.[7]

In July 2000, the epidemiological findings of the two key studies used to justify a new fine particle air quality standard were verified by an independent analysis.[8] The biological mechanism for the mortality effects is still being researched. Policymakers at EPA continue to refer to their projection of 15,000 premature deaths that could be avoided by meeting the new standard.[9] EPA makes no distinction between lives prolonged for weeks versus lives extended for decades.

The problem of bias toward research that shows an adverse health effect in hazard identification is exemplified by EPA's assessment of the benefits of reducing tropospheric ozone—smog.

The acute health effects of ozone have been amply demonstrated—wheezing, coughing, tightness in the chest, etc. Chronic effects, particularly premature death, have been suggested but epidemiological evidence has been elusive despite three or four decades of research.

Just as the EPA was finalizing a new eight-hour ozone standard in 1997, preliminary research suggesting that ozone might cause premature deaths began to be cited. EPA used this research to calculate the upper-bound estimate for benefits of the new ozone standard. More than 98 percent of the upper-bound benefit estimate results from valuing reduced mortality.[10] Decades of findings of no chronic effects were, more or less, discarded as a level of 0.08 parts per million was established for the new standard.

Dose-Response Assessment

Dose-response assessment—extrapolating from animal bioassays to predict cancer potency of a substance in humans, for example—is also problematic. Extrapolating from high-dose (maximum tolerated doses) physiological responses to low-dose responses introduces a 1,000 to 10,000 degree of uncertainty.[11] Pharmacokinetic behavior of the toxic agent (administered versus effective dose and issues related to maximum tolerated dose) further complicate dose-response assessment. In addition, the response of animals to mega doses over relatively short periods may not be representative of much smaller doses in humans over long time frames because of possible thresholds and/or repair processes.

Dose-response assessments may also be affected by uncertain animal-to-human scale-up measures (body weight, surface area, organ weights, receptor sites). Animal versus human exposure patterns (continuous versus intermittent, for example) are another factor. Finally, differences between mechanisms of carcinogenicity between humans and animals can affect this element of the risk assessment process.

Exposure Assessment

Exposure assessment may be one of the weakest links in the risk assessment process. While very significant resources are expended to attempt to determine what adverse health effects may be caused by specific agents—air and water pollutants, pesticides, etc.—and a good deal of research has gone into efforts to improve dose-response assessment, real world exposure assessments have

been relatively neglected.

Often, this step in the risk assessment process is simply modeled as a maximally exposed individual. This could be the exposure that a person would receive if she spent every hour of every day for 70 years outdoors at the fence line of an air pollution source or the amount of a toxic substance that a child could be expected to ingest if she played every day at a hazardous waste site and ate the contaminated soil.

To properly assess the risk from a contaminant, the exposure assessment needs to reflect the actual distribution of exposure levels of those populations likely to be affected. Because all persons are not equally responsive to a given contaminant, it is also important to properly represent these individual differences when modeling the affected population.

Exposure assessment problems are particularly evident for air pollution—fine particles and ozone, for example. How comparable are the *ambient* levels of these pollutants to actual human *exposures*? Research on actual exposures as opposed to assumed exposures is badly needed.

Elevated levels of ambient ozone and fine particles are associated with hot stagnant weather. During these episodes, children likely *decrease* outdoor exposures, preferring to stay indoors in air conditioning if it is available. If outdoor levels of these pollutants are highly correlated with indoor concentrations, then they are more credible suspects for adverse health effects. If not, then, as a nation we may not receive the public health benefits we hope for from a new round of costly air pollution controls.

Jesse H. Ausubel, Director of the Program for the Human Environment at Rockefeller University and former member of EPA's Science Advisory board, recommends expansion of EPA's National Human Exposure Assessment Survey (NHEXAS).[12] NHEXAS was launched in 1994 to "gather information about the magnitude, extent, and causes of human exposures to specific pollutants and measure the total 'dose' of selected pollutants."

Phase I measured actual exposures to a variety of pollutants of several hundred people who wore personal exposure monitors. Levels of chemicals in air, food, water and other beverages, and in the soil and dust around the participants' homes were also measured. Chemicals or metabolites in their blood and urine were tested. And the participants filled out time-activity questionnaires and food diaries to help pinpoint chemical exposures and activity patterns. Phase I sampling ran from

1995 to 1996.

