Pesticide Exposure Routes and Risks

It's important to understand how pesticides are engineered to enter their target pest. The active chemicals are bound to both water soluble and fat-soluble surfactants so they enter the pest in multiple ways. The water-soluble portion goes into the plant through small openings on its surface and enters the soil layer where it's then taken up through the plant's root system. This water-soluble portion is what contributes to polluted runoff. The fat-soluble portion binds to the plant's outer waxy surface, and then stays on that surface. It will bind to any other fat-soluble surface as well – skin, shoes, clothing, soccer balls, baseballs, bats, gear bags, water bottles, etc. If it doesn't dissolve in water, it's a fat-soluble surface. Once it binds to that surface, it remains there until it fully degrades.

Depending on the chemical, it can take months, and in some cases years, to fully degrade. For example, Dicamba, which is one of the most widely used chemicals in over-the-counter lawn herbicide products and a component of 2,4-D, remains active for three to 12 weeks, depending upon weather conditions. How long a chemical persists is based upon chemical half-life, which is the amount of time required for half the pesticide present after application to break down. If a pesticide has a one-month half-life, then 50% of that pesticide is still present after 30 days. It then takes another 30 days for THAT portion to degrade another 50%, and so on. Many assume that these chemicals are simply GONE after a few days. That is simply not true by virtue of how they're engineered.

In terms of how people (children in particular) get exposed, there are three primary ways: Inhalation, transdermal absorption (through the skin), and oral ingestion. Inhalation exposures occur when the pesticide (portion that is bound to water soluble surfactant) volatilizes into the air as water vapor. The more recent the application, the more of the pesticide is volatilized and inhaled. Weather conditions can also increase inhalation exposures. The more water vapor in the air, the higher the concentration of pesticide inhaled. If you can smell a pesticide, you are inhaling it, and it is making its way into your blood stream. Transdermal absorption occurs when our skin comes in contact with a treated surface. Just like a plant's outer surface, skin is a waxy surface. Otherwise, we'd blow up like a sponge every time we get wet. The fat-soluble portion of a pesticide binds to the skin's surface and is absorbed into the bloodstream. The more recent the application, the higher the amount of pesticide absorbed.

Ingestion occurs largely from hand-to-mouth activity when pesticides are bound to the skin or from putting something into the mouth that has pesticides bound to it, like a water bottle, toy, dirt or dust that's on your hands, etc. Children are at a much higher risk of pesticide exposure and injury for several reasons. Compared to adults, children have higher inhalation rates per pound of body weight and, by virtue of their size, are much closer to the ground. Their hand-to-mouth activity is dramatically higher than adults and so they are more frequently and more heavily exposed to environmental toxins. Once exposed, children are at a disadvantage because their developing immune systems and organs cannot process toxins as quickly or effectively as adults. All of these factors apply to our pets as well.

There is clear, scientifically proven danger associated with repeated pesticide exposures. The following all too common scenario shows how easily and frequently children in particular get repeated exposures: A child plays on a sprayed baseball field on Monday, absorbs/inhales/ingests the pesticides on that day. It will take his or her body upwards of two weeks to fully metabolize these toxins. The child plays on the same field five days later and gets another exposure. The child plays soccer three days later on another recently sprayed field. They play baseball the following week, etc. This results in a slow build-up of toxins in the body over time because the frequency of exposure is higher than the body's ability to fully process out the toxins. This low-level, long-term exposure is of the greatest concern and a major risk factor for increased risk of disease over time. Most recent research is showing that the smaller the exposure, the more damage it can cause because the immune system doesn't recognize it. The chemicals then act as free radicals, causing cell and gene damage. individual chemical persistence varies and the conditions under which these chemicals are supposed to degrade can vary widely. Once these chemicals make their way into the car or home, they degrade much more slowly - no direct sunlight, no water, etc., to facilitate the degradation process and why pesticide residues are often found in household dust. Consider a best-case scenario in terms of a pesticide with a very short half-life (many have half-lives of several weeks to months), and only one exposure. If an individual pesticide has a half-life of only a few days, it will be present at the application site for at least two weeks, depending upon weather conditions, and a single exposure means that it will be present in the bloodstream for at LEAST 25 days. This can be much longer in children because their systems are less developed and take even longer to process toxins. For kids who regularly play sports on treated fields or whose parents have their lawns sprayed, they are guaranteed repeated exposures. The body simply does not have enough time to process out the toxin before it gets another dose, leading to a long-term build-up in the bloodstream.

One may ask how it can be proven that pesticides directly cause these health issues. The conversation needs to be less about proof and more about risk to both the individual and wider population. Every person has their own highly individualized set of genetics as well as their own highly individualized exposures over time. Some may not be as negatively affected because their bodies can handle more pesticide exposures before genetic mutations occur that trigger disease. For people whose may already be genetically predisposed to a disease, exposures can be significantly more harmful. In fact, exposure to any toxin, pesticides included, can cause genetic and damage, disruption of normal cellular division and processes, and unregulated replication of cells. The following paper, "Pesticides and Childhood Cancer" (Environmental Health Perspectives 106, (Suppl 3), 893-908: 1998) is based on a presentation at the U.S. EPA Conference on Preventable Causes of Cancer in Children held in 1997. The paper is a thorough review of case reports or case-control studies showing links between pesticides (herbicides and insecticides) and childhood malignancies including leukemia, neuroblastoma, Wilms' tumor, soft-tissue sarcoma, Ewing's sarcoma, non-Hodgkin's lymphoma, and cancers of the brain, colorectum, and testes. The authors conclude that there is potential to prevent at least some childhood cancer by reducing or eliminating pesticide exposure. Since this 1997 report, there is a mounting body of scientific research data showing that these small exposures

over time increase the risk of a multitude of diseases including pediatric and adult-onset cancers, neurological/learning disorders, infertility, birth defects, Parkinson's disease, diabetes, hermaphroditic abnormalities, and asthma.