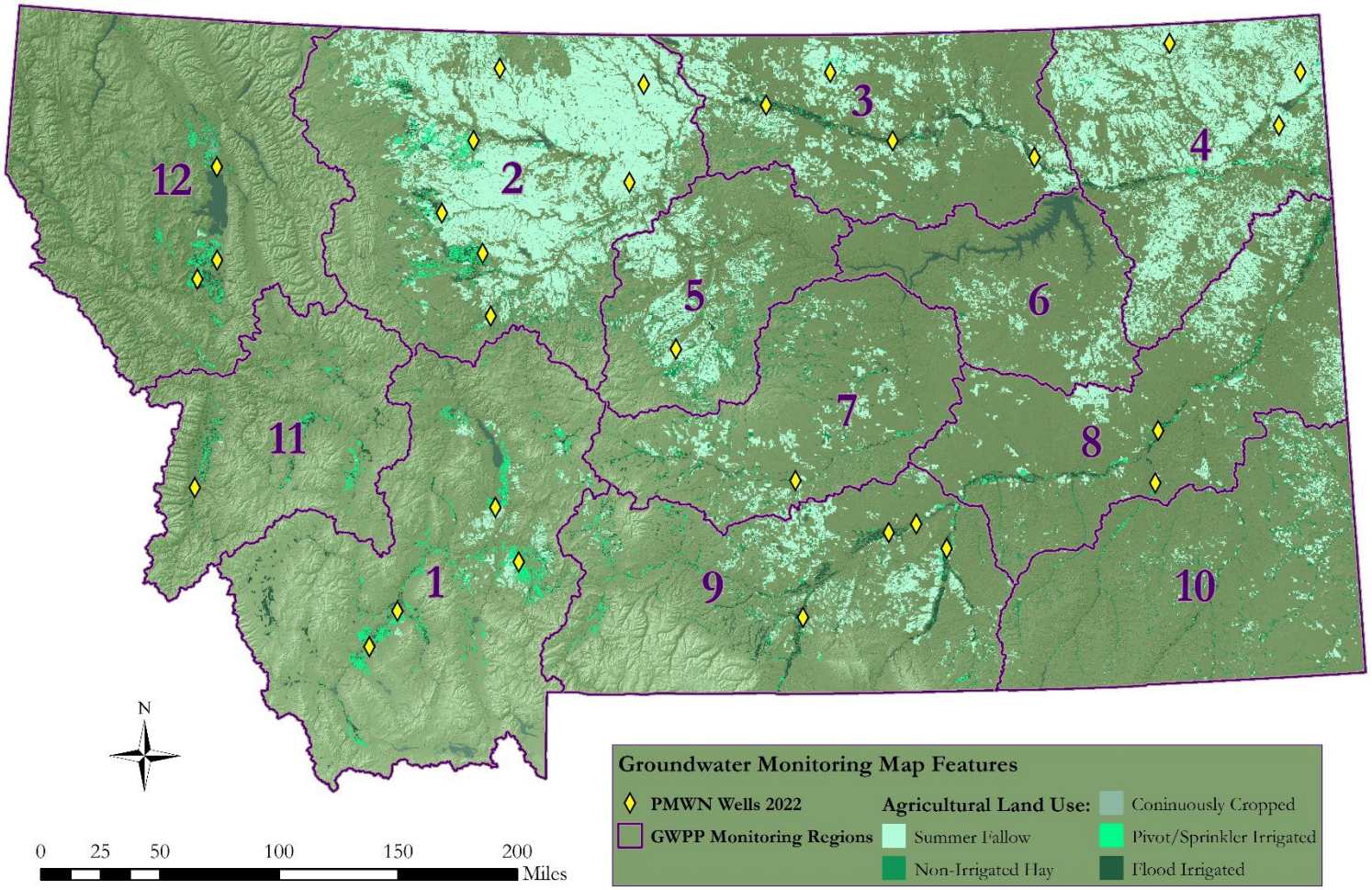
Groundwater Protection Program

Brett Heitshusen
Sustainable Agriculture Section Supervisor
(406) 444-3271
bheitshusen@mt.gov

Montana Department of Agriculture, Groundwater Protection Program
2022 Monitoring Locations



During the 2022 sampling season, the Groundwater Protection Program (GWPP) collected water samples from 28 permanent groundwater monitoring wells and 5 Montana Salinity Control Association groundwater monitoring wells. In total, 109 samples were collected and analyzed for up to 108 pesticides and pesticide metabolites. Results are summarized in the following tables by region. Of the 101 detected chemicals, representing 21 varieties of pesticides, no detections exceeded the respective drinking water standard, or the action threshold of 50% of the respective drinking water standard. All detected pesticide concentrations were < 4 % of their respective drinking water standard.

