

## Chapter 6 – GENERAL PROPERTIES OF HERBICIDES

Herbicides belong to a group of chemicals known as pesticides, which prevent, destroy, repel, or mitigate any pest. Herbicides are any chemical substance that is used to specifically kill plants. Other familiar pesticides are insecticides, rodenticides, and fungicides.

### MODE OF ACTION

An herbicide's mode of action is the biochemical or physical mechanism by which it kills plants. Most herbicides kill plants by disrupting or altering one or more of a their metabolic processes. Some disrupt the cellular membranes of plants, allowing cellular contents to leak out, but do not directly disrupt other metabolic processes. Some species or whole groups of plants are not susceptible to certain herbicides because they use different biochemical pathways or have slightly different enzymes. Animals typically suffer little or no effect from most herbicides sold today because these compounds principally affect processes exclusive to plants, like photosynthesis or production of aliphatic amino acids.

#### HERBICIDE FAMILIES VS. MODE OF ACTION

Herbicides that are chemically similar are said to belong to the same "herbicide family". The compounds in a given family typically exhibit similar characteristics and function, due to their chemical and structural similarities. For example, clopyralid, picloram, and triclopyr are all grouped in the pyridine family.

An herbicide's mode of action is the mechanism (biochemical or physical) by which it kills or suppresses plants. The mode of action is generally dictated by its chemical structure, and therefore, herbicides in the same family, tend to have the same Mode of Action. For instance, clopyralid, picloram, and triclopyr are all in the pyridine family and are all auxin mimic herbicides, while glyphosate is an amino acid inhibitor. Some herbicides from different families, however, can have the same mode of action. For example, the phenoxy 2,4-D is an auxin mimic, just like the pyridines picloram, clopyralid, and triclopyr.

The Herbicide Table in this handbook indicates the family and mode of action for each herbicide covered in this manual.

An herbicide is often chosen for use based on its mode of action. If one herbicide is ineffective, another herbicide with a different mode of action may provide better results. When and how an herbicide is applied may be determined by its mode of action.

"Pre-emergent" herbicides are those applied to the soil before the weed germinates, and either disrupt germination or kill the germinating seedling. "Post-emergent" herbicides are those that are applied directly to already established plants and/or soil. Some herbicides are effective both before ("pre-emergent") and after ("post-emergent") germination.

Some of the most common modes of action include:

- Auxin mimics (2,4-D, clopyralid, picloram, and triclopyr), which mimic the plant growth hormone auxin causing uncontrolled and disorganized growth in susceptible plant species;
- Mitosis inhibitors (fosamine), which prevent re-budding in spring and new growth in summer (also known as dormancy enforcers);
- Photosynthesis inhibitors (hexazinone), which block specific reactions in photosynthesis leading to cell breakdown;
- Amino acid synthesis inhibitors (glyphosate, imazapyr and imazapic), which prevent the synthesis of amino acids required for construction of proteins;
- Lipid biosynthesis inhibitors (fluazifop-p-butyl and sethoxydim), that prevent the synthesis of lipids required for growth and maintenance of cell membranes.

### **Auxin Mimics**

Picloram, clopyralid, triclopyr, and 2,4-D are referred to as synthetic auxins. Auxin is a plant hormone that regulates growth in many plant tissues. Chemically, 2,4-D is classified as a phenoxy acetic acid, while picloram, clopyralid, and triclopyr are pyridine derivatives. When susceptible plants are treated with these herbicides, they exhibit symptoms that could be called 'auxin overdose', and eventually die as a result of increased rates of disorganized and uncontrolled growth.

In use since 1945, 2,4-D is one of the most studied herbicides in the world. It is known to affect many biochemical processes in plants, but it is still not clear which of the biochemical alterations 2,4-D and other auxin-mimic herbicides cause that is ultimately responsible for killing plants. It is possible that plants are weakened more or less equally by several of these disruptions with no one process being the most important.

