SDSU Extension

iGrow Soybean

BEST MANAGEMENT PRACTICES

Chapter 32:
Soybean Herbicide Injury

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Failure to follow a pesticide label or plants experiencing drift or tank contamination can exhibit dramatic, yet characteristic plant symptoms. If the damage occurs early and is not severe, yield loss may not occur. However, if injury occurs during a critical growth stage or is severe, the damage may result in a total crop loss. The purpose of this chapter is to describe and illustrate typical plant symptoms due to herbicide injury and to discuss the mechanism or mode of action of commonly used herbicides. Symptoms and images of selected herbicides are provided below.

Herbicide control mechanisms

Herbicides have been characterized by the method by which they control susceptible plants. The methods can be divided into mechanism or mode of action groups. Herbicides can produce similar symptoms on susceptible plants (target weeds and soybeans). These categories are provided in Table 32.1. A more complete discussion is provided at http://wssa.net/wp-content/uploads/WSSA-Mechanism-of-Action.pdf. To minimize resistance, weed management strategies should integrate herbicides with different mechanisms of action.

Table 32.1. Weed Science Society of America (WSSA) suggested herbicide mechanism-of-action (MOA) group number, mechanism, and examples. (Chart adapted from University of Illinois, Weed Science–Extension)

MOA Group	Mechanism	Herbicide Chemistry Examples
1	ACETYL COA CARBOXYLASE (ACCase) INHIBITORS	Aryloxyphenoxypropionate (FOPs) Cyclohexanedione (DIMs) Phenylpyrazonlin (DENs)
2	ACETOLACTATE SYNTHASE (ALS) or ACETOHYDROXY ACID SYNTHASE (AHAS) INHIBITORS	Imidazolinones (Imis) Pyrimidinylthiobenzoates Sylfonylaminocarbonyltriazolinones Sulfonylureas (SU) Triazolopyrimidines
3	MICROTUBULE ASSEMBLY INHIBITOR	Benzamide Benzoic acid (DCPA) Dinitroaniline Phosphoramidate Pyridine
15	VERY-LONG-CHAIN FATTY ACID INHIBITOR	Acetamide Chloroacetamide Oxyacetamide Tetrazolinone herbicides
23		Carbetamide, chlorpropham, and propham (Note: Group 23 types of herbicides are no longer or very rarely used int U.S. crop production.)
4	SYNTHETIC AUXINS	Benzoic acids Phenoxycarboxylic acids Pyridine carboxylic acids Quinoline carboxylic acids
5	PHOTOSYSTEM II INHIBITORS Site A	Phenylcarbamates Pyridazinones Triazines Triazinones Uracils
6	Site B	Benzothiadiazinones Nitriles Phenylpyridazines
7	Site A but binds differently than Group 5	Amide Ureas
8	FATTY ACID AND LIPID BIOSYNTHESIS INHIBITORS	Phosphorodithioates Thiocarbamates
16		Benzofuranes
9	ENOLPYRUVYL SHIKIMATE-3-PHOSPHATE (EPSP) SYNTHASE INHIBITORS	Glycines (glyphosate)
10	GLUTAMINE SYNTHETASE INHIBITORS	Phosphinic acids (glufosinate and bialophos)
11	CAROTENOID BIOSYNTHESIS INHIBITORS (bleaching herbicides)	Amitrole
12		Amides Anilidex Furanones Phenoxybutan-amides Pyridiazinones Pyridines
13	Inhibits DOXP synthase	Clomazone
27	Inhibits 4-HPPD enzyme	Callistemones Isoxazoles Pyrazoles Triketones
14	PROTOPORPHYRINOGEN OXIDASE (PPG oxidase or Protox) INHIBITORS	Diphenylethers N-phenylphthalimides Oxadiazoles Oxazolidinediones Phenylpyrazoles Pyrimidindiones Thiadiazoles Triazolinones

