

Diagnosing Herbicide Injury - 2007¹

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About 100-110 chemicals comprise the active ingredients in the several hundred herbicide formulations now available for weed control in Florida. All of these compounds are thoroughly and extensively evaluated for crop tolerance, persistence in soils, selectivity, toxicological and environmental effects before the product is marketed for use. A chemical is of little use if it injures the target crop or persists in soils for very long periods of time in areas where extensive crop rotation schemes are practiced. Some herbicides such as those used in citrus are persistent for longer periods than do most chemicals used in agronomic or vegetable crops. Herbicides with extended soil residual periods are extremely valuable in areas where crop rotation is not important (i.e., citrus groves) or in areas where total vegetative control is desired (i.e., fence rows and around out buildings).

Most herbicides, when applied according to label directions, do not exhibit soil carry-over problems. The compounds that may have some carry-over characteristic generally will provide the user with a warning statement on the label. Herbicide injury does not only occur with soil-applied weed control compounds; many times herbicide injury may be

exhibited on susceptible species due to off-site drift of spray material or vaporization. Problems with drift or vaporization have led to the Florida Organo-Auxin Herbicide Rule (see fact sheet SS-AGR-12 *Florida's Organo-Auxin Herbicide Rule*). This rule limits the application of several organo-auxin herbicides with aerial equipment in certain parts of the state and requires specific record keeping and wind speed determinations to be completed before this group of compounds can be applied.

Although isolated cases of herbicide injury may occur when labeled practices are adhered to, most herbicide injury occurs whenever certain errors in rate calculations, spray calibration, chemical selection, drift of sprays or when unusual soil or climatic conditions exist. When herbicide injury does occur, diagnosis is often difficult and somewhat confusing since the symptoms of injury may vary depending on the herbicide, plant species, environment, time or method of application, and stage of growth of the plant. In addition, nutritional problems, physiological disorders, diseases, nematodes, and insects may often cause similar injury to certain herbicide families.

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A fairly good reference publication entitled *Herbicide Injury Symptoms and Diagnosis*, 1978, from the North Carolina State University, shows several herbicide injury symptoms but many times there may not be a picture of injury caused by the particular herbicide with which you are concerned. Many herbicides can be classified into certain families of chemistry which often exhibit similar injury symptoms. Even if you are not familiar with a particular herbicide, you may be able to recognize the symptoms by knowing general injury characteristics that a herbicide exhibits.

The following information is intended to help interested parties classify herbicides by families (See Table 26). General symptoms of herbicide injury are given and may be of help in eliminating certain herbicide families as the probable cause of injury.

Herbicide Families and Injury Symptoms

Family: Amides

Mode of Action and Symptoms: These herbicides are selective preemergence or preplant materials that exhibit little if any translocation within the plant. Amides generally interfere with cell division and inhibit growth in the terminal leaves, shoots, or root meristems. Rainfall or mechanical incorporation is needed for maximum activity. Amides are rapidly degraded upon entry into the soil therefore leaching is minimal. Symptoms vary but are usually associated with root growth inhibition, malformed shoots or leaves, and stunted growth. Within this group, naptalam has the unique property of altering the geotropic response of plants, often resulting in roots growing upward out of the soil. As a general rule, amides control grasses better than broadleaves. See Table 1.

Table 1. Herbicides (Amides)

<u>Trade Name</u>	<u>Common Name</u>
Devrinol	napropamide
Kerb	pronamide
Stam	propanil
Gallery	isoxaben

Family: Amino Acid Derivatives

Mode of Action and Symptoms: Amino acid derivatives (Table 2) are translocated foliar-applied herbicides which interfere with aromatic amino acid synthesis. Treated plants stop growing, wilt, become chlorotic and then necrotic. This is a slow process and may require 10 to 14 days. Trees and shrubs treated with sublethal doses may initiate new leaves which are twisted, curled, or generally malformed. As a group, these compounds are more effective on grasses than broadleaf weeds but they are generally considered to be nonselective foliar herbicides with little or no soil activity. Penetration is fairly slow so rainfall too soon after application may reduce control.

Table 2. Herbicides (Amino Acid Derivatives)

<u>Trade Name</u>	<u>Common Name</u>
Liberty, Finale, Ignite, Rely	glufosinate
Roundup WeatherMax, Rodeo, Glyphos, Glyphomax, Glyphomax Plus	glyphosate
Glypro, Glypro Plus, Glyphosate, Glyphosate Original, Touchdown	
Touchdown 5	trimesium salt of glyphosate

Family: Aryl triazinone

Mode of Action and Symptoms: Sulfentrazone disrupts cell membranes. It is believed that they work similar to the diphenyl ether herbicides which inhibit protoporphyrinogen oxidase (PROTOX) which leads to peroxidation causing the toxic buildup of singlet oxygens. Symptoms include necrosis and death upon exposure to light. Foliar contact with sulfentrazone causes rapid desiccation and necrosis of exposed plant tissue. See Table 3.

