SS-AGR-327

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IFAS Extension

Diagnosing Herbicide Injury in Peanut[1]

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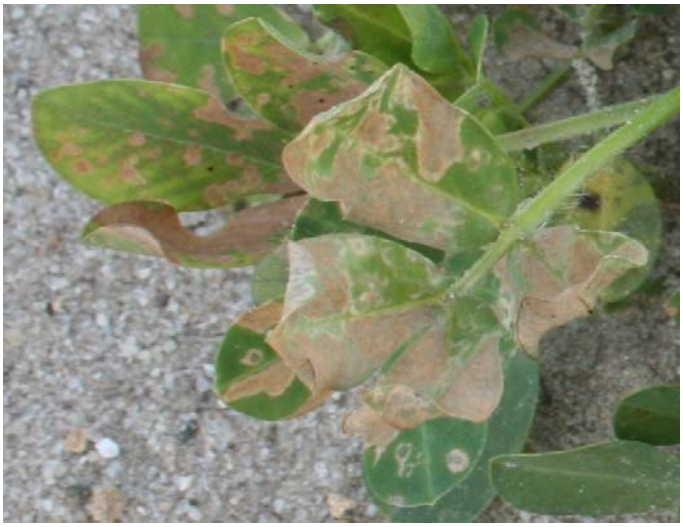

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1) Photosynthesis Inhibitors

Mechanism of action: Photosynthesis-inhibiting herbicides block the electron flow that results after the leaf absorbs light energy. Consequently, the chloroplast is unable to process the absorbed light energy. This massive buildup of energy eventually leads to cell membrane disruption and leaf death.

Behavior in plants: Most of these herbicides have significant soil activity and are applied preemergence. The herbicide is absorbed by roots and translocated by xylem upward to the shoots following the transpiration stream. There is no phloem translocation and the herbicide accumulates in the leaf, particularly at the margin of the leaves.

Symptomology: Injury symptoms on susceptible plants begin with interveinal chlorosis of the leaves, then yellowing at the margins followed by necrosis. Older leaves are more affected than newer leaves.

Herbicides with this mode of action: Atrazine, Diuron, Bromoxynil, Metribuzin (Lexone/Sencorex),Bentazon, (Basagran)**,** Flumeturon**,** Prometryn

1)Figure #1: Slight Atrazine injury.

1)Figure #2: Severe Atrazine injury.

1)Figure #3: Yellowing followed by marginal necrosis

2) Amino acid Synthesis Inhibitors (ALS Inhibitors)

Mechanism of action: The ALS-inhibiting herbicides block the acetolactate synthase (ALS) enzyme. The ALS enzyme is responsible for the formation of essential amino acids in the plant (isoleucine, leucine, and valine). Without these amino acids, proteins (complex molecules that control all plant functions) cannot be formed and the plant slowly dies.

Behavior in plants: These herbicides are absorbed by roots and leaves and are translocated extensively in both the xylem and phloem. As these herbicides translocate to the areas of most rapid growth, injury symptoms are most commonly observed in the terminals of rapidly growing plants. It may take two weeks to develop injury symptoms depending on weather conditions (temperature, soil moisture, etc.) and the overall rate of plant growth.

Symptomology: Injury on peanut begins as stunted growth and overall chlorosis (yellowing) in the youngest leaves. The chlorosis is followed by necrosis (leaf death) some days later. Occasionally the peanut plant will develop blackened terminal leaves. In some species, leaf veins will turn red, but this is not common in peanut. Injury can easily be confused with glyphosate injury.

Herbicides with this mode of action:

•**Imidazolinones:** Imazapyr (Arsenal), Imazapic (Cadre)***,** Imazethapyr (Pursuit)***,** Imazaquin (Scepter)

•**Sulfonylureas:** Chlorimuron***,** Halosulfuron**,** Metsulfuron**,** Nicosulfuron, Prosulfuron, Primisulfuron, Chlorsulfuron, Tryfloxysulfuron

•**Sulfananilides:** Diclosulam***,** Chloramsulam**,** Flumetsulam

•**Pyrimidinylthiobenzoic acid:** Staple (Pyrithiobac)

(*) Labeled in peanut.