MOA Group	Mechanism	Herbicide Chemistry Examples
17	POTENTIAL NUCLEIC ACID INHIBITORS or NON-DESCRIPT MODE OF ACTION	Organic arsenicals
25		Arylaminopropionic acids
26		Unclassified herbicides
18	DIHYDROPTEROATES SYNTHETASE INHIBITORS	Carbamate herbicide Asulam
19	AUXIN TRANSPORT INHIBITORS	Phthalamates (naptalam) Semicarbazones (diflufenzopyr)
20	CELLULOSE INHIBITORS	Nitriles
21		Benzamides
28		Triazolocarboxamides
29		Alkylazine
22	PHOTOSYSTEM INHIBITORS	Bipyridyliums
24	OXIDATIVE PHOSPHORYLATION UNCOUPLERS	Dinitrophenols (dinoterb)

4 MOA Group 4 – Synthetic Auxins
Phenoxycarboxylic acids

Herbicide examples: 2,4-D; MCPA; Stinger®

Mechanism-of-action: Acts as a synthetic auxin, disrupting nucleic acid metabolism and protein synthesis, which ultimately leads to plant death.

Figure 32.1. Plant damage due to 2,4-D application. (Photo courtesy of Michael Moechnig, SDSU)

Injury symptoms: Most injury occurs as drift from roadside applications or tank contamination. Rolled, puckered leaves, and bending stems can be present within hours after application (Fig. 32.1). The injury can be seen even if the plant is exposed to 1/100th of a normal application rate. New leaves may have parallel venation and be "strapped" and root growth will be stunted if exposed to higher rates of chemical. Although symptoms are present, crop yield loss can range from none to severe depending on climate following the injury. Fewer problems with these herbicides will be observed when new resistant varieties to 2,4-D are released.

Soybean injury symptoms from synthetic auxins
Rolled leaves
Leaf strapping
Root stunting
Parallel leaf venation
Bending and twisting of stems (epinasty)

Injury cause
Drift from adjacent fields
Tank contamination (improper cleaning from previous application)

Benzoic acids

Herbicide examples: Dicamba (Banvel®, Clarity®)

Mechanism-of-action: Acts as a synthetic auxin; see 2,4-D.

Injury symptoms: Symptoms are similar to 2,4-D (Fig. 32.2). New varieties are being released that are dicamba resistant.

Figure 32.2. Leaf damage due to dicamba application.
(Photo courtesy of Michael Moechnig, SDSU)

Soybean injury symptoms from benzoic acid herbicides
Same as 2,4-D, but may occur at lower application rates than 2,4-D

Injury cause
Drift
Tank contamination

1 MOA Group 1 – Acetyl CoA Carboxylase (ACCase) Inhibitors *(also known as Lipid Synthesis Inhibitors)*

Herbicide example: Quizalofop (Assure II®)

Mechanism-of-action: Inhibits the formation of lipids used for membranes and stops growth of new tissue of grasses.

Injury symptoms: The ACCase inhibitors rarely injure soybean. However, some injury symptoms such as bleached areas and white spots followed by browning and necrotic areas can occur with these herbicides (Fig. 32.3). In this case, the inert solvent used in the manufacture of Assure II® caused spotting on the soybean leaf after a high application rate was applied to control grasses. This injury is often cosmetic and rarely results in yield loss.

Figure 32.3. Quizalofop application damage to soybean. Note: The herbicide did not damage the soybean. The solvent in Assure II® was the source of the injury when applied at high rates. (Photo courtesy of http://www.btny.purdue.edu/extension/weeds/HerbInj2/InjuryHerb2.html and http://ipm.illinois.edu/pubs/soyinjury.pdf)

9 MOA Group 9 – Enolpyruvyl Shikimate-3-phosphate (EPSP) Synthase Inhibitor *(also known as Amino Acid Derivative Herbicides)*

Herbicide example: Glyphosate (Roundup®)

Mechanism-of-action: Amino acid synthesis inhibitor; stops synthesis of aromatic amino acids (those that contain a phenyl ring).

Injury symptoms: Initially yellowing on plant, followed by plant death. Environmental conditions that slow growth (e.g., extreme heat, cold, or drought) reduce the effects of glyphosate. Youngest leaves near the growing point yellow and die first, then whole plant is affected (Fig. 32.4). Early on, symptoms may appear to be potassium deficiency or soybean cyst nematode damage. Causes of injury to nonresistant glyphosate varieties may be drift from another field, misapplication after emergence to fields planted to nonresistant glyphosate varieties, or tank contamination.