The sequence of events following treatment with an auxin mimic herbicide differs from one species to another and depends on the age and physiological state of the individual plant. Marked changes in the permeability of the plant's cell wall or membrane can generally be detected within minutes of application. This change may lead to a rapid and sustained loss of H<sup>+</sup> ions (protons) from the cell wall, which makes the wall more elastic, and often results in measurable cell growth within an hour. The loss of H<sup>+</sup> ions may also lead to an accumulation of K<sup>+</sup> ions in the stomatal guard cells, causing those cells to swell, increasing the size of the stomatal opening. The increased stomatal opening helps bring about a short-lived increase in photosynthesis, presumably because it allows higher concentrations of CO<sub>2</sub> to reach the photosynthesizing cells inside the leaf.

Other biochemical changes that occur within a day of treatment include a large increase in the concentration of soluble sugars and amino acids inside cells. This increase coincides with an increase in messenger RNA synthesis and a large increase in rates of protein synthesis. Treated plants also frequently produce ethylene, a gaseous plant hormone.

Grasses and other monocots are generally not susceptible to auxin-mimic herbicides. The reason for this selectivity is unclear because there are no apparent differences between the binding sites targeted by auxins in monocots and dicots. It may, however, be due to differences in vascular tissue structure or differences in ability to translocate or metabolize the herbicide (DiTomaso 1999).

### **Mitosis Inhibitors**

Fosamine ammonium is another herbicide that acts as a plant growth regulator. It is sometimes referred to as a “dormancy enforcer,” but the specific mechanism of action has not been identified, even though there is evidence that fosamine ammonium inhibits mitosis in susceptible plants. When applied to deciduous plants up to two months before leaf drop, the compound is absorbed with little or no apparent effect. The following spring however, the plants fail to leaf-out because bud development is either prevented or limited to spindly, miniature leaves. Plants often die as the season progresses because they cannot produce enough photosynthate to sustain themselves. A distinctive feature of this mode of action is that treated plants do not go through a “brown-out” phase, as is often seen after the application of other herbicides. Susceptible non-deciduous plants such as pines, die soon after application because they simply do not produce enough photosynthate.

### **Photosynthesis Inhibitors**

There are two types of photosynthesis inhibitors. Hexazinone is an example of the type that inhibits the transfer of electrons in photosystem II. It blocks electron transport from  $Q_A$  to  $Q_B$  in the chloroplast thylakoid membranes by binding to the D-1 protein at the  $Q_B$ -binding niche. The electrons blocked from passing through photosystem II are transferred through a series of reactions to other reactive toxic compounds. These compounds disrupt cell membranes and cause chloroplast swelling, membrane leakage, and ultimately cellular destruction.

Paraquat and diquat are examples of the second type of photosynthesis inhibitor. They accept electrons from Photosystem I, and after several cycles, generate hydroxyl radicals. These radicals are extremely reactive and readily destroy unsaturated lipids, including membrane fatty acids and chlorophyll. This destroys cell membrane integrity, so that cells and organelles “leak”, leading to rapid leaf wilting and desiccation, and eventually to plant death (WSSA 1994).

### **Amino Acid Synthesis Inhibitors**

Glyphosate and imazapyr kill plants by preventing the synthesis of certain amino acids produced by plants but not animals. Glyphosate blocks the action of the enzyme 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase, which inhibits the biosynthesis of certain aromatic amino acids such as phenylalanine, tyrosine, and tryptophan. These amino acids are required for protein synthesis, which, in turn, is necessary for plant growth and maintenance. Other biochemical processes such as carbohydrate translocation, can also be affected by these herbicides. Although these effects are considered secondary, they may be important in the total lethal action of glyphosate.

Imazapyr, another amino acid synthesis inhibitor, kills plants by inhibiting the production of the branched-chain aliphatic amino acids (valine, leucine, and isoleucine), which are required for DNA synthesis and cell growth. It does this by blocking acetohydroxy acid synthase (AHAS), also known as acetolactate synthase (ALS). Plants treated with imazapyr usually die slowly. The time it takes for a treated plant to die is most likely related to the amount of stored aliphatic amino acids available to the plant. AHAS (ALS) is widespread in plants but the biochemical pathway it catalyzes is not found in animals.