2)Figure #1: Tryfloxysulfuron injury (Chlorosis in young leaves).

2)Figure #2: Metsulfuron injury (Chlorosis in young leaves).

3) Growth Regulators

Mechanism of action: Auxin is a plant hormone that controls the rate of plant growth. The growth regulator herbicides (also called hormone herbicides or auxinic mimic herbicides) bind to auxin receptors within the plant, mimicking natural auxin. This action causes rapid and uncontrolled cell division and growth, resulting in vascular tissue destruction and multi-system disruption. These herbicides are highly mobile in the plant and tend to accumulate in meristematic regions.

Behavior in plants: 2,4-DB is almost identical to 2,4-D, but contains additional elements that render it non-herbicidal. Many species quickly remove this protective side-chain and release 2,4-D within the plant. But legumes (peanut, soybean, pea, etc.) are unique in that they do not remove the side-chain from 2,4-DB, leaving this herbicide largely inactive within the plant. The rate of 2,4-DB to 2,4-D conversion is what determines the level of control that will be achieved with a 2,4-DB application.

Symptomology: Symptoms of herbicide injury on peanut are twisting and curling (epinasty) of stems and petiols. Leaf shape can appear out of proportion (uncharacteristically long and narrow, called strapping) and venation appears abnormal. Bleaching near the base of the leaf is common. With some herbicides (Picloram and Dicamba) we can see leaf cupping, and in some cases it is possible to observe malformation of pods.

Herbicides with this mode of action: 2,4-D, 2,4-DB *****, Dicamba, Picloram, Triclopyr, Aminopyralid

(*) Labeled in peanut

3)Figure #1: 2,4-D Amine injury (twisting and curling)

3)Figure #2: Dicamba injury (cupping)

3)Figure #3: 2,4-DB injury (strapping)

4) Pigment Inhibitors

Mechanism of action: Chlorophyll absorbs light and directs it to be converted to a chemical that can be used by the plant. But chlorophyll can be damaged if too much sunlight is present, so carotenoid pigments are present to dissipate this excess energy and protect chlorophyll. Pigment-inhibiting herbicides act by blocking the formation of carotenoids so that chlorophyll is destroyed by light energy from the sun. These herbicides can be applied preemergence or postemergence.

Symptomology: With no carotenoid pigments produced, the chlorophyll is destroyed, leaving no leaf pigments of any kind. The result is bleached plants (white foliage). The level of bleaching can be complete or incomplete, depending on herbicide dose. Affected plants often recover from light bleaching while total bleaching is rapidly followed by necrosis (dead leaf tissue).

Herbicides with this mode of action: Norflurazon***,** Mesotrione**,** Clomazone, Isoxaflutole, Tembotrione (Laudis)

(*) Labeled in peanut

4)Figure #1: Norflurazon injury (bleaching).

4)Figure #2: Severe Norflurazon injury (bleaching).

4)Figure #3: Severe Tembotrione injury (bleaching)

5) Cell Membrane Disruptor

Mechanism of action: The cell membrane disruptor herbicides (also called the PPO herbicides) inhibit an enzyme that synthesizes chlorophyll. Consequently, accumulation of a pre-chlorophyll molecule occurs. The pre-chlorophyll molecule can accept light energy, but cannot pass it along to the photosynthesis process. As a result, toxic radicals that destroy cell membranes are formed in the chloroplast. Rapid desiccation and necrosis of leaves occurs within 1–3 days, depending on light and weather conditions.