Ausubel recommends that NHEXAS be greatly expanded beyond the pilot phase to produce "a comprehensive total human exposure database and models that accurately estimate and predict human exposures to environmental chemicals for both single and multiple pathways." This information on human activity could then be linked to environmental and biological data to produce better estimates of human exposures to various chemicals and combinations. Making exposure more of a science should increase understanding of aggregate exposures that may provide "surprisingly powerful levers to reduce ambient bads or increase goods," says Ausubel. In this regard, he suggests that NHEXAS "needs a less 'toxic' bias," less of a focus on toxic chemicals *per se*.

EPA is reviewing the NHEXAS data to help develop a continuous national database of exposure data, tentatively called the National Human Exposure Monitoring Survey (NHEMS). Members of EPA's Science Advisory Board Integrated Human Exposure Committee have urged the agency to push for funds for this project internally and with other federal agencies.[13] The panel's concern is well-founded given the timid approach to improving exposure assessment that federal environmental health agencies have taken over the past several decades.

- **To Foster Accurate Communication of Risks, the Environmental Health Sciences Community Should Respond to High-Profile Statements That May Be Incomplete or Inaccurate.**

What responsibility do environmental health researchers have to correct inaccurate statements that have policy consequences? For example, while researching the significance of environmental contaminants as threats to children's health, Stephen Huebner and I read quite a few statements about cancer risks that were misleading. [14]

At EPA's September 15, 1997 Conference on Preventable Causes of Children's Cancer, Administrator Carol Browner acknowledged that the death rate from childhood cancer has declined dramatically but went on to say:

> But an equally dramatic rise in the overall number of kids who get cancer threatens to overshadow the gains we have made.
>
> In the past two decades, we have seen higher rates of acute lymphoblastic leukemia in children, higher rates of a type of brain cancer in children, and higher

> rates of Wilms' Tumor of the kidney.…And we don't know exactly why.
>
> Many leading health experts suspect that toxins found in our environment may very well play a role in the growing incidence of certain childhood cancers. The world that our children are born into now includes tens of thousands of new chemicals that simply were not around just a few decades ago—substances that are present in our air, in our water, in our homes, in our foods.[15]

Until about November 1998, the Web site for EPA's Office of Children's Health Protection (OCHP) cited cancer rates for the under-15 age group compiled in the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) database in order to buttress the agency's statement that childhood cancer rates for some types of cancer are on a dramatic rise.[16] The OCHP fact sheet on children's health stated that, for the 1973 to 1994 period, the incidence of acute lymphocytic leukemia was up by about 5 percent, brain cancer up about 40 percent, and Wilms' tumor up by about 46 percent.

But these findings were fundamentally flawed for two reasons: (1) they were based on a simple end-point analysis comparing incidence rates for 1973-1974 to those for 1993-1994; and (2) they provided no sense of the baseline frequency of these cancers. The correct way to analyze this data is by ordinary least squares regression to determine if there is a significant trend taking place rather than just a good deal of random variation in the data.

When the analysis is done properly, it shows that acute lymphocytic leukemia (ALL) is increasing at a rate of just 0.3 added cases per million birth–fourteen-year-olds each year (1973 to 1995). The base rate for ALL in 1973 was about 27 per million for this age group. Childhood brain cancers rose at an annual rate of 0.5 cases per million over the period. The 1973 base rate was 24 cases annually per million children. Kidney and renal pelvic tumors (a broader category than Wilms' tumor) shows no trend. These types of cancers occurred at a rate of just seven per million children in 1973.

EPA's misinterpretation of the SEER data did not go unnoticed by the keepers of that data—the National Cancer Institute (NCI). According to the December 1999 issue of *Science*, Richard Klausner, director of NCI, was "thunderstruck" by an article in the 29 September 1997 *New York Times* that stated that "the rate of cancer among children has been rising for decades."[17] The article further suggested that "growing exposure to new chemicals in the envi-

ronment" might be the cause. Klausner found that the source for this claim was the EPA's conference on preventable causes of cancer in children—an event that did not include any of NCI's scientists. He tried to reach EPA Administrator Carol Browner, but it was weeks before she returned his call. Klausner said, "I was concerned about an injudicious description of the trends."[18]

In response, NCI sped up its already-planned review of childhood cancer rates. This more detailed analysis of the SEER data by Martha S. Linet and her colleagues at the National Cancer Institute reached the following conclusion:

> There is no substantiated change in incidence for the major pediatric cancers, and rates have remained relatively stable since the mid-1980s. The modest increases that were observed for brain/CNS cancers, leukemia, and infant neuroblastoma were confined to the mid-1980s. The patterns suggest that the increases likely reflected diagnostic improvements or reporting changes.[19]

The point is that risk management policies may be based on this type of inaccurate information. If that is the case, shouldn't environmental health scientists feel an obligation, as did Richard Klausner, to correct high-profile statements that are off the mark?