### **Lipid Biosynthesis Inhibitors**

Fluazifop-p-butyl and sethoxydim are both grass specific herbicides that inhibit the synthesis of enzymes required for lipid synthesis. Both inhibit acetyl CoA carboxylase, the enzyme responsible for catalyzing an early step in fatty acid synthesis. Non-susceptible broadleaf species have a different binding site, rendering them immune. The inhibition of acetyl CoA carboxylase and the subsequent lack of lipid production leads to losses in cell membrane integrity, especially in regions of active growth such as meristems. Eventually shoot and rhizome growth ceases, and shoot meristems and rhizome buds begin to die back.

## **FORMULATIONS**

A herbicide formulation is the total marketed product, and is typically available in forms that can be sprayed on as liquids or applied as dry solids. It includes the active ingredient(s), any additives that enhance herbicide effectiveness, stability, or ease of application such as surfactants and other adjuvants, and any other ingredients including solvents, carriers, or dyes. The application method and species to be treated will determine which formulation is best to use. In most cases, manufacturers produce formulations that make applications and handling simpler and safer. Some herbicides are available in forms that can reduce risk of exposure during mixing, such as pre-measured packets that dissolve in water, or as a liquid form already mixed with surfactant and dye (e.g., Pathfinder II<sup>®</sup>).

### **Sprayable/liquid formulations include:**

1. Water-soluble formulations: soluble liquids (SL), soluble powders or packets (SP), and soluble granules (SG). Only a few herbicidal active ingredients readily dissolve in water. These products will not settle out or separate when mixed with water.
2. Emulsifiable formulations (oily liquids): emulsifiable concentrates (E or EC) and gels (GL). These products tend to be easy to handle and store, require little agitation, and will not settle out of solution. Disadvantages of these products are that most can be easily absorbed through the skin and the solvents they contain can cause the rubber and plastic parts of application equipment to deteriorate.
3. Liquid suspensions (L for liquid or F for flowable) that are dispersed in water include: suspension concentrates (SC), aqueous suspensions (AS), emulsions of water-dissolved herbicide in oil (EO), emulsions of an oil-dissolved herbicide in water (EW), micro-encapsulated formulations (ME), and capsule suspensions (CS). All these products consist of a particulate or liquid droplet active ingredient suspended in

a liquid. They are easy to handle and apply, and rarely clog nozzles. However, they can require agitation to keep the active ingredients from separating out.

4. Dry solids that are suspended in water: wettable powders (W or WP), water-dispersible granules (WDG, WG, DG), or dry flowables (DF). These formulations are some of the most widely used. The active ingredient is mixed with a fine particulate carrier, such as clay, to maintain suspension in water. These products tend to be inexpensive, easy to store, and are not as readily absorbed through the skin and eyes as ECs or other liquid formulations. These products, however, can be inhalation hazards during pouring and mixing. In addition, they require constant agitation to maintain suspension and they may be abrasive to application pumps and nozzles.

#### **Dry formulations include:**

1. Granules (G) – Granules consist of the active ingredient absorbed onto coarse particles of clay or other substance, and are most often used in soil applications. These formulations can persist for some time and may need to be incorporated into the soil.
2. Pellets (P) or tablets (TB) – Pellets are similar to granules but tend to be more uniform in size and shape.
3. Dusts (D) – A dust is a finely ground pesticide combined with an inert or inactive dry carrier. They can pose a drift or inhalation hazard.

#### **Salts vs. Esters**

Many herbicidally active compounds are acids that can be formulated as a salt or an ester for application. Once the compound enters the plant, the salt or ester cation is cleaved off allowing the parent acid (active ingredient) to be transported throughout the plant. When choosing between the salt or ester formulation, consider the following characteristics:

##### ***Salts***

- Most salts are highly water soluble, which reduces the need for emulsifiers or agitation to keep the compound suspended.
- Salts are not soluble in oil.
- Salts generally require a surfactant to facilitate penetration through the plant cuticle (waxy covering of leaves and stems).
- Salts are less volatile than esters.
- Salts can dissociate in water. In hard water the parent acid (i.e. the active ingredient) may bind with calcium and magnesium in the water, precipitate out, and be inactivated.