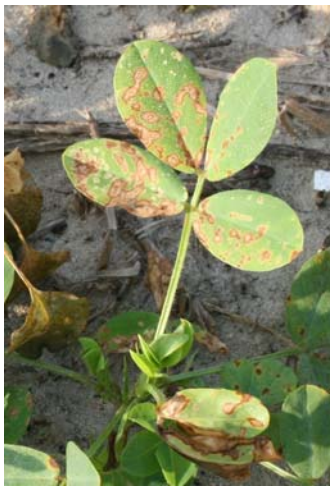
Although many herbicides in this group are labeled for preemergence applications only, all of these herbicides possess foliar activity.

Symptomology: Bronzing and necrosis (speckling) are characteristics of these herbicides when applied post-emergence. Injury from cell membrane disruptor herbicides can be easily confused with paraquat injury except that no bronze coloration will be associated with paraquat applications.

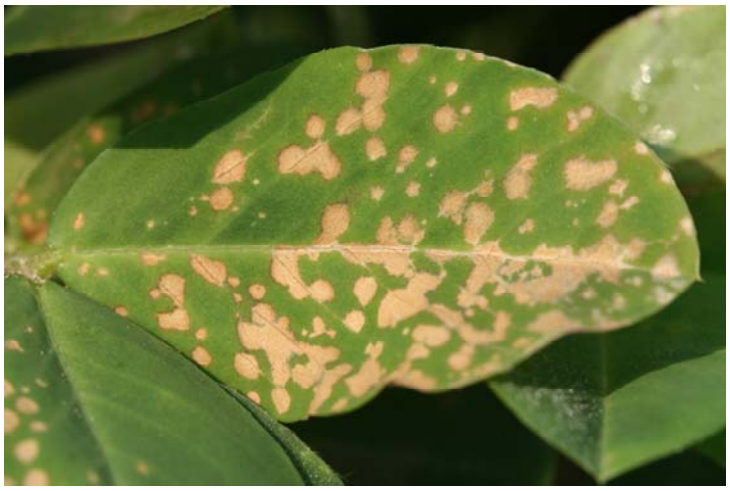
Herbicides with this mode of action:

Flumioxazin***,** Fomesafenm, Sulfeltrazone, Lactofen*****, Carfentrazone, Acifluorfen

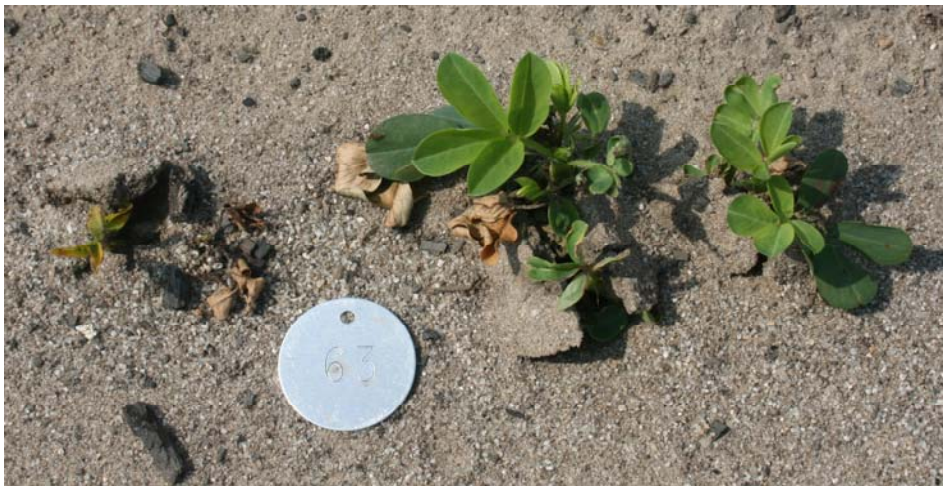
(*) Labeled in peanut



5)Figure #1: Cobra injury (necrosis).



5)Figure #2: Necrosis (speckling).



5)Figure #3: Valor injury after rainfall followed by cool temperature.