Table 3. Herbicides (Aryl triazinone)

<u>Trade Name</u>	<u>Common Name</u>
Spartan	sulfentrazone
Aim	carfentrazone

Family: Aryloxyphenoxy propionate

Mode of Action and Symptoms: Aryloxyphenoxy propionates exhibit postemergence activity on grasses at very low rates. They inhibit acetyl-CoA carboxylase (ACCase), an enzyme required for fatty acid synthesis. This prevents new cell growth. As a general rule, these compounds can be applied in broadleaf crops with little or no injury. Some compounds within this group express soil activity when applied at higher rates. As a group, these compounds are readily translocated from the point of uptake to meristematic activity areas. Grasses damaged usually show symptoms within seven days after application. An initial chlorotic yellowing, and in some cases a reddening of the leaf tissue, is eventually followed by complete chlorosis and necrosis. They are 'rain fast' within one hour of application. Weather appears to have very little effect on their activity as long as susceptible plants are not stressed. Antagonism and reduced control have been noted from tank mixing this group with other herbicides. See Table 4.

Table 4. Herbicides (Aryloxyphenoxy propionates)

<u>Trade Name</u>	<u>Common Name</u>
Assure II	quizalofop-P
Fusilade DX, Fusilade II	fluazifop-P
Hoelon	diclofop-methyl
Whip, Acclaim	fenoxaprop-ethyl

Family: Benzoates

Mode of Action and Symptoms: Pyriithiobac (Table 5) inhibits acetolactate synthase (ALS), also called acetohydroxylated synthase (AHAS), a key enzyme in the biosynthesis of the branched-chain amino acids isoleucine, leucine, and valine. Plant death results from events occurring in response to ALS inhibition, but the actual sequence of phytotoxic processes is unclear. Injury symptoms vary by species and generally include chlorosis and necrosis of the meristematic region followed by general foliar chlorosis, necrosis, and plant death.

Table 5. Benzoates

<u>Trade Name</u>	<u>Common Name</u>
Staple	pyriithiobac

Family: Benzoic Acids

Mode of Action and Symptoms: Benzoic herbicides (Table 6) were first tested in the early 1940s and have auxin-like (growth hormone) properties which result in excessive cellular growth. Benzoics move from leaves to the terminal meristems of leaf, shoot, and root, and can also move in the transpiration stream. In some cases, benzoic herbicides applied to plant foliage may come in contact with the soil and then be absorbed by plant roots as well. Dicamba, especially when used for turf weed control, may be moved into surrounding susceptible ornamental roots and can be translocated to the leaves, thus causing herbicide injury. Secondary effects of these herbicides are thought to interfere with RNA and protein synthesis. Symptoms are much like the phenoxy's but often with more epinasty (leaf cupping).

Table 6. Benzoic Acids

<u>Trade Name</u>	<u>Common Name</u>
Vanquish	dicamba
Clarity, Banvel	

Family: Benzothiadiazoles

Mode of Action and Symptoms: Presently only one commercial herbicide fits in this group, but it is of significant importance. Benzothiadiazoles (Table 7) kill broadleaf weeds and some nutsedges by inhibiting photosynthesis which inhibits chlorophyll from changing light energy to plant food. This class exhibits very little translocation, therefore it can only be used postemergence and requires thorough coverage of susceptible plants to be effective. Common symptoms include chlorotic yellowing followed by total necrosis. A transient leaf bronzing is sometimes evident on snapbeans and soybeans.

Table 7. Herbicides (Benzothiadiazoles)

<u>Trade Name</u>	<u>Common Name</u>
Basagran	bentazon

Family: Bipirydillums

Mode of Action and Symptoms: Bipirydilliums (Table 8) were first discovered in the mid-1950s. They are primarily contact herbicides when applied to green plant tissue. They inhibit photosynthesis which causes total disruption of cell membranes. Very little translocation occurs so thorough coverage is essential for greatest activity. Herbicide molecules carry a strong positive charge and are tightly bound to soil colloidal matter upon contact, resulting in no soil activity. They require the presence of sunlight for activity and plants treated on cloudy days or in the dark will not express symptoms until placed in the light. Besides herbicide uses, the group can also be used as pre-harvest desiccants. Symptoms include total rapid plant necrosis in areas covered with spray particles. It is possible to have one-half of a leaf totally desiccated while the other half may still be green. Herbicides in this family may remain active up to 72 hours when sprayed on plastic mulch. When plants are exposed to an aerosol mist, first symptoms are virus-like, and small necrotic areas will later be evident.