##### ***Esters***

- Esters can penetrate plant tissues more readily than salts, especially woody tissue
- Esters generally are more toxic to plants than salts
- Esters are not water soluble and require an emulsifying agent to remain suspended in water-based solvents
- Esters have varying degrees of volatility

**Adjuvants (including surfactants)**

An adjuvant is any material added to a pesticide mixture that facilitates mixing, application, or pesticide efficacy. An adjuvant enables an applicator to customize a formulation to be most effective in a particular situation. Adjuvants include surfactants, stickers, extenders, activators, compatibility agents, buffers and acidifiers, deposition aids, de-foaming agents, thickeners, and dyes. See the Adjuvant Chapter (Chapter 8) in this handbook for more details on adjuvants.

***Surfactants***

Surfactants are the most important adjuvants. They are chemical compounds that facilitate the movement of the active herbicide ingredient into the plant. They may contain varying amounts of fatty acids that are capable of binding to two types of surfaces, such as oil and water. Some herbicide formulations come with a surfactant already added, in others, surfactants can be added prior to application. Whether a surfactant should be added will be determined by the type of herbicide being applied and the target plant. Read the label for recommendations of appropriate surfactants.

**MECHANISMS OF DISSIPATION**

Dissipation refers to the movement, degradation, or immobilization of an herbicide in the environment.

**Degradation**

Degradation occurs when an herbicide is decomposed to smaller component compounds, and eventually to CO<sub>2</sub>, water, and salts through photochemical, chemical, or biological (microbial metabolism) reactions (Freed and Chiou 1981). Biodegradation accounts for the greatest percentage of degradation for most herbicides (Freed and Chiou 1981). When a single herbicide degrades, it usually yields several compounds (“metabolites”), each of which has its own chemical properties including toxicity, adsorption capacity, and resistance to degradation. Some metabolites are more toxic and/or persistent than the parent compound. In most cases, the nature of the metabolites are largely unknown.

***Photodegradation***

Photodegradation refers to decomposition by sunlight. Sunlight intensity varies with numerous factors including latitude, season, time of day, weather, pollution, and shading by soil, plants, litter, etc. Studies of the photodegradation of herbicides are often conducted using UV light exclusively, but there is some debate as to whether most UV light actually reaches the surface of the earth. Therefore, photodegradation rates determined in the laboratory may over-estimate the importance of this process in the field (Helling et al. 1971).

***Microbial Degradation***

Microbial degradation is decomposition through microbial metabolism. Different microbes can degrade different herbicides, and consequently, the rate of microbial degradation depends on the microbial community present in a given situation (Voos and

Groffman 1997, McCall et al. 1981). Soil conditions that maximize microbial degradation include warmth, moisture, and high organic content.

Herbicides may be microbially degraded via one of two routes. They may be metabolized directly when they serve as a source of carbon and energy (i.e. food) for microorganisms (Hutzinger 1981), or they may be co-metabolized in conjunction with a naturally occurring food source that supports the microbes (Hutzinger 1981). Herbicides that are co-metabolized do not provide enough energy and/or carbon to support the full rate of microbial metabolism on their own.

There is sometimes a lag time before microbial degradation proceeds. This may be because the populations of appropriate microbes or their supplies of necessary enzymes start small, and take time to build-up (Farmer and Aochi 1987, Kearney and Karns 1960). If this lag time is long, other degradation processes may play more important roles in dissipation of the herbicide (Farmer and Aochi 1987). Degradation rates of co-metabolized herbicides tend to remain constant over time.

### ***Chemical Decomposition***

Chemical decomposition is degradation driven by chemical reactions, including hydrolyzation (reaction with hydrogen, usually in the form of water), oxidation (reaction with oxygen), and disassociation (loss of an ammonium or other chemical group from the parent molecule). The importance of these chemical reactions for herbicide degradation in the field is not clear (Helling et al. 1971).