6) Lipid Synthesis Inhibitors (ACCase Inhibitors)

Mechanism of action: These herbicides inhibit an enzyme (ACCase) that produces lipids, which are used to build cell membranes. Although all plants contain an ACCase enzyme, broadleaf and grass plants have distinctly different types of this enzyme. ACCase-inhibiting herbicides cannot bind to this enzyme in broadleaf plants, rendering these plants totally immune to the herbicide. Conversely, almost all grasses are sensitive and these herbicides are used for postemergence grass control (this is why people call them graminicides). They do not have soil activity.

Symptomology: Peanut is very tolerant of these herbicides and no injury is caused by them. However, under hot and humid weather conditions, the crop oil adjuvants used for these applications can cause transient leaf burning on tolerant broadleaf plants.

Herbicides with this mode of action: Sethoxydim*, Cletodim**

(*) Labeled in peanut

7) Seedling growth inhibitors

There are two types of seedling growth inhibitors: "root inhibitors" and "shoot inhibitors." They have different modes of action, but both control plants at the seedling stage below the ground.

Dinitroanalins:

Mechanism of action: The dinitroanalin herbicides (also called DNAs, or the yellow herbicides) inhibit root formation in susceptible plants. Root inhibition occurs when the herbicide blocks the process of mitosis (cell division) in the root tip. Affected plants eventually die of drought stress, regardless of soil water status, due to the inability to develop a sufficient root structure to support the water needs of the developing leaf and shoot. But there is a good correlation between lipid content of seeds and sensitivity to these herbicides. The higher the seed lipid content, the more tolerance to these herbicides. As peanut seeds have high lipid content, it is difficult it observe injury from these herbicides. Besides, peanut taproot is much less affected than lateral roots. The peanut taproot may elongate sufficiently to place the apical meristems below the zone of treated soil where lateral roots develop. Thus, herbicide **placement** is another factor in plant selectivity.

Behavior in plants: The yellow herbicides are not translocated in plants and only work at the root tip. Roots that extend beyond the treated zone will regain normal growth and development.

Symptomology: Peanut symptoms from these herbicides are stunted roots that are thick and "stubby." These symptoms are often associated with high rates of herbicides, improper incorporation (placement) or shallow planting followed by excessive rainfall.

Herbicides with this mode of action: Pendimentalin***,** Trifluralin**, Oryzalin, Ethalfluralin*

(*) Labeled in peanut

(**) Labeled only in peanuts grown in Texas, Oklahoma and New Mexico.

Chloroacetamides:

Mechanism of action: These herbicides inhibit several metabolic processes such as lipid and protein synthesis. They are absorbed into the shoots of emerging plants. The herbicide must be present during emergence to be effective. No activity will be observed from later applications to emerged weeds.

Symptomology: Peanut are tolerant to these herbicides, but injury can be observed under certain conditions. The primary symptoms in peanut are root deformation or curling (J-rooting). Symptoms are most commonly observed when peanuts are emerging in cool and wet soil conditions. Severely affected plants rarely make full recovery from initial injury.

Herbicides with this mode of action: Metolachlor***,** Alachlor* (not for use in Florida), Dimethenamid***,** Acetochlor

(*) Labeled in peanut

7)Figure #1: Dual injury (root curling).

7)Figure #2: Dual injury: Severe injury (left), slight injury (middle), and no injury (right).

8) Glufosinate

Mechanism of action: Glufosinate inhibits the activity of an enzyme that converts glutamate and ammonia into glutamine. Inhibiting this enzyme leads to the accumulation of toxic levels of ammonia within the cell. The buildup of ammonia quickly leads to multisystem failure within the photosynthesis pathway and causes irreversible cell damage. Glufosinate has no soil activity.

Symptomology: This is a foliar applied herbicide with contact activity (no translocation). Peanut is very sensitive to glufosinate, but injury can be easily mistaken for paraquat or cell disruptor herbicides. The key difference between glufosinate and these other herbicides is that glufosinate will not cause bronzing on the affected leaves. However, glufosinate and paraquat are almost impossible to distinguish between since both herbicides may produce irregular chlorosis followed necrosis.

