Connections: Communicating Risk Assessment

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Just this week, I've fielded questions on CCA-treated wood, car exhaust fumes and lead — all in relation to the safety of growing food in urban areas. The conversations prompted me to revisit a topic for this month's column that I've addressed before —risk communication. So here is what I call Risk 101, basic tools to communicate the risk assessment process.

The first and foremost concept in risk assessment is the toxicity of the compound in question. If something is highly toxic, a dab will do you (in, that is). Not so toxic, you are talking about sitting down with bigger and bigger portions. The "technical" term for this is LD50 — the quantity of a compound that it takes to kill 50 percent of the target organism exposed to the compound.

The vast majority of compounds that we are concerned about in the organics recycling industry have nothing to do with acute toxicity. No cases of your hand falling off when you stick it in the compost pile unless you do it when the turner is going down the row. (The term for that is stupidity rather than toxicity.) We are concerned with contaminants from a chronic exposure perspective, which brings us to the famous equation of risk assessment:

Toxicity = Dose x duration of exposure.

What does this equation mean? I like to think about things in terms of food. Dose refers to how many strips of bacon you eat in one sitting. Exposure is a measure of how many mornings per week leading to per month and then eventually per decade that you actually eat bacon. In other words if once a year you gorge yourself on bacon but abstain the rest of the time, you have a high dose but a very short exposure. If, on the other hand, you feel like your one slice a day is a small enough portion that you are entitled to eat that bacon every day of the week and have done so for years, you have a low dose but very high duration of exposure.

If you are anxious about the acute toxicity of a compound, that means you are sitting there in front of that mound of bacon, sure that as soon as you eat it your cholesterol will shoot right up and you will have a heart attack and die before you've even finished chewing. If you are worried about chronic exposure, you are sure that even though you limit the amount of bacon that you eat, each piece will add just a little more plaque to your arteries and that over time, this will gradually increase your cholesterol to the point where your doctor will order you to go on a tofu only diet or else.

The next important concept in risk assessment is route of exposure, often referred to as the exposure pathway. This is further complicated when the site of biological response is introduced into the discussion. Yet again, food analogies save the day. If ice cream stays in the freezer it does no one any harm (it also gives no one any pleasure but that is another story). Say you are weak like me, however, and eat the ice cream. For ice cream, direct ingestion, aka eating it, is the primary exposure pathway of concern. Dermal contact just melts it. Inhalation? For me ice cream is much more about the taste than the smell. In other words, for every contaminant of concern, it is really important to realize that it will likely have limited ways it can cause harm. You need to identify those ways or pathways and figure out which are realistically causes to worry. Like I said, the ice cream in the freezer isn't doing anyone any harm.

So we all agree that for ice cream the ingestion pathway is the critical one. Note though, that while the ice cream enters the system through the ingestion pathway, the harm is not caused so much in the stomach, but rather on top of the stomach, or potentially in the thighs. Ice cream enters the receptor through the digestive tract, but the site of biological response is the roll of fat that has accumulated outside of my digestive system

proper and makes closing my pants problematic.

Uncertainty Of A Biological Response

The compounds that are generally the focus of public concern are associated with subtle changes rather than mortality. From my discussions with people, it seems that a lot of the worries stem from uncertainty about the potential for a biological response. This uncertainty is based on the fear of yet another ill effect lurking in the shadows from products that are found everywhere in our homes. Endocrine disrupting compounds (EDCs) are a great example. And now it turns out that, just like ice cream, they also may be partially responsible for the obesity epidemic in the U.S. In a recent New York Times column, Nick Kristoff cited research that showed that very low rates of exposure to chemicals including pthalates and bisphenol A (two common ingredients in plastics) have been shown to cause obesity in mice. This is very scary. Here we have an unexpected biological response with who knows how many yet to be determined biological responses waiting to be discovered. This is an example of a negative effect as a result of chronic exposure to a contaminant at extremely low concentrations (1 part per billion in the child or receptor).

The public reaction when they find out that there are EDCs in compost, reclaimed water or biosolids is often certainty that the threat from these compounds outweighs any benefits resulting from using the products. A consideration of exposure pathways is not figured into this certainty. If we compare these EDCs to ice cream, it is like being sure that you will gain weight if there is even a trace of ice cream in the freezer, whether or not you actually eat it. Or being sure that you will gain weight if you grow tomatoes with compost that had ice cream as part of the feedstocks. It is easy to recognize those apprehensions as silly. And there is a growing body of science that documents that the concerns about EDCs in organics and soils are pretty similar to my assessment of ice cream as a compost feedstock.

Understanding exposure pathways can do a lot to alleviate these worries. Respect these concerns as you explain the pathway approach in risk assessment. This is scary stuff to many people and not taking their unease seriously is a great way to turn people off your products.

The basic point to communicate in this example is that bisphenol A in the compost is not the same as bisphenol A in the ice cream container. Bisphenol A in the compost or the soil (while I am using this particular compound as an example, the same can be said for the vast majority of the compounds that are classified as endocrine disruptors) will most likely be degraded into CO2 by soil microorganisms in a matter of days or weeks, or months at the longest.

So can the bisphenol A in the compost cause you harm? Remember harm here comes after long-term exposure. Even if it was volatile and had the potential to make the compost smell like nail polish, it is in such low concentrations in compost products that you wouldn't be able to inhale any. Touching the soil won't do it either. And remember that even if it did, with chronic exposure as the issue, that would mean arms deep in the soil for long periods of time. Eating the soil, day after day, month after week is the big concern — but the palatability of the soil is the easy control on that.

That leaves you with it being taken up by plants and you eating the plants or it moving to groundwater and you drinking the groundwater. And research is showing no plant uptake or plant uptake only when lots and lots of extra of the compound are in the soil system. The same is true with movement to ground or surface waters. The next point to remember here is that if these compounds weren't so ubiquitous in household products, they wouldn't be showing up in the compost to begin with. Limiting use in the home will limit exposure — and reduce concentrations in organics.

Hopefully this has been helpful. All the writing has left me with an appetite though — time to go raid the freezer.

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