- **Environmental Health Risks Should Be Placed in the Context of More Commonly Experienced Health Risks.**

What risks should people, especially parents, be worried about? Childhood asthma risk, for example, is a serious problem but not a leading cause of childhood deaths.

"Chronic Obstructive Pulmonary Disease" doesn't make the top 10 causes of death for one- to four-year-olds; there were fewer than 30 asthma deaths in this age group in 1997. It is the number eight killer for five- to fourteen-year-olds—129 deaths, or less than 2 percent of the total (see Table 1). In comparison, accidents and adverse effects account for 37 percent of the deaths in one- to four-year-olds and 42 percent for five- to fourteen-year-olds. In testimony before a Senate Labor and Human Resources Committee, Dr. C. Everett Koop, former U.S. Surgeon General, claimed that 90 percent of childhood injuries and deaths are preventable.[20]

Congenital anomalies (birth defects) caused 589 deaths among one- to four-year-olds in 1997 and 447 deaths in the five to fourteen age group. In this case, also, many of these deaths may be preventable.

Table 1

Ten Leading Causes of Death in 1997 for Children Ages 1-4 and 5-14

Ages 1-4	Annual Deaths	Rate per[1] 100,000	Ages 5-14	Annual Deaths	Rate per[1] 100,000
1. Accidents and adverse effects	2,005	13.1	1. Accidents and adverse effects	3,371	8.7
2. Congenital anomalies (birth defects)	589	3.8	2. Malignant neoplasms (cancers)	1,030	2.7
3. Malignant neoplasms (cancers)	438	2.9	3. Homicide and legal intervention	457	1.2
4. Homicide and legal intervention	375	2.4	4. Congenital anomalies (birth defects)	447	1.2
5. Heart disease	212	1.4	5. Heart disease	313	0.8
6. Pneumonia and influenza	180	1.2	6. Suicide	307	0.8
7. Conditions originating in perinatal period	75	0.5	7. Pneumonia and influenza	141	0.4
8. Septicemia	73	0.5	8. Chronic Obstructive Pulmonary Disease	129	0.3
9. Benign neoplasms, carcinoma in situ	65	0.4	9. Human Immunodeficiency Virus	102	0.3
10. Cerebrovascular diseases	56	0.4	10. Cerebrovascular diseases	76	0.2
			Benign neoplasms	76	0.2
Other causes	1,433	9.3	Other causes	1,612	4.2
All causes	5,501	35.8	All causes	8,061	20.8

[1]Age-adjusted death rate per 100,000 children in this age group.

Source: Hogert, D.L., Kochanek, K.D., Murphy, S.L., *National Vital Statistics Report*, vol. 47, no. 19 (Hyattsville, Md.: Centers for Disease Control and Prevention National Center for Health Statistics, June 30, 1999): 27.

About 1 in 1,000 infants are born with either spina bifida or anencephaly, affecting about 4,000 pregnancies a year. The Centers for Disease Control and Prevention (CDC) estimates that about three-fourths of the pregnancies affected by these birth defects could be prevented if women consumed an adequate daily dose of folic acid before and during early pregnancy.[21] The Food and Drug Administration and the Public Health Service worked with U.S. food processors to add folic acid to enriched breads, flours, pastas, etc. to reduce this risk. Fetal alcohol syndrome affects approximately 2,000 babies a year and, obviously, is preventable.[22]

Malignant neoplasms (cancers) are the number three killer of one- to four-year-olds but are only slightly more common than homicides—2.9 deaths per 100,000 versus 2.4 deaths per 100,000. In the five to fourteen age group, cancers are the second leading cause of death, more than twice as

common as homicides or birth defects but less than one-third the fatalities due to accidents.