Figure 32.4. Glyphosate damage to soybean. (Photo courtesy of Michael Moechnig, SDSU)

Soybean injury symptoms from EPSP synthase inhibitors
Yellow then brown foliage
Growing point dies

Injury cause
Misapplied to nonresistant varieties after emergence
Tank contamination

10 MOA Group 10 – Glutamine Synthetase Inhibitors *(also known as Phosphoric Acid Type Herbicides)*

Herbicide example: Glufosinate (Liberty®)

Mechanism-of-action: Glufosinate stops the conversion of glutamate + ammonia to the amino acid glutamine, resulting in the accumulation of toxic levels of ammonia in leaf tissue.

Injury symptoms: Symptoms appear within three to five days after treatment. Water-soaked lesions may appear and then leaves become pale yellow (such as nitrogen deficiency) or purple (may look like phosphorus deficiency) (Fig. 32.5). Often occurs if the field was planted with a nonresistant herbicide variety.

Soybean injury symptoms from Glutamine synthetase inhibitors
Pale yellow or purple leaves
Whitening of the leaves
Water-soaked lesions

Injury cause
Misapplied or tank contamination

Figure 32.5. Glufosinate damage to soybean. (Photo courtesy of Michael Moechnig, SDSU)

2 MOA Group 2 – Sulfonylurea (SU) Herbicides and Imidiazalinone (Imi) Herbicides

Herbicide examples: Tribenuron (Express®); thifensulfuron (Harmony®); metsulfuron (Ally XP®)

Mechanism-of-action: Both SU- and Imi-type chemistries of herbicides inhibit the formation of branched chain amino acids.

Injury symptoms: Injury symptoms appear seven to ten days after exposure. Sensitive plants generally show overall yellowing (chlorosis) and stunting. Purpling of the veins is often noted (Fig. 32.6) and roots may show a bottle-brush appearance (Fig. 32.7).

If applied at the correct rate, injury symptoms are often temporary. Symptoms may be noticed even if applied according to the label rates and timings if temperatures and humidity are high, although plants often recover with no yield loss. SU and Imi herbicides typically are applied at low rates (ounces of active ingredient per acre) and tank contamination may be a problem. In high pH soils, carryover of the SU chemistry types may be problematic, whereas carryover of Imi chemistry-type herbicides is more likely in low pH soils.

Figure 32.6. Sulfonyl herbicide damage to soybean showing purple veins. (Photo courtesy of Michael Moechnig, SDSU)

Figure 32.7. Sulfonyl herbicide damage to soybean roots showing a bottle-brush appearance. (Photo courtesy of http://www.btny.purdue.edu/extension/weeds/HerbInj2/InjuryHerb3.html#imazaquin)

Soybean injury symptoms from SU or IMI chemistries
Stunted plant, stunted internodes
Yellow translucent leaves
Purpling of the veins
Death of growing point
Bottle-brush roots

Injury cause
Variety sensitivity
Applied during times with high temperatures and humidity
Tank contamination

13 27 MOA Groups 27 and 13 – Inhibits DOPX synthase or 4 HPPD enzyme

Herbicide examples: Group 27 – isoxaflutole (Balance®), tembotrione (Laudis®), and mesotrione (Callisto®); Group 13 – clomozone (Command®)

Mechanisms-of-action: There are several herbicides with different specific mechanisms of action that have similar symptoms although they have different specific target enzymes. Both block enzymes that do not allow for carotenoid pigments to be formed, but the enzymes differ. Carotenoids are plant pigments that include chlorophyll or protect membranes from destruction by reactive oxygen species (ROS).

Injury symptoms: White areas on plants or albino plants appear during emergence. Command® carryover may be seen early in the season; plants may recover from early season injury (Fig. 32.8).

Figure 32.8. Command® injury appears as white edges on plants.
(Photo courtesy of Michael Moechnig, SDSU)

Soybean injury symptoms from Carotenoid biosynthesis inhibitors
White tissue
Poor emergence
Stunted plants
Growing point dies

Injury cause
Applied on cool, wet, or sandy soils
Spray drift
Carryover problem

14 MOA Group 14 – Protoporphyrinogen Oxidase (PPG oxidase, PPO, or PROTOX) Inhibitors

Herbicide examples: Carfentrazone (Aim®) (can be applied pre-emergence); Acifluorfen (Blazer®) (applied post-emergence only)

Mechanism-of-action: Inhibits protoporphyrinogen oxidase causing a cascade of events, which eventually result in cell membrane destruction and death of the plant.