### **Immobilization/Adsorption**

Herbicides may be immobilized by adsorption to soil particles or uptake by non-susceptible plants. These processes isolate the herbicide and prevent it from moving in the environment, but both adsorption and uptake are reversible. In addition, adsorption can slow or prevent degradation mechanisms that permanently degrade the herbicide.

Adsorption refers to the binding of herbicide by soil particles, and rates are influenced by characteristics of the soil and of the herbicide. Adsorption is often dependent on the soil or water pH, which then determines the chemical structure of the herbicide in the environment. Adsorption generally increases with increasing soil organic content, clay content, and cation exchange capacity, and it decreases with increasing pH and temperature. Soil organic content is thought to be the best determinant of herbicide adsorption rates (Farmer and Aochi 1987, Que Hee and Sutherland 1981, Helling et al. 1971). Adsorption is also related to the water solubility of an herbicide, with less soluble herbicides being more strongly adsorbed to soil particles (Helling et al. 1971). Solubility of herbicides in water generally decreases from salt to acid to ester formulations, but there are some exceptions. For example, glyphosate is highly water-soluble and has a strong adsorption capacity.

The availability of an herbicide for transport through the environment or for degradation is determined primarily by the adsorption/desorption process (WHO 1984). Adsorption

to soil particles can stop or slow the rate of microbial metabolism significantly. In other cases, adsorption can facilitate chemical or biological degradation (Farmer and Aochi 1987). Adsorption can change with time and, in most cases, is reversible (i.e. the herbicide can desorb from the soil or sediments and return to the soil solution or water column).

### **Movement/Volatilization**

Movement through the environment occurs when herbicides are suspended in surface or subsurface runoff, volatilized during or after application, evaporated from soil and plant surfaces, or leached down into the soil. Although generally studied and discussed separately, these processes actually occur simultaneously and continuously in the environment (Hutzinger 1981).

Volatilization occurs as the herbicide passes into the gaseous phase and moves about on the breeze. Volatilization most often occurs during application, but also can occur after the herbicide has been deposited on plants or the soil surface. The volatility of an herbicide is determined primarily by its molecular weight. Most highly volatile herbicides are no longer used.

Volatility generally increases with increasing temperature and soil moisture, and with decreasing clay and organic matter content (Helling et al. 1971). The use of a surfactant can change the volatility of a herbicide (Que Hee and Sutherland 1981). In extreme cases, losses due to volatilization can be up to 80 or 90% of the total herbicide applied (Taylor and Glotfelty 1988). Of the herbicides described in detail in this handbook, only 2,4-D and triclopyr can present significant volatilization problems in the field (T. Lanini, pers. comm.).

## **BEHAVIOR IN THE ENVIRONMENT**

Perhaps the most important factor determining the fate of herbicide in the environment is its solubility in water (Hutzinger 1981). Water-soluble herbicides generally have low adsorption capacities, and are consequently more mobile in the environment and more available for microbial metabolism and other degradation processes. Esters, in general, are relatively insoluble in water, adsorb quickly to soils, penetrate plant tissues readily, and are more volatile than salt and acid formulations (Que Hee and Sutherland 1981).

### **Soils**

An herbicide's persistence in soils is often described by its half-life (also known as the DT50). The half-life is the time it takes for half of the herbicide applied to the soil to dissipate. The half-life gives only a rough estimate of the persistence of an herbicide since the half-life of a particular herbicide can vary significantly depending on soil characteristics, weather (especially temperature and soil moisture), and the vegetation at the site. Dissipation rates often change with time (Parker and Doxtader 1983). For example, McCall et al. (1981) found that the rate of dissipation increased until



approximately 20% of the applied herbicide remained, and then declines. Nonetheless, half-life values do provide a means of comparing the relative persistence of herbicides.