Table 8. Herbicides (Bipirydilliums)

<u>Trade Name</u>	<u>Common Name</u>
Reward, Diquat	diquat
Gramoxone Extra, Gramoxone MAX, Boa	paraquat

Family: Carbamates

Mode of Action and Symptoms: Development of this family (Table 9) closely followed 2,4-D and they are often referred to as carbamates. A few materials in this group are applied preemergence, but most of the newer generation in this group are applied postemergence. Somewhat like the chloroacetamides, the carbamates are meristematic inhibitors which have the ability to translocate. Those in the group that are soil-applied can be taken up by seeds, shoots, and to a lesser degree, roots. Symptoms of injury

include inhibition of root growth, stunted plants, and, when postemergence applied, yellowing and chlorosis.

Table 9. Herbicides Carbamates

<u>Trade Name</u>	<u>Common Name</u>
Asulox	asulam

Family: Chloroacetamides

Mode of Action and Symptoms: Chloroacetamides (Table 10) are thought to disrupt synthesis of very long chain fatty acid synthesis. Chloroacetamides do have the ability to translocate within the transpiration stream from roots to leaves. Chloroacetamides damage on corn may appear as twisted malformed leaves or leafing out underground. Soybean injury appears typically as heart-shaped leaves. Leaf crinkling or cupping and twisting has been seen in several vegetable species. If injury is not severe, plants will recover from symptoms. Chloracetamides will not control plants that have already emerged from the soil.

Table 10. Herbicides (Chloroacetamides)

<u>Trade Name</u>	<u>Common Name</u>
Pennant, Dual II	S-metolachlor
Magnum, Dual	metolachlor
Magnum, Cinch	propachlor
Me-Too-Lachlor, Stalwart	
Ramrod	

Family: Cyclohexanediones

Mode of Action and Symptoms: Cyclohexanediones inhibit acetyl CoA carboxylase (ACCCase), the enzyme catalazing the first committed step in fatty acid synthesis. This blocks the production of phospholipids used in building new membranes required for cell growth. Growth stops soon after application. Leaf chlorosis and eventually necrosis develop within 1-3 wk of application. Leaves eventually turn reddish-purple. See Table 11.

Table 11. Herbicides (Cyclohexanediones)

<u>Trade Name</u>	<u>Common Name</u>
Poast, Poast Plus, Vantage	sethoxydim
Select, Envoy	clethodim

Family: Dinitroanilines

Mode of Action and Symptoms: Most dinitroanilines (Table 12) require soil incorporation to avoid volatilization and/or photodecomposition, which could render them useless as herbicides. Several within this group, i.e., Surflan, Prowl, and Sonalan, are less susceptible to volatility and photodecomposition and may be applied directly to the soil surface. Dinitroanilines are mitotic inhibitors that prevent root growth. Shoot growth may also be inhibited if the herbicide is absorbed by the shoot or may be indirectly affected by reduced root growth. Damage symptoms are generally associated with inhibition of lateral root growth resulting in short, stubby, and/or swelled roots. Carry-over injury to corn or other grass crops typically appears as root pruning, short thickened roots, and red-tinged leaf margins. Injury is often in a distinct pattern due to localized concentrations caused by application or incorporation problems. Stunting and swollen hypocotyls are early injury symptoms in many vegetable crops, specifically cucurbits. Since dinitroanilines act at the root tip, control of emerged plants is generally not observed.

Table 12. Herbicides (Dinitroanilines)

<u>Trade Name</u>	<u>Common Name</u>
Balan	benefin
Prowl, Pendimax 3.3	pendimethalin
Sonalan	ethalfuralin
Surflan	oryzalin
Treflan	trifluralin

Family: Diphenylethers

Mode of Action and Symptoms: Diphenylethers (Table 13) are believed to be inhibitors of protoporphyrinogen oxidase (Protox), an enzyme important in the synthesis of chlorophyll and heme biosynthesis. Lipids and proteins are attacked and oxidized, resulting in a loss of chlorophyll and

carotenoids and in leaky cell membranes which causes cells to rapidly disintegrate. Membrane destruction is due to creation of free oxygen radicals. Translocation within the plant is limited, so adequate spray coverage is essential for control. Broadleaf weeds are affected more than grasses. Activity is usually enhanced by higher volume sprays and the addition of surfactants. Symptoms are generally expressed on the foliage as a contact burn with "bronzing" or necrosis. Goal, among others, has a good deal of preemergence activity. Symptoms include crinkling and cupping, most times downward, and with interveinal chlorosis at higher rates.

