Comments on the Environmental Protection Agency's Draft "Guidelines for Carcinogen Risk Assessment" and the Draft "Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens"

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Overview

The Environmental Protection Agency (EPA) apparently has as one of its current goals the identification and risk assessment of chemicals in the environment that pose a risk of human cancer. Further, the EPA has stated its intention to identify and assess the risk of chemicals in the environment that pose a particular risk to children who may be more vulnerable to the effect of environmental chemicals than are adults, and once exposed may face a higher risk of cancer later in life.

The apparent purpose of this attempt at identifying "carcinogenic" chemicals, and assessing the risk they allegedly pose to children, seems to be to provide a rationale for further EPA regulatory efforts which purport to reduce the risk of cancer in children in the United States.

But the proposed EPA "cancer prevention" proposal is inherently flawed because it is based on assumptions that have no basis in scientific reality. Most important, the EPA proposal to identify "carcinogens" and quantitate the risk they pose is predicated on the belief that trace levels of chemicals in the environment pose a risk of human cancer and that the risk posed by these "chemical carcinogens" is particularly devastating for children. Given that there is no evidence that exposure to trace levels of chemicals in the environment whether they are "carcinogens" or not contributes to the toll of human cancer in the United States, it is ludicrous to attempt to quantitate the level of a risk that is not known to exist. Further, it is counterintuitive to argue, as the EPA does, that the risk of cancer in those exposed to these environmental chemicals is "up to 10 times greater in children than in adults." Obviously, if the risk posed to adults is zero, the postulated "elevated" risk for children would be ten times zero: also zero.

What Causes Human Cancer?

The science of cancer epidemiology has provided the data that support our current understanding
of the causes or risk factors for human cancer. Cancer epidemiology involves the study of cohorts of humans to identify variables that may be associated with increased or decreased risks of different forms of cancer. In some cases (prospective studies), cohorts of individuals who are free from cancer are followed to learn if there are differences in cancer rates between those exposed to a possible cause (such as cigarette smoking) and those who are not exposed. In other cases (retrospective studies), individuals with a specific form of cancer are studied to learn if they have a greater exposure than do non-cancer patients to a specific agent.

The science of cancer epidemiology has identified eight known categories of cancer causes/risk factors. These are:

1. Use of tobacco, particularly in the form of cigarettes that are regularly inhaled.

Tobacco use, particularly cigarette smoking, is causally linked to cancer of the lung, bladder, pancreas, cervix, esophagus, and other sites. Indeed tobacco use is now causally linked with at least one-third of cancer deaths annually in the United States.

2. Alcohol use in connection with tobacco use. The synergistic effects of alcohol and tobacco account for a substantial portion of the esophageal cancer diagnosed in the U.S. each year. Excessive consumption of alcohol may also be causally linked with liver cancer.

3. Overexposure to sunlight (ultraviolet rays), which is the leading preventable cause of melanoma and various superficial skin cancers.

4. Excessive exposure to radiation.

5. Exposure to certain pharmaceuticals, including DES and, in some circumstances, post-menopausal estrogen replacement therapy.

6. Occupational exposure to certain chemicals and substances, including vinyl chloride, aniline dyes, and asbestos.

7. Sexual and reproductive patterns. (Having multiple sexual partners increases the risk of cervical cancer; not having children or having a first child relatively late in life increases the risk of breast cancer.)

8. Specific dietary-related patterns (obesity in post-menopausal women increases the risk of breast cancer and uterine cancer; there is some evidence that diets high in fruits and vegetables offer protection from some forms of cancer).

The establishment of the above eight causes/risk factors is based on data from human studies.

It is critical to note that human exposure to trace levels of environmental chemicals is NOT on the list of known risk factors for cancer. Thus, in attempting to identify and assess the degree of risk for adult or childhood exposure to trace levels of environmental chemicals, the EPA is focused not on an established but rather on a hypothetical risk of human cancer.
It is also worth noting that given the above facts about cancer causation, it is clear that the mandate of the EPA does not extend into control of ANY of the known causes of human cancer. For example, the EPA has no regulatory authority over tobacco (with the possible exception of exposure to secondhand smoke), alcohol, diet, pharmaceuticals, sunlight, most aspects of radiation exposure, or sexual/reproductive patterns.

On what basis does the Environmental Protection Agency hypothesize that trace environmental chemicals pose a human cancer risk and then go on to further hypothesize that children are more vulnerable to this risk?

The EPA relies on laboratory animal studies, in which chemicals, when ingested or inhaled by animals at high dose, reveal higher than expected rates of cancers. ACSH has written extensively on the limitations of extrapolation from animal testing in predicting human cancer risk (See "Of Mice and Mandates: Animal Experiments, Human Cancer Risk, and Regulatory Policies [1].")

In summary, these limitations include:

a) the very high doses of chemicals used in laboratory testing may themselves causes mutagenic/carcinogenic effects; and

b) substances that increase the risk of cancer in laboratory animals abound in nature.

Indeed, the overwhelming majority of the animal carcinogens to which we are exposed daily are natural in origin and thus outside the regulatory reach of the EPA.

Thus, in evaluating EPA's approach to identifying "carcinogens" in the environment and measuring the extent of the risk these pose to adults and children, ACSH concludes that the approach is inherently flawed because it focuses on a purely hypothetical risk.

ACSH strongly recommends that before EPA proceeds any further with its pursuit of "environmental carcinogens" and its argument that children are "more vulnerable," the Agency seek the opinion of scientists at the National Cancer Institute (NCI). The NCI is the foremost authority on cancer etiology in the United States and perhaps the world. Unlike the EPA, the staff of the NCI is replete with physicians and scientists whose expertise is cancer epidemiology the study of the causes and distribution of human cancer.

Specifically, EPA should ask the NCI scientists these questions:

**DO YOU KNOW OF ANY EVIDENCE THAT HUMAN EXPOSURE TO TRACE LEVELS OF SYNTHETIC OR NATURAL CHEMICALS IN THE ENVIRONMENT POSES A RISK OF HUMAN CANCER?**

**DO YOU KNOW OF ANY EVIDENCE THAT SUCH EXPOSURES CONTRIBUTE TO THE TOLL OF HUMAN CANCER IN THE UNITED STATES?**
If, as ACSH predicts, the NCI responds to the effect that there is no evidence that trace levels of environmental chemicals pose a risk of human cancer, it will become evident that the current EPA plan to undertake "risk assessment" for trace chemical "carcinogens" is a pursuit without any scientific merit a pursuit that will have no impact whatsoever on the reduction of cancer risk in either children or adults.

For additional commentary, please see "Are Children More Vulnerable to Environmental Chemicals? Scientific and Regulatory Issues in Perspective [2]."