The distribution of an herbicide in the soil is determined primarily by the amount, type, and surface area of clays and organic matter in the soil, the amount and quality of soil moisture, and soil temperature and soil pH (Helling et al. 1971). Most natural soils have pH values between 5 and 8 (V. Claassen, pers. comm.). Rainfall and the amount of leaching that has occurred strongly influences these values. In wet areas and/or coarse soils, cations can be leached out, leaving the soil acidic. In arid and semi-arid regions, soils retain cations and are more alkaline. Acidic soils can also be found in bogs where organic acids lower the soil's pH.

### **Water**

Water bodies can be contaminated by direct overspray, or when herbicides drift, volatilize, leach through soils to groundwater, or are carried in surface or subsurface runoff. Amounts of leaching and runoff are largely dependent on total rainfall the first few days after an application. Total losses to runoff generally do not exceed five to ten percent of the total applied, even following heavy rains (Taylor and Glotfelty 1988). High soil adsorption capacity, low rates of application, and low rainfall reduce total runoff and contamination of local waterways (Bovey et al. 1978).

The behavior of an herbicide in water is dictated by its solubility in water. Salts and acids tend to remain dissolved in water until degraded through photolysis or hydrolysis. Esters will often adsorb to the suspended matter in water, and precipitate to the sediments. Once in the sediments, esters can remain adsorbed to soil particles or be degraded through microbial metabolism. Highly acidic or alkaline waters can chemically alter an herbicide and change its behavior in water. The average pH of surface waters is between five and nine (Hutzinger 1981).

## **ENVIRONMENTAL TOXICITY**

The toxicology information reported in this handbook is for the technical grade of the herbicide unless otherwise noted. In some cases, it is not the herbicide itself that is the most toxic component of the applied formula. Adjuvants, such as petroleum solvents (e.g. diesel fuel, deodorized kerosene, methanol), can be highly toxic (Ware 1991). In addition, impurities resulting from the manufacturing process can be more toxic than the active ingredient itself.

### **Birds and Mammals**

A herbicide's toxicity is described by its LD50, which is the dose received either orally (taken through the mouth) or dermally (absorbed through the skin) that kills half the population of study animals. The oral LD50s reported here were determined for adult male rats. The dermal LD50s were determined for rabbits. The LD50 is typically reported in grams of herbicide per kilogram of animal body weight. LD50s are determined under varying circumstances so comparisons between different herbicides

may provide only a rough sense of their relative toxicities. Dermal LD50 values may be more meaningful to herbicide applicators because they are more likely to be exposed to herbicide through their skin rather than by oral ingestion. In any event, very few people, even among applicators, are exposed to herbicide doses as high as the LD50.

The LD50 does not provide any information about chronic, long-term toxic effects that may result from exposure to lesser doses. Sublethal doses can lead to skin or eye irritation, headache, nausea, and, in more extreme cases, birth defects, genetic disorders, paralysis, cancer, and even death. Impurities derived from the formulation of the herbicide and the adjuvants added to the formulation may be more toxic than the herbicide compound itself, making it difficult to attribute increased risks of cancer or other effects directly to a herbicide (Ibrahim et al. 1991).

The most dramatic effects of herbicides on non-target plants and animals often result from the habitat alterations they cause by killing the targeted weeds. For example, loss of invasive riparian plants can cause changes in water temperature and clarity that can potentially impact the entire aquatic community, and the physical structure of the system through bank erosion. Removing a shrubby understory can make a habitat unsuitable for certain bird species and expose small mammals to predation.

### **Aquatic Species**

A herbicide's toxicity to aquatic organisms is quantified with the LC50, which is the concentration of herbicide in water required to kill half of the study animals. The LC50 is typically measured in micrograms of pesticide per liter of water.

In general, ester formulations are more dangerous for aquatic species than salt and acid formulations because ester formulations are lipophilic (fat-loving), and consequently, can pass through the skin and gills of aquatic species relatively easily. Ester formulations, additionally, are not water soluble, and are less likely to be diluted in aquatic systems.