Injury symptoms: If applied pre-emergence to soybean, stem lesions, or burning of the cotyledon leaves is apparent (Fig. 32.9). More apt to occur if heavy rain occurs after application, but before emergence. If applied post-emergence, appearance of necrotic (dead tissue) speckling on leaves within a few days after exposure (Fig. 32.10). Note that the new leaves are unaffected by the application. The herbicide is a contact herbicide, which means the herbicide does not move throughout the plant. Symptoms occur if applications are made during hot, humid weather, or if cool conditions occur after application. If applied at the correct rate, the symptoms are often cosmetic and do not lead to yield losses.

Left: Figure 32.9. Carfentrazone injury—herbicide applied pre-emergence to the soil.
(Photo courtesy of http://ipm.illinois.edu/pubs/soyinjury.pdf)
Right: Figure 32.10. Acifluorfen injury—herbicide applied post-emergence to the leaves.
(Photo courtesy of Michael Moechnig, SDSU)

Soybean injury symptoms from PROTOX inhibitors
Stem lesions and/or cotyledon browning
Yellowing or reddening of new leaves
Speckling of the older exposed leaves
Stunting of plant
Death of tissue and browning
Growing point dies

Injury cause
Misapplication
Heavy rain before emergence (if applied pre)
Application during hot, humid weather (if applied post)
Application occurs just before a cool period
Tank contamination

22 MOA Group 22 - Photosystem I Inhibitors

Herbicide example: Paraquat (Gramoxone®)

Mechanism-of-action: Herbicide accepts electrons from photosystem I and forms an herbicide radical. This radical reduces molecular oxygen to form superoxide radicals. The radicals are extremely reactive and destroy membrane fatty acids, which lead to the destruction of the cell membrane and death.

Injury symptoms: Symptoms are often observed within hours, especially on sunny days. Leaves develop water-soaked lesions, very quickly, and then speckling and dead tissue are observed (Fig 32.11). Note, this is a contact herbicide and the new leaves show no injury symptoms. However, the plants shown in Figure 32.11 are severely injured and plants may not survive.

Figure 32.11. Paraquat injury to soybean. (Photo courtesy of Michael Moechnig, SDSU)

Soybean injury symptoms from photosystem I inhibitors
Limp leaves
Within hours, water-soaked appearance (looks like frost damage)
Within days, brown tissue where water-soaked areas were first observed

Injury cause
Drift
Tank contamination

5 MOA Group 5 – Photosystem II Inhibitor - Triazine

Herbicide examples: Atrazine (Aatrex®); metribuzin

Mechanism-of-action: Stops electron flow during photosynthesis, which slows plant growth. These herbicides bind at site A in PSII and they may also impact lipid and protein oxidation, which leads to leaky cell membranes and plant death.

Injury symptoms: Atrazine is not labeled on soybean, but injury may occur if there is soil carryover from the previous year (Fig. 32.12). Carryover may occur if the soil pH is high (>7.8) or if weather was cool and dry during the prior year. Carryover injury can be made more severe if an application of metribuzin is applied in the current year. In addition, tank contamination from previous applications may occur. Triazine carryover injury symptoms start as yellowing of the seedling and then death of the oldest leaves. Roots are malformed. If severe, plants will not survive. Metribuzin can be applied to soybean, but it has a short tolerance window. As always, read and follow labeled instructions. In addition, some varieties are less tolerant to metribuzin.

Figure 32.12. Atrazine injury.
(Photo courtesy of http://ipm.illinois.edu/pubs/soyinjury.pdf)

Soybean injury symptoms from triazine herbicides
Yellow and brown leaves with outer edges of the leaf and older leaves most affected

Injury cause
Cool wet conditions slowing soybean growth
Crop oil synergy if metribuzin is applied post-emergence

6 MOA Group 6 – Photosystem II Inhibitor - Benzonitriles

Herbicide example: Bentazon

Mechanism-of-action: Stops electron flow in photosynthesis in photosystem II, but unlike atrazine, binding of the herbicide occurs at site B. The final effects are similar to atrazine if applied post as a tank-contamination or drift problem.