Region 1

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
Clothianidin	1	0.017	650	Neonicotinoids
Imazamethabenz	2	0.003	1,700 (sum parent + metabolite)	Imidazolinone
Imidacloprid	1	0.002	380	Neonicotinoid
Prometon	2	0.007	100	Triazine

Region 2

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
Clopyralid	1	0.290	1,000	Pyridine carboxylic acid
Fluroxypyr	1	0.062	7,000	Pyridine carboxylic acid
Imazamethabenz	6	0.005	1,700 (sum parent + metabolite)	Imidazolinone
Imazamox	3	0.160	20,000	Imidazolinone
Imidacloprid	3	0.004	380	Neonicotinoid
Pinoxaden	5	0.141	2,000 (sum parent + metabolite)	Phenylpyrazolin
Prometon	2	0.003	100	Triazine
Pyrasulfotole	3	0.084	70	Pyrazole

Region 3

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
No Detections				

Region 4

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
Aminopyralid	3	0.071	3,000	Pyridine carboxylic acid
Flucarbazone	6	0.007	3,000 (sum parent + metabolite)	Sulfonyl amino carbonyl triazolinone
Imazethapyr	1	0.011	17,000	Imidazolinone

Region 5

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
2,4-D	1	0.032	70 (sum parent + metabolite)	Phenoxycarboxylic acids
Azoxystrobin	1	0.0085	1,200	Strobilurins
Flucarbazone	3	0.0094	3,000 (sum parent + metabolite)	Sulfonyl amino carbonyl triazolinone

Region 7

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
No Detections				

Region 8

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
Alachlor	6	0.060	2 (sum parent + metabolite)	Chloroacetamide
Bentazon	3	0.004	210	Benzothiadiazole
Bromacil	2	0.007	700	Uracil
Imazethapyr	2	0.005	17,000	Imidazolinone
Imidacloprid	1	0.002	380	Neonicotinoid
Metolachlor	6	0.014	1,000 (sum parent + metabolite)	Chloroacetamide

Region 9

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
Atrazine	3	0.039	3 (sum parent + metabolite)	Triazine
Clothianidin	5	0.163	650	Neonicotinoid
Metolachlor	6	0.042	1,000 (sum parent + metabolite)	Chloroacetamide
Pinoxaden	5	0.009	2,000 (sum parent + metabolite)	Phenylpyrazolin
Prometon	3	0.002	100	Triazine
Pyrasulfotole	3	0.256	70	Pyrazole
Simazine	2	0.005	4	Triazine

Region 11

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
Imazapic	1	0.010	3,000	Imidazolinone
Imidacloprid	1	0.005	380	Neonicotinoid

Region 12

Analyte	Number of Detections	Average Detected Concentration (ppb)	Drinking Water Standard (ppb)	Pesticide Class
2,4-D	2	0.013	70 (sum parent + metabolite)	Phenoxycarboxylic acids
Imazamethabenz	2	0.061	1,700 (sum parent + metabolite)	Imidazolinone
Imidacloprid	1	0.003	380	Neonicotinoid
Metolachlor	2	1.785	1,000 (sum parent + metabolite)	Chloroacetamide

Analyte Glossary

2,4-D:
2,4-D is a selective systemic herbicide with a wide variety of uses, which include ornamental lawns and turf, right-of-ways, range and pasture, forest management areas, and several crops including corn and small grains. 2,4-DB, 2,4-DP, MCPA, and MCPP are variants of 2,4-D. It is highly soluble in water, non-volatile and has a low potential to leach to groundwater based on its chemical properties. It is non-persistent in soil but may persist in aquatic systems under certain conditions.

Alachlor:
Alachlor is a selective systemic herbicide absorbed by germinating plant shoots. Alachlor is labeled for crop use in beans, corn, and sorghum. It can be applied via soil or water treatment, or aerial application. Alachlor tends to break down easily in the environment and is somewhat mobile; however, Alachlor ESA (ethane sulfonic acid) and OA (oxanilic acid) are stable metabolites which easily leach through soils.