Moreover, the portion of cancer deaths among all age groups thought to be attributable to environmental contaminants is quite small. Interestingly, the OCHP fact sheet previously mentioned cited a 1981 paper by Richard Doll and Richard Peto which estimates that exposure to environmental pollutants may be responsible for 1 to 5 percent of all cancer deaths (including children's).[23] A 1996 paper by Doll concludes:

> Pollution, which is popularly thought to be a major hazard, must cause some cases, but the risks that can be quantified—those of polycyclic aromatic hydrocarbons, trace metals and benzene from the use and combustion of fossil fuels in industry and transport, dioxins from the combustion of waste, pesticide residues in food and discharges from the nuclear industry—all appear to be so minute that the social cost of trying to reduce them further may well outweigh any benefit from reduction in the incidence of cancer.[24]

A recent report from the Harvard Center for Cancer Prevention estimates that just 2 percent of cancer incidence in the United States may be caused by environmental pollution. Food additives and contaminants may explain another 1 percent of cancer cases, but it is salt, not pesticide residues, that is the cancer risk (stomach cancer).[25]

Clearly, childhood cancer is not of epidemic proportions. Moreover, we should not expect to see significant improvements in its rather modest baseline incidence or mortality rate by focusing on reducing environmental contaminants.

The point is that to improve children's health, in this case to reduce childhood deaths, shouldn't greater attention be placed on the largest threats, especially those that are most easily prevented? Of course, this is not an "either/or" proposition; we can address accidents and birth defects at the same time that we research the causes of asthma and cancer incidences and deaths. But if we want to get the attention of the public, especially parents, to get them involved in preventable risks, we might consider more emphasis on accidents and birth defects whenever children's health issues are discussed.

Summary

Environmental health risks continue to be a concern to the American public despite improving environmental conditions. This is due in part to the way in which news reporting favors bad news

over good news. Environmental groups also are often prone to exaggerate environmental health risks in an effort to mobilize the public. Nonetheless, addressing the four issues raised in this paper could help remedy some of these misperceptions and lead to better environmental health policies.

Research on environmental contaminants is largely driven by laws and regulations that seek to reduce their public health impacts. As a result, the research can take on some of the biases embedded in the statutes. We do not get a complete picture of the risks *and* benefits of a particular environmental contaminant because research is skewed toward identifying risks only. The solution to this problem is to anticipate the needs of the risk manager (policymaker) to utilize the fruits of the risk assessor's labors. Researchers need to take a broad approach to evaluating environmental health risk; one that considers potential benefits as well as harms from so-called contaminants.

The quality of environmental health risk assessments is improving, but each element of a risk assessment has its uncertainties. Hazard identification suffers from a bias toward findings of harm. Results that find no effect are not given the same credence as positive results. Dose-response assessment is most plagued by the problems of administering continuous maximum tolerated doses to relatively short-lived animals and extrapolating the results to low intermittent doses experienced over long human lifetimes. But exposure assessment—the actual exposure likely to be experienced by various sensitive populations—has been the most neglected aspect of environmental health risk assessments thus far.

The Environmental Protection Agency may now be poised to use the results of its pilot National Human Exposure Assessment Survey (NHEXAS) launched in 1994 to go forward with a more ambitious National Human Exposure Monitoring Survey (NHEMS). Though NHEMS is an important program, it suffers from a disadvantage in competing for funding. Medical schools and other non-profit research organizations are quite experienced at garnering funding for programs to further hazard identification and dose-response assessment but are not necessarily the logical entities to conduct exposure assessments.

To improve public perceptions of environmental health risks, more accurate information needs to be communicated. Unfortunately, many pronouncements by high-level government officials,

and especially statements by environmental leaders, are less than accurate. Though not a task relished by environmental health experts, it is incumbent upon these professionals to attempt to correct the record on these misstatements. A good example of taking this responsibility seriously was provided by the recent analysis of childhood cancer incidence by the National Cancer Institute. Though EPA has not publicly recanted its earlier inaccurate statements about dramatically rising trends in certain childhood cancers, it has removed some of these misstatements from the Web site for its Office of Children's Health Protection.

Very little has been done to place environmental health risks in some type of context. But if the objective is to protect public health and not to foster one aspect of research into health risks, environmental health risks need to be compared to other more common health risks. Children's health, in particular, could be harmed, not helped, if public attention is drawn away from preventable and prevalent harms from accidents to focus on less common threats such as the very tiny fraction of cancer cases and deaths that can be attributed to environmental pollution and contaminants.

Research on health risks from environmental contaminants is progressing on many fronts. But in the process, some very basic issues are receiving less attention than they deserve. The four suggestions discussed in this policy brief are offered in the hope that environmental health researchers and policy makers will use them as a starting point for addressing some of the weaknesses in current environmental health policy.

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