### **Soil Microbes**

Herbicides have varying effects on soil microbial populations depending on herbicide concentrations and the microbial species present. Low residue levels can enhance populations while higher levels can cause population declines. In many cases, studies are too short in duration to determine the true long-term impacts of herbicide use on soil microbes.

## **HUMAN TOXICOLOGY**

When proper safety precautions are taken, human exposure to herbicides used in natural areas should be minimal. Properly fitted personal protection equipment and well-planned emergency response procedures will minimize exposure from normal use as well as emergency spill situations.

**Exposure**

Agricultural workers are often exposed to herbicides when they unintentionally re-enter a treated area too soon following treatment. People who mix and apply herbicides are at the greatest risk of exposure. The most common routes of exposure are through the skin (dermal) or by inhalation (to the lungs). Accidental spills or splashing into the eyes is also possible and with some compounds, can result in severe eye damage and even blindness.

Agricultural herbicide applicators are typically exposed to herbicide levels ranging from micrograms to milligrams per cubic meter of air through inhalation, but exposures through the skin are thought to be much greater (Spear 1991). Spilling concentrated herbicide on exposed skin can be the toxic equivalent of working all day in a treated field (Libich et al. 1984). Dermal exposure can occur to the hands (directly or through permeable gloves), splashes onto clothing or exposed skin, and anywhere you wipe your hands (e.g., thighs, brow). Some tests have found relatively high levels of dermal exposure to the crotch and seat of workers who got herbicide on their hands, and then touched or wiped the seat of their vehicles. Because adsorption through the skin is the most common route of exposure for applicators (Marer 1988), the dermal LD50 may provide more practical information on the relative toxicity of an herbicide rather than the oral LD50, which is based on oral ingestion.

**Toxic Effects**

A person's reaction to pesticide poisoning depends on the toxicity of the pesticide, the size of the dose, duration of exposure, route of absorption, and the efficiency with which the poison is metabolized and excreted by the person's body (Marer 1988, Ware 1991). Different individuals can have different reactions to the same dose of herbicide. Smaller people are, in general, more sensitive to a given dose than are larger people (Marer 1988).

Herbicides can poison the body by blocking biochemical processes or dissolving or disrupting cell membranes. Small doses may produce no response while large doses can cause severe illness or death. The effects may be localized, such as irritation to the eyes, nose, or throat, or generalized, such as occurs when the compound is distributed through the body via the blood stream. Symptoms can occur immediately after exposure or develop gradually. Injuries are usually reversible, but in extreme cases can be permanently debilitating (Marer 1988).

Common symptoms of low-level exposure (such as occurs when mixing or applying herbicides in water) to many herbicides include skin and eye irritation, headache, and nausea. Higher doses (which can occur when handling herbicide concentrates) can cause blurred vision, dizziness, heavy sweating, weakness, stomach pain, vomiting, diarrhea, extreme thirst, and blistered skin, as well as behavioral alterations such as apprehension, restlessness, and anxiety (Marer 1988). Extreme cases may result in convulsions, unconsciousness, paralysis, and death.

Impurities produced during the manufacturing process and adjuvants added to the formulation may be more toxic than the herbicide compound itself. Consequently,

LD50s determined for the technical grade of the herbicide may not be the same as that for the brand name formulation. Combinations of herbicides furthermore, can have additive and synergistic effects in which a formulation of two or more herbicides is two to 100 times as toxic as any one of the herbicides alone (Thompson 1996). Labels should be read carefully for manufacturer's warnings and safety precautions that may be required for a particular formulation.

*NOTE: It is important to remember while interpreting study results discussed in this manual and elsewhere that changes in technology have lowered the detectable residue level 1,000-fold over the last twenty years. Herbicide residues that could only be detected to the parts per million (ppm) level (e.g. one microgram of pesticide per kilogram of soil) in the early 1970's can now be detected at the parts per billion (ppb) level (e.g., one microgram of pesticide per 1,000 kilograms of soil). When a study reports finding no residues it really means that no residues above the lowest detectable level were found. This can be an important difference in comparing the results of studies conducted in the 1960's and 70's to studies from the 1980's and 90's.*

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