Aminopyralid:
Aminopyralid is applied to wheat, hay, and pastures to control broadleaf noxious weeds. It is a persistent herbicide and does not break down readily in the environment, and can stay active even in manure, mulch, or compost. It is non-volatile, highly soluble in water and, based on its chemical properties, is mobile and has a high potential for leaching to groundwater. It may be moderately persistent in soil systems but would not be expected to be persistent in surface water under normal conditions.

Atrazine:
Atrazine is a restricted use selective systemic herbicide that is used on sorghum and corn in foliar and soil applications. It can also be used in some ornamental turf applications and has previously been labeled for use on roadsides. Atrazine and its degradants tend to be persistent in the environment and have moderate to high leachability in soils.

Azoxystrobin:
Azoxystrobin is a post-emergence broad spectrum fungicide. It is registered for use in a variety of agricultural crops including berry plantings, some truck crops, mint, cherries, corn, peas, lentils, beans, potatoes, canola, sugar beets, and small grains such as oats, rye, sorghum, and wheat. It is also registered for seed treatment of ornamental plants and foliar treatment of ornamental turf. Azoxystrobin has only low solubility, but it has medium potential for particle bound transport and it is moderately persistent in soil and can be very persistent in water.

Bentazon:
Bentazon is a contact herbicide that is used for post-emergence control of annual weeds in several crops and can also be used in non-crop areas. Crop sites include beans, peas, sorghum, and corn, and non-crop sites include ornamentals and turf, rights-of-way, and roadsides. Bentazon is a stable in water but tends to break down in soils. It is highly soluble in water and moderately mobile in soils.

Bromacil:
Bromacil is a nonselective uracil herbicide absorbed mainly through plant roots. It is labeled for use in non-crop areas including rights-of-way, industrial sites, parking areas, fencerows, and railroads. Bromacil is mobile in soils, highly leachable, and persistent in the environment.

Clopyralid:
Clopyralid is a pyridine compound and functions as a selective systemic herbicide that is absorbed through plant leaves and roots. It is labeled for non-crop uses, including fallow land, roadsides, rights-of-way, pasture, rangeland, and CRP lands. Clopyralid is a persistent herbicide that may remain active in animal manure or compost and does not break down readily in the environment. It has a high solubility and is highly leachable to groundwater. It can be persistent in both soil and water systems depending upon conditions.

Analyte Glossary

Clothianidin:
Clothianidin is an insecticide used to control sucking and chewing pests. It is registered for use in corn, potatoes, canola, and small grains and may be applied via seed treatment. Clothianidin is also registered for use in some indoor products, some products for use on specific vegetables and fruits, and in some products for use on turf and ornamentals. It is moderately soluble and can accumulate in both soil and water.

Flucarbazone:
Flucarbazone is an herbicide that is absorbed through roots and foliage and can be translocated in plants. Flucarbazone is labeled predominantly for use in wheat but can also be used in ornamental turf and some specific grasses. It is highly soluble and very leachable in soils. It is not persistent in soil, but it can be very persistent in water.

Fluroxypyr:
Fluroxypyr is a post-emergence, foliar herbicide used control broadleaf weeds in cereals, fallow land, and on-farm noncropland. Fluroxypyr can be a stable, persistent compound in soil and water, and is very water soluble.

Imazamethabenz:
Imazamethabenz is a selective, systemic herbicide labeled for use in wheat, barley, and sunflowers. It is highly soluble in water, leachable in soil, and tends to be persistent in both soil and water.

Imazamox:
Imazamox is a post-emergence herbicide used to control broadleaved plants. It is a contact herbicide with residual activity. Imazamox is labeled for use in some crops including small grains, lentils, sunflowers, canola, and alfalfa, and for non-crop applications including rights-of-way, industrial areas, and some aquatic environments. It is highly soluble and leachable and tends to be persistent in most soil and water environments.

Imazapic:
Imazapic is a selective and systemic herbicide with both contact and residual activity. It is generally used to target annual and perennial grasses and some broadleaf weeds. Imazapic is labeled for use in limited agricultural sites and several non-crop areas such as range and pasture, petroleum tank farms, fence rows, ornamental planting areas, right-of-way, and roadsides. Imazapic is highly water soluble, leachable in soils, but not likely to remain stable in soils.