Table 13. Herbicides (Diphenylethers)

<u>Trade Name</u>	<u>Common Name</u>
Ultra Blazer	acifluorfen
Cobra	lactofen
Goal	oxyfluorfen

Family: Imidazolinones

Mode of Action and Symptoms: Imidazolinones (Table 14) are meristematic inhibitors which interfere with amino acid synthesis. This group of herbicides has the same site of action as the sulfonylureas. Both groups inhibit acetolactate synthase (ALS) or acetoxy acid synthase (AHAS) which is involved with the production of the essential amino acids leucine, isoleucine, and valine. Imidazolinones have both foliar and soil activity and vary greatly in soil persistence. As a general rule, they are more active on broadleaf weeds than grasses and are extremely low in mammalian toxicity. Symptoms include an immediate cessation of growth in susceptible species accompanied by a shortening of internodes and a general compactness of plant growth followed by interveinal chlorosis and eventually necrosis. Purpling of leaves and root pruning may also be observed. Imidazolinones are fairly slow in acting with symptoms lingering three to four weeks or longer in affected plants.

Table 14. Herbicides (Imidazolinones)

<u>Trade Name</u>	<u>Common Name</u>
Arsenal, Chopper	imazapyr
Scepter, Image	imazaquin
Pursuit, Pursuit Plus	imazethapyr
Cadre, Plateau	imazapic
Raptor	imazamox
Assert	imazamethabenz

Family: N-Phenylphthalimide

Mode of Action and Symptoms: The mechanism of action of N-Phenylphthalimides (Table 15) is thought to be similar to that of the diphenylether herbicides. They are believed to be inhibitors of protoporphyrinogen oxidase (Protox), an enzyme important in the synthesis of chlorophyll and heme biosynthesis. Lipids and proteins are attacked and oxidized, resulting in a loss of chlorophyll and carotenoids and in leaky cell membranes which causes cells to rapidly disintegrate. Symptoms of **flumiclorac** may appear within 1 day under bright sunlight and can include wilting and bleaching. Leaves then become brown, desiccated, and necrotic. Sensitive plants emerging from soils treated with the herbicide **flumioxazin** become necrotic and die shortly after exposure to sunlight. Foliar contact with **flumioxazin** causes rapid desiccation and necrosis of exposed plant tissues.

Table 15. N-Phenylphthalimide

<u>Trade Names</u>	<u>Common Name</u>
Resource	flumiclorac
Valor	flumioxazin

Family: Phthalic Acids

Mode of Action and Symptoms: The exact mode of action of the phthalic acids (Table 16) is unknown. **DCPA** may inhibit mitosis by affecting cell wall formation and microtubule arrangement of both root and shoot tips. **Endothall** inhibits messenger RNA, and thus limits protein synthesis. It decreases the rate of respiration and lipid metabolism and interferes with normal cell division. Under both terrestrial and aquatic conditions, **endothall** symptoms include defoliation and brown desiccated tissue.

Table 16. Phthalic Acids

<u>Trade Name</u>	<u>Common Name</u>
dacthal	DCPA
several	endothall

Family: Nitriles

Mode of Action and Symptoms: Two common compounds fit in this category but the two exhibit different symptoms. Dichlobenil (Table 17) is usually applied preemergence to the weeds and acts primarily on the growing points of shoots and roots and usually results in swelling or collapse of stems, roots, and leaf petioles. In certain cases, marginal leaf chlorosis also may be observed. Dichlobenil has activity on germinating plants as well as shallow-rooted weeds which may have already germinated before application. Bromoxynil is the second common member of this family and is usually applied postemergence. It exhibits contact type activity on broadleaf weeds with little translocation. Bromoxynil acts as a photosynthetic inhibitor and results in rapid desiccation and necrosis of treated plants. Bromoxynil is registered in grain crops and seedling turf, and BXN cotton for postemergence broadleaf control.

Table 17. Herbicides (Nitriles)

<u>Trade Name</u>	<u>Common Name</u>
Buctril	bromoxynil
Casoron	dichlobenil

Family: Organic Arsenicals

Mode of Action and Symptoms: Organic arsenicals (Table 18) are an old family of herbicides generally thought to inhibit malic enzyme. This leads to a build up of malic acid and eventually cellular lysis. They are used in cotton and the turf industry as postemergence foliar compounds to control grasses and some broadleaf weeds. Symptoms include leaf chlorosis followed by necrosis. Leaves may exhibit a slight purple color before total necrosis occurs.

Table 18. Herbicides (Organic Arsenicals)

<u>Trade Name</u>	<u>Common Name</u>
(several trades)	MSMA
(several trades)	DSMA

Family: Phenoxys

Mode of Action and Symptoms: Phenoxys (Table 19) are a relatively old group of compounds that date back to the 1940s. The first herbicide of this group to be introduced was 2,4-D. Herbicides in this family have auxin-like properties which result in excessive cellular growth with symptoms appearing as abnormal growth of the plant. The first symptom of injury is usually stem twisting followed by deformities in terminal tissue which may lead to cupping or strapping of the leaves and total bending and twisting of the stems. Phenoxys are usually foliarly applied and are translocated within the food stream of plants. In some cases, phenoxys applied at higher rates may also