Herbicides with this mode of action: Glufosinate

8)Figure #1: Glufosinate injury.

8)Figure #2: Glufosinate injury.

8)Figure #3: Glufosinate injury.

9) Glyphosate

Mechanism of action: Glyphosate inhibits an enzyme (EPSP) which is responsible for the production of aromatic amino acids (tryptophan, tyrosine, and phenylalanine). Proteins regulate all activity that occurs within a cell, but without these essential amino acids no proteins can be formed. Halting amino acid production slowly starves the plant and several days or weeks are required before symptoms or plant death is observed.

Symptomology: The most common symptom of glyphosate injury is generalized chlorosis (leaf yellowing) that begins in the newest leaves and progresses to the entire plant. However, chlorotic mottling and even bleaching can be observed. The degree and type of injury observed will depend on application rate and peanut growth stage. Glyphosate injury can be distinguished from injury from photosynthesis inhibitors since glyphosate will not accumulate at the leaf margins and die from the tip to the base. Distinguishing glyphosate injury from ALS herbicide injury is difficult if not impossible. Regardless, unless glyphosate injury is severe and persists for several weeks, peanuts generally make full recovery with peanut yield only slightly affected.

9)Figure #1: Severe Glyphosate injury.

9)Figure #2: Slight Glyphosate injury. See chlorosis at the margins of the leaflets.

9)Figure #3: Slight Glyphosate injury in a newer leaf. See the older leaf with no injury.

9)Figure #4: Slight Glyphosate injury. See chlorosis at the margins of the leaflets.

9)Figure #5: Glyphosate injury. Well defined pattern.

9)Figure #6: Old symptoms of Glyphosate injury.

9)Figure #7: Slight Glyphosate injury. See chlorosis at the margins of the leaflets.

10) Paraquat*

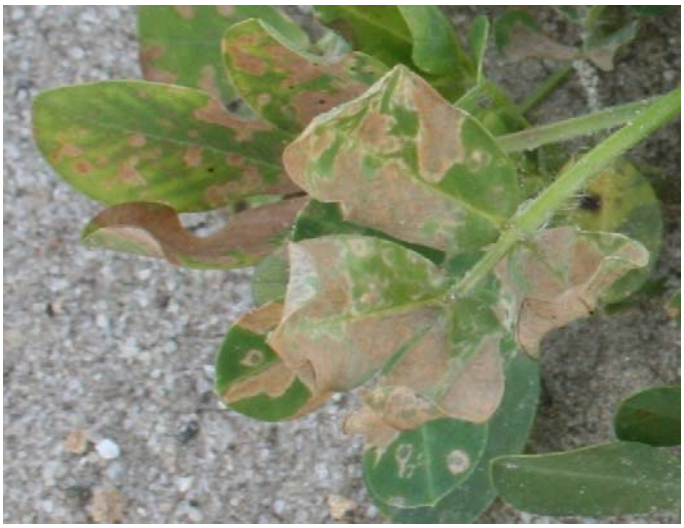
Mechanism of action: Paraquat is a postemergence herbicide with contact foliar activity. It is rapidly absorbed by green tissue and inhibits photosynthesis. The specific site of action is different from that of atrazine and the results are a much more rapid onset of leaf death. Regardless, paraquat causes the formation of radical oxygen, which is toxic to plant cell membranes, within the chloroplast.

Symptomology: Paraquat causes rapid leaf burning on all species present at the time of application. Injury can be confused with the cell membrane disruptors or glufosinate. Paraquat can be separated from the cell membrane disruptors since paraquat does not result in bronzing around the necrotic lesion. However, injury symptoms of paraquat and glufosinate are almost indistinguishable. The only difference between the two is that paraquat injury will generally appear within 1 day of application where glufosinate may require 3 to 4 days to develop symptoms.

Herbicides with this mode of action: Paraquat*

(*) Labeled in peanut

10)Figure #1: Paraquat injury.



10)Figure #2: Paraquat injury.

10)Figure #3: Paraquat injury.