Imazethapyr:
Imazethapyr is a systemic herbicide with contact and residual activity. It is registered for use in many crop applications, including beans, peas, alfalfa, lentils, corn, and some grasses. It can be applied soil or foliar treatment at several sites. Imazethapyr is persistent in the environment, highly leachable in soils, and may be subject to particle-bound transport.

Imidacloprid:
Imidacloprid is a synthetic, neonicotinoid insecticide. It is labeled for use with foliar and soil applications and seed treatments in several crops, ornamental plants, turf, trees, and greenhouses. Imidacloprid is very soluble and leachable and tends to be stable and relatively persistent in most soil and water environments.

Analyte Glossary

Metolachlor:
Metolachlor is a selective herbicide that inhibits seed germination and mitosis. Metolachlor can be used on ornamental turf and some containerized plants, but is commonly used in crops such as corn, sorghum, and some beans and peas. Metolachlor ESA (ethane sulfonic acid) and OA (oxanilic acid) tend to be very leachable in soil and more persistent than its parent compound.

Pinoxaden:
Pinoxaden is a post emergence herbicide for the control of several grass weed species in all varieties of spring wheat (excluding durum), winter wheat, and barley. Pinoxaden is persistent and mobile in the environment and has the potential to reach aquatic environments and organisms via sheet and channel run-off, discharged groundwater into surface waters, and spray drift from either ground or aerial spray application.

Prometon:
Prometon is a non-selective systemic herbicide absorbed by foliage and roots. It is labeled for non-crop uses only, including right-of-way, fencerows, sidewalks, playgrounds, and parking areas. Prometon is a persistent compound in the environment and can move readily in water in runoff or leach through soil.

Pyrasulfotole:
Pyrasulfotole is a selective herbicide registered for use on cereal grains and is commonly used in wheat and barley. Pyrasulfotole is more persistent in water than in soil, highly leachable in soils and very mobile in surface water runoff.

Simazine:
Simazine is a systemic triazine herbicide chemically related to atrazine. It is labeled for use in agricultural, nursery, forests, ornamental turf, and non-crop settings. It is commonly used in corn and rights-of-way. Simazine can accumulate in soil over time if applied repeatedly and is somewhat mobile in soils and moderately persistent in water.

Pesticide Class Glossary

Benzothiadiazole,
Triazine,
Triazolinone, and
Uracil:

Benzothiadiazoles, Triazines, Triazolinones, and Uracils are a photosystem II inhibitors.
Photosynthetic Inhibitors control many broadleaf and some grass weeds. In general, these herbicides inhibit photosynthesis by binding to D1 proteins of the photosystem II complex in chloroplast thylakoid membranes. Herbicide binding at this protein blocks electron transport and stops CO2 fixation and production of energy needed for plant growth. The death of plants, however, does not occur primarily from photosynthates depletion but rather from an indirect effect on other processes. Blocking electron transport in PSII systems promotes the formation of highly reactive molecules that initiate a chain of reactions causing lipid and protein membrane destruction that results in membrane leakage allowing cells and cell organelles to dry and rapidly disintegrate. In addition, some PSII Inhibitors affect other plant processes such as carotenoid biosynthesis and synthesis of anthocyanin, RNA, and proteins.

Injury Symptoms: Injury symptoms from soil-applied treatments will not appear until after photosynthesis begins. Susceptible broadleaf plants will exhibit interveinal or veinal-chlorosis, depending on the herbicide. Necrosis begins around the leaf margins and progresses toward the center of the leaves. Susceptible grasses will become chlorotic and necrotic beginning at the leaf tips and progressing toward the base of the leaves. Injury symptoms from foliar applications will appear as leaf burn as cell membranes are destroyed. Leaf burn symptoms generally occur most rapidly with hot and humid conditions. It is not unusual to observe symptoms from foliar applied PSII Inhibitors within 72 hours after application.