Injury symptoms: Symptoms appear as leaf tip chlorosis, general wilting, speckling, and necrotic lesions to tissue where application has occurred (Fig. 32.13). Young tissues that emerge after application are generally unaffected as the herbicide does not translocate within the plant. Injury may occur if cool or very high temperatures occur after application or high crop oil concentrations are used. Recovery is generally rapid. This is often variety specific with some more tolerant than others.

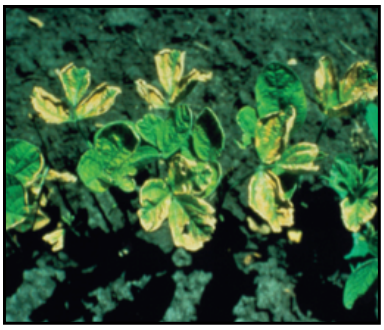

Figure 32.13. Bentazon injury.
(Photo courtesy of http://ipm.illinois.edu/pubs/soyinjury.pdf)

Soybean injury symptoms from benzonitrile herbicides
Yellow and brown leaves

Injury cause
Crop oil with the post-emergence application

3

MOA Group 3 – Mitosis Inhibitor - Microtubule Assembly - Dinitroanalines

Herbicide examples: Trifluralin (Treflan®); pendimethalin (Prowl®)

Mechanism-of-action: Inhibits the growth of roots or shoots of seedlings by binding to tubulin, which leads to loss of microtubules assembly, structure, and function. This in turn leads to stoppage of cell division.

Injury symptoms: Symptoms are apparent during or soon after plant emergence (Fig. 32.14). Symptoms include shortened, thickened, and swollen roots (root clubbing). They may also be purple in color. Injury occurs if DNA herbicide is incorporated too deeply into the seeding zone. Factors contributing to plant injury include wet, cool soils, compaction, and drought.

Figure 32.14. Dinitroanaline injury. (Photo courtesy of http://www.btny.purdue.edu/extension/weeds/HerbInj2/InjuryHerb3.html#imazaquin)

Soybean injury symptoms from dinitroanalines
Stunted plants
Roots short and thick

Injury cause
Carryover
Misapplication
Over-application

15 MOA Group 15 – Mitosis Inhibitor - Very-long-chain Fatty Acid Inhibitor - Acetanilides

Herbicide examples: Metolachlor (Dual®); acetochlor (Harness®)

Mechanism-of-action: Growth inhibitor that stops the formation of very long fatty acids. This stoppage has effects on the formation of all cell membranes. Seedling roots and shoots of susceptible plants stop growing.

Injury symptoms: Death of the plant occurs soon after emergence or no plants emerge in the area. Plants that survive may appear heart-shaped due to shortened mid-veins of the leaves (draw-string) leaves (Fig. 32.15). Roots may also be shortened.

Figure 32.15. Acetanilide injury.
(Photo courtesy of Michael Moechnig, SDSU)

Soybean injury symptoms from acetanilides
Poor emergence
Stunted plants
Poor root development
Heart-shaped leaves on emerged plants

Injury cause
Over-application or high rates used
Cool, wet soils

8 16 MOA Groups 8 and 16 – Fatty Acid and Lipid Biosynthesis Inhibitor

Herbicide example: EPTC (Eptam®)

Mechanism-of-action: Inhibit biosynthesis of fatty acid and lipids (not through ACCase); biosynthesis of proteins; and inhibits gibberellin synthesis.

Injury symptoms: Appear during or soon after plant emergence. Reduction in cuticular wax deposition that may lead to increased disease and stress severity (Fig. 32.16). Injured seedlings may show reduced coleoptile length, stunting, or delayed emergence. Leaves may stick together and have cupped or crinkled necrotic edges. Leaf buds may not open.

Soybean injury symptoms from EPTC
Stunted plants
Leaves stuck together
Uneven emergence across field

Injury cause
Over-application
Cool, wet soils

Figure 32.16. EPTC injury.
(Photo courtesy of http://weedscience.missouri.edu/herbinjsymptoms/thiop.htm)

References and additional information

Summary of herbicide mechanism of action according to the Weed Science Society of America (WSSA). Available at http://wssa.net/wp-content/uploads/WSSA-Mechanism-of-Action.pdf

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