

Insecticides: Environmental Fate and Toxicity

An initial warning: Be fair and cautious in what analogies you use to represent low concentrations ...

Mackay, D. 1988. On low, very low, and negligible concentrations. *Environmental Toxicology and Chemistry* 7: 1-3.

Mackay notes that many people like to portray low concentrations as negligible by using analogies that minimize ...

- 1 ppm =
- 1 ppb =

...but if a cubic meter of a solid or liquid contains 10^{28} molecules...

- 1 part per quadrillion = 10 billion molecules

Mackay referred to this as "the enormity of tinyess."

He also offered some more understandable analogies ... analogies that can be visualized:

In a cubic meter of space:

- 1 ppm =
- 1 ppb =
- 1 ppt =

Mackay argued that the significance of low concentrations depends on how the chemicals in question act in an organism.

- "Disruptives"... low concentrations may be negligible
- "Distributives"... partitioning among media may magnify concentrations
- "Directives" ... if the chemical damages DNA for example, a single or a few molecules at the "right" place might be enough to cause injury

Reasons for concerns about pesticides in environmental quality and human health result from:

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Persistence is one determiner of the magnitude of residues in soil or on foods. Persistence can be represented by determining a pesticide's half-life. Half-lives in soil for several insecticides:

- DDT
- Heptachlor
- Chlordane
- Ethyl parathion
- Chlorpyrifos
- Diazinon

Ranking persistence (in a very general way):

Longest

- Inorganics such as lead arsenate
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Medium

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Shortest

- Botanicals
- Soaps
- Microbials

Rates of breakdown are dependent on:

- concentration (extremely high concentrations degrade more slowly)
- temperature and moisture (increasing levels of either tend to speed breakdown)

- pH (organophosphates especially ... alkaline conditions speed hydrolysis, even in the spray tank)
- UV light speeds breakdown (especially for microbes)

Breakdown products (metabolites) can themselves be persistent & toxic ...

- aldrin to dieldrin; heptachlor to heptachlor epoxide ... metabolite is more persistent and more toxic
- Alar (daminozide) to UDMH ... a carcinogen by current standards (apple story of 1980s)
- Aldicarb to aldicarb sulfoxide (metabolite is more toxic than the original active ingredient) (watermelon story of 1990s)

Transport

Residues may be carried away from application sites, often to unwanted destinations. Transport in/by water is influenced by persistence, water solubility, and soil sorption (K_{OC}):

Compound	Soil Half-life	K_{OC}	Water Sol. (ppm)
DDT		--	0.006
chlordane		--	0.1
parathion (methyl)	5 days	9800	57
chlorpyrifos (Lorsban)	30-90 days	4600	1
terbufos (Counter)	21-35 days	578	5
aldicarb (Temik)	70 days	28	6,000
carbofuran (Furadan)	30-90 days	45	320
carbaryl (Sevin)	10 days	230	40
permethrin (Pounce, Ambush)	30 days	10,600	0.04
esfenvalerate (Asana)	35 days	5,300	0.002
atrazine	60 days	100	33
alachlor	15 days	170	242

In general, the values that trigger some concern about a pesticide's potential for environmental transport are a **half-life greater than 21 days**, a **soil sorption index of 300 to 500 (or less)**, and a **water solubility of greater than 30 ppm**. Triggering one or more of these concerns does NOT mean that a pesticide should not be used at all; it simply means that uses should be appropriate.

So ... Certain pesticides end up in ground water and surface water for specific reasons.

Compounds most common in groundwater detections are

- old chlorinated compounds
- aldicarb in a few areas (not IL)
- nitrates
- the herbicides atrazine, metolachlor, alachlor, and a few others.

Reasons:

Low solubility / high soil sorption do not prevent surface water contamination

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Risks of unwanted transport at mixing and loading sites (and toxic waste sites) are high for all compounds regardless of sorption, solubility, or normal persistence. High concentrations outweigh other characteristics. Some related issues to consider ...

- Locations of ag chem facilities (and other point sources of various contaminants) in relation to community water wells
- Location and construction of farm wells and mixing/loading practices
- "Land-farming" to dispose of contaminated soils

Toxicity: the ability of a compound to cause injury or death

Oral LD₅₀ values for:

Pesticide	mg/kg
DDT	113-118
chlordane	457-590
methyl parathion	14
chlorpyrifos	135-163
terbufos	2-5
malathion	885-2800
aldicarb	1
carbaryl	850
carbofuran	8-14
permethrin	430-4,000
rotenone	60-1500
nicotine	50-60
sabadilla	4,000
pyrethrins	1200-1500
microbials	NA

LOW numbers indicate GREATER toxicity!!

LD₅₀ values are not complete indicators even for acute toxicity.

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OBVIOUSLY ... Environmental toxicity is also an issue ... toxicity to fish (pyrethroids, rotenone, many others), bees (carbaryl, many others), birds (DDT, now Furadan), and plants (lead arsenate, others) are all concerns.

Pesticides as carcinogens ... many have been identified

In Ames et al. 1987 and additional readings (though in many ways little has changed in terms of applying research on carcinogenicity to pesticide regulation in the last 20 years) ...

- Cancer tests use maximum tolerated doses (MTD's) as first screen. Does this constant high dose cause different effects than what we should expect from occasional low doses? Are there threshold doses below which injury would not occur?
- Ames' bacterial mutagenicity test: Lots of positives among natural and synthetic compounds. Did this mean all those natural compounds really are carcinogens?
- Data (relatively few) that exist from animal trials on the carcinogenicity of suspect natural compounds show about the same percent positives as animal trials on synthetics. Do the samples (trials) represent the populations of compounds?

Possible conclusions:

- Ames and others in this camp are wacko, wrong, paid off, or misdirected.
- Lots of compounds really are carcinogens. (And there's no need to add more synthetic ones.) OR (And the synthetic ones are negligible additions with useful roles.)
- The way we identify carcinogens is greatly flawed. (So what's a better way and what do we do until we improve the protocol?)

Erroneous logic:

- Humans evolved in the presence of natural compounds; they are therefore safer. (Consider that tests of carcinogenicity are done on rodents and that they too evolved in the presence of natural compounds. Also consider that cancer remains for the most part a disease associated primarily with aging ... how much impact on the evolution of a species?)
- All known human carcinogens also cause cancer in high-dose rodent studies, so all compounds that cause cancer in high-dose rodent trials must be human carcinogens.
- A ppb just isn't going to cause any effect.

Broader Conclusions:

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