Chloroacetamide:

Chloroacetamides are long chain fatty acid inhibitors. Long Chain Fatty Acid Inhibitors are preemergent herbicides that are used to control annual grasses and some small-seeded broadleaf weeds in a variety of crops. They do not control or seriously damage emerging plants. The primary site of absorption and action of these herbicides on broadleaf species is the roots, while the primary site of absorption and action on grass species is the emerging shoot. Long Chain Fatty Acid Inhibitors are not readily translocated in the plant. Long chain fatty acid inhibitors include acetamide, chloroacetamide, oxyacetamide, and tetrazolinone herbicides that are currently thought to inhibit very long chain fatty acid (VLCFA) synthesis. These compounds typically affect susceptible weeds before emergence but do not inhibit seed germination.

Injury Symptoms: Symptoms on grass plants include failure of the shoot to emerge from the coleoptile or whorl of the plant, giving the plant a buggy-whip appearance. Susceptible grass seedlings often fail to emerge from the soil. Injury symptoms on broadleaf plants include enlarged cotyledons, restricted growth of the true leaves, dark green color, and stunting. Other symptoms on broadleaf plants include leaf crinkling, leaf red coloring, chlorosis, necrosis, and leaf distortion.

Pesticide Class Glossary

Carboxylic acids (Synthetic Auxins):

Carboxylic acids are synthetic auxins which target the TIR1 receptor. Synthetic Auxins and Auxin Transport Inhibitors are generally used for controlling broadleaf weeds in grass crops, pastures, and industry. These herbicides include some of the more effective chemicals for perennial broadleaf weed and brush control. These herbicides, also known as plant growth regulators, are readily absorbed through both roots and foliage and translocate by phloem or xylem to meristematic tissue interfering with cell formation that results in abnormal root and shoot growth. The killing action of synthetic auxins is not caused by any single factor but rather by the disruption of several growth processes in susceptible plants. It seems, however, that the primary action of these herbicides is likely to affect cell wall plasticity and nucleic acid metabolism. Synthetic auxins also affect protein synthesis, cell division and growth, and stimulate ethylene evolution, which may in some cases produce the characteristic epinastic symptoms associated with exposure to these herbicides.

The synthetic auxins include the following herbicide families: **benzoic acids, phenoxycarboxylic acids, pyridine carboxylic acids, and quinoline carboxylic acids** that act similar to that of endogenous plant auxin. Auxin Transport Inhibitors such as diflufenzopyr, however, inhibit the movement of auxinic compounds out of cells. Consequently, when combined with a synthetic auxin herbicide such as dicamba, the dicamba can move into the cells but cannot move back out of the cell, thus maintaining a greater concentration of the auxinic herbicide within the cell. Diflufenzopyr has minor herbicide activity when applied alone but enhances the activity of auxinic herbicides.

Injury Symptoms: Most auxin herbicides cause similar injury symptoms, but symptom intensity and appearance depend on the herbicide, level of exposure, growth stage, crops, and environmental conditions. In general, trees and shrubs are less sensitive than susceptible annual plants. In addition, younger plants are more susceptible to Auxin Inhibitors than mature plants. Symptoms may range from slight, at low exposure, to severe or death from high levels of exposure. Initial symptoms are twisting, leaf curling, and cupping, which may occur within hours of exposure. Leaves that are not fully expanded at the time of exposure may be stunted and distorted. A few days after exposure, general chlorosis may develop at high exposure levels. Leaves will drop and shoot tips may die, followed by stem dieback in trees and vines. Growth may resume depending on the level of exposure. Regrowth is sometimes limited to buds on the lower part of the plants. Regrowth may exhibit severe shoot and petiole twisting, leaf cupping, stunting, curling, strapping, feathering, roughness, crinkling of the leaf surface, vein discoloration, and fingering of the leaf margins. Auxin herbicides may inhibit interveinal tissue growth making veins appear to be joined together and extended to form finger-like projections. In trees, ornamental shrubs, and grapevines, symptoms may continue to appear until the end of the growing season. In grapevines, 2,4-D symptoms from high concentrations may continue to appear in the second year after exposure. Severely injured grapevines may not recover for two years or more. Furthermore, high concentrations of Auxin Inhibitors may cause stem cracking or dark reddish coloring. Stem elongation of plants may be enhanced (at low concentrations) or inhibited (at high concentrations) by growth Auxin Inhibitors.

Injury from high concentrations of Auxin Inhibitors may change fruit size, shape, and appearance or cause abortion of fruits. Slight auxin herbicide symptoms, however, may have no effect on fruit maturity. Exposure to high concentrations of auxin herbicides may also delay fruit ripening when plants are severely affected. For example, delayed maturity from exposure to high 2,4-D concentrations may exist in a grapevine for one to three years before normal ripening returns.

