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Cancer Risks From Synthetic Pesticides In Perspective

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Think about the word "carcinogen." What pops into your mind? Pesticides? Smoking? Asbestos? Pesto? There probably aren't too many people who actually thought "pesto," but it would fit into the category. Basil, the primary ingredient of pesto, is loaded with estragole, which is responsible for that unforgettable flavor and is a rodent carcinogen.

Surprised? Well, hold onto your seat, basil is just the beginning. Mustard contains allyl isothiocyanate; comfrey contains symphytine; and mushrooms contain various hydrazines, all of which are carcinogens with higher potencies in rats or mice than poly-chlorinated biphenyls (PCBs). It seems that virtually every plant contains natural compounds that act as protection against insects, microorganisms, and herbivores. About half of these compounds tested so far have proven to be carcinogenic, at least in rats or mice. And we eat about 10,000 times the amount by weight of these natural pesticides as we do synthetic pesticide residues.

There are many other naturally occurring sources of carcinogens in our daily lives. Many carcinogens are created during cooking. Charred meat, browned bread crusts, and fermented products are major sources of carcinogens in the diet. Our body's own metabolism is responsible for releasing carcinogenic oxygen radicals. The large number of naturally occurring carcinogens is likely to overwhelm any effect from the small amounts of synthetic pesticides we consume. While this doesn't mean that cancer risks from synthetic pesticide residues should be ignored, it does put them into perspective. Is it necessary to panic about a trace amount of ethylene dibromide (an agricultural fumigant) when the pesto packs a stronger punch?

Another thing to remember is that when we consume synthetic pesticide residues and natural pesticides, we generally consume them on or in fruits and vegetables. Fruits and vegetables also contain anti-carcinogens, including vitamins E and C and beta-carotene. These anti-carcinogens, combined with your body's defenses, protect against both natural and synthetic carcinogens.

Of course, it makes sense to eliminate the largest risks from one's diet and environment. To do that, some measure of carcinogenicity is necessary. Unfortunately, most of the data is for rodent, not human, carcinogens. In fact, it is impossible to calculate human risk based on a rodent exposure, but we may be guided in our decisions by rodent data. Prudence would dictate limiting those substances that are highly potent rodent carcinogens.

Bruce Ames of the University of California and his colleagues have developed a scale for comparing carcinogens based on human exposure and rodent carcinogenicity. While this scale does not allow for actually calculating risk, it does serve to point out compounds that may be of greater concern than others. The greater the human exposure to the rodent carcinogen or the greater the potency of the carcinogen in rodents, the higher the human exposure/rodent potency ratio (HERP).

Table 1

HERP	*Daily human exposure percentage*
140	EDB: workers' daily intake, high exposure
5.8	Formaldehyde: workers' average daily intake
6.2	Comfrey-pepsin tablets (9 daily), from comfrey root
2.8	Beer (12 ounces) from ethyl alcohol
0.1	Mushroom, one raw (15 g) from hydrazines
0.1	Basil (1 g of dried leaf) from estragole
0.07	Brown mustard (5 g), from allyl isothiocyanate
0.03	Peanut butter (32 g, one sandwich) from aflatoxin content of 64 mg
0.03	Comfrey herb tea, 1 cup
0.003	Bacon, cooked (100 g)
0.0004	EDB: daily dietary intake, U.S. average
0.0003	DDE/DDT: daily dietary intake, U.S. average
0.0002	PCBs: daily dietary intake, U.S. average
2.1	Mobile home air (14 hours/day)
0.6	Conventional home air (14 hours/day)

Looking at this data, one can see that a can of beer exposes you to a larger amount (based on body weight) of a rodent carcinogen than does your average intake of PCBs, DDE/DDT, and ethylene dibromide combined. The formaldehyde in conventional and mobile home air results in much higher HERP's than do the chemical residues listed.

Remember, it is impossible to say, based on the data available, that a synthetic pesticide is more or less likely to cause cancer than a natural pesticide. The lower exposure rates as seen in Table 1 suggest less of a risk from synthetic pesticide residue, but don't prove it. It does seem reasonable to conclude that occupational exposures to high concentrations of synthetic chemicals and indoor air pollution may be a much greater risk than either natural or synthetic pesticides in the diet.

It appears that we continually are bombarded by carcinogens in our diet and have been for all of our history. Remembering this may help us keep our perspective on the carcinogenicity of pesticide residues, especially when there is much that could be done to reduce major sources of cancers, such as smoking and occupational exposure. Relatively speaking, pesticide residue is a minor cause of cancer and may even be irrelevant when considering the vast number of natural pesticides with similar or greater potencies.

Reference:

Ames, B.N., R. Magaw, and L. Gold. Ranking Possible Carcinogenic Hazards. Science 236:271-80.

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