Pesticide Class Glossary

Imidazolinone and
Sulfonyl Amino-Carbonyl-Triazolinone:
Imidazolinones and Sulfonyl Amino-Carbonyl-Triazolinones are acetolactate sythase (ALS) or acetohydroxy acid sythase (AHAS) inhibitors. The acetolactate synthase (ALS) inhibiting herbicides, also called acetohydroxyacid synthase (AHAS), have a broad spectrum of selectivity and are used at low rates as soil-applied or postemergence treatments in many cropping systems, trees and vines, roadsides, range and pasture, turf, and vegetation management. ALS herbicides are readily absorbed by both roots and foliage and translocated in both the xylem and phloem to the site of action at the growing points. These herbicides inhibit acetolactate synthase, a key enzyme in the pathway of biosynthesis of the branched-chain amino acids isoleucine, leucine, and valine. Plant deaths result from events occurring in response to inhibition of branched-chain amino acids, but the actual sequence of phytotoxic processes is unclear.

Injury Symptoms: Injury symptoms caused by ALS Inhibitors are generally similar. However, the intensity of ALS Inhibitor symptoms varies depending on the herbicide concerned, the rate of application, and the plant species receptor involved. It is not possible, by visual observation alone, to determine what particular ALS Inhibitors may have caused plant damage. It must also be noted that many symptoms that may on first inspection appear to be related to ALS Inhibitors might in fact be symptoms resulting from other biotic or abiotic causes that are entirely unconnected to ALS exposure. In general, injury symptoms caused by ALS inhibiting herbicides are not apparent until several days after treatment. Diagnostic symptoms arising from ALS Inhibitors generally begin with chlorosis. Chlorosis associated with such ALS exposure is shiny or mottled, sometimes with discolored foliage vein. The lower sides of the leaves usually develop a purplish/reddish color. Finally, leaves may die and become necrotic. Symptoms of ALS Inhibitor injury are usually minimal on leaves that are fully expanded before exposure. Fully expanded leaves will appear wilted, crinkled, and chlorotic. Leaves that develop after treatment are chlorotic, crinkled, stunted, and distorted. Affected plants also can exhibit interveinal chlorosis, chlorotic banding on grass leaves, red leaf venation, purpling, necrotic (brown) leaf margins, and gradual death. ALS Inhibitors may stop terminal and lateral growth and cause shoot tips to die. Stems may develop a dark red color with necrotic lesions and cracks.

Growth of ALS sensitive trees, shrubs, and vines usually slows or ceases one to three weeks after exposure (this period will fluctuate depending on the maturity of the tree concerned, plant species, herbicide, rate, and environmental conditions). Where there is high ALS herbicide uptake, the meristematic tip on the stem will die followed by stem dieback. The buds below the dead parts usually grow after a period of time forming new growth that is generally stunted or distorted. After a period of time the plant will resume growth; however, such new growth will have short internodes and multiple branching. At the end of the growing season, the tree may have a "Christmas tree-like" appearance. Several of these symptoms, such as general and interveinal chlorosis, yellow spotting, necrosis, and stem dieback, may result from other causes not ALS Inhibitor damage. If ALS damage is suspected, the progression of symptoms and the study of ALS Inhibitor symptomology, in its entirety, are critical.

Pesticide Class Glossary

Neonicotinoid:

Neonicotinoids are nicotinic acetylcholine receptor (nAChR) agonists. Neonicotinoids are fairly new chemicals, but they have established themselves as key components in insecticides because of their unique selectivity. The mode of action of neonicotinoids is similar to the natural insecticide nicotine. They selectively bind and interact with the insect nicotinic acetylcholine receptor site. When neonicotinoids bind to the binding site of an insect, their electronegative tip, consisting of a nitro or cyano group, interacts with a unique cationic subsite of the insect's receptor. On the other hand, the action of protonated nicotinoids requires a cationic interaction for binding to a mammal receptor. In insects, neonicotinoids cause paralysis which leads to death, often within a few hours; however, they are much less toxic to mammals, and under the WHO/EPA classification these compounds are placed toxicity class II or class III. Because the neonicotinoids block a specific neural pathway that is more abundant in insects than in warm-blooded animals, these insecticides are selectively more toxic to insects than mammals. This target site selectivity is a major factor in the favorable toxicological properties of neonicotinoids.

Phenylpyrazolin:

Phenylpyrazolins are acetyl CoA Carboxylase (ACCase) Inhibitors. ACCase Inhibitors are primarily used for postemergence grass control in broadleaf crops. These herbicides are absorbed through the foliage and translocated in the phloem to the growing point, where they inhibit meristematic activity. ACCase Inhibitors include herbicides belonging to Aryloxyphenoxypropionate (FOPs), cyclohexanedione (DIMs), and phenylpyrazolin (DENs) chemistries. These herbicides inhibit the enzyme acetyl-CoA carboxylase (ACCase), which catalyzes the first step in fatty acid synthesis and is important for membrane synthesis.
In general, broadleaf species are naturally resistant to FOPs, DIMs, and DENs herbicides because of a less sensitive ACCase enzyme. However, ACCase inhibiting herbicides may cause symptoms on certain broadleaf crops. Natural tolerance of some grasses is due to a less sensitive ACCase enzyme or a higher rate of metabolic degradation.

Injury Symptoms: Symptoms on sensitive plants may vary depending on the crop, rate of exposure, and growth stage. Injury symptoms caused by the ACCase Inhibitors appear several days after treatment. Symptoms include chlorosis (yellowing) of newly formed leaves with possible reddening or purpling of older leaves. Tissues at the growing point turn brown and eventually decompose (rotten growing point), a symptom called deadheart. These herbicides may cause general chlorosis and necrosis (white to shiny appearance). Lower exposure rates may cause interveinal bleaching (white) or chlorosis. In corn and sorghum, bleaching is more intense on whorl leaf. In broadleaf plants, symptoms include chlorosis, mottled chlorosis, necrotic spots, leaf crinkling, and leaf distortion.

Pesticide Class Glossary

Pyrazole:

Pyrazoles are a carotenoid boisynthesis inhibitors, targetting the 4-hydroxyphenyl-pyruvate-dioxygenase (HPPD) pathway. These herbicides applied as preemergence or postemergence in different cropping systems, landscape and ornamental, industrial use, and aquatic setting. In general, they are active on broadleaf weeds but control selected grasses. The Carotenoid Biosynthesis Inhibiting herbicides interfere directly or indirectly with carotenoid production that protects chlorophyll from excessive light and photo oxidation. Ultimately, chlorophyll production is inhibited, and plant foliage turns white and appears bleached. Although injury symptoms are similar with these herbicides; the specific site of action is different. Callistemones, isoxazoles, and triketones are examples of herbicides that inhibit phydroxyphenyl pyruvate dioxygenase (HPPD). This enzyme catalyzes a key step in plastoquinone biosynthesis and its inhibition gives rise to bleaching symptoms. These symptoms result from an indirect inhibition of carotenoid synthesis due to the involvement of plastoquinone as a cofactor of phytoene desaturase.

Injury Symptoms: Injury symptoms from the carotenoid biosynthesis herbicides are expressed as white to translucent foliage. Plant foliage appears beached and sometimes with purpling of the leaf margins. In trees and woody ornamentals, symptoms may appear as veinal chlorosis or bleaching. In carotenoid biosynthesis herbicide drift cases bleaching and slight chlorosis may develop within 72 hours of exposure depending on rates and plant species, followed by necrosis in the most severe cases. Leaves that develop after exposure may be crinkled and distorted. Three to four weeks after exposure, plants may recover and appear normal.

Strobilurin:

Strobilurins are part of the larger group of QoIs (Quinone outside Inhibitors), which act to inhibit the respiratory chain at the level of Complex III. Strobilurins specifically bind to the quinol oxidation (Qo) site of cytochrome b to inhibit mitochondrial respiration. This binding blocks electron transfer between cytochrome b and cytochrome c1 and inhibits the synthesis of nicotinamide adenine dinucleotide (NADH) oxidation and the mitochondrial membrane protein adenosine triphosphate (ATP). Although highly effective, fungicide chemistries like those in FRAC group 11, with a very specific mode of action, are susceptible to fungicide resistance development by some fungi.

More information on pesticides' mode of action, like that provided in this document, can be found at the University of California's Herbicide Symptoms web page: https://herbicidesymptoms.ipm.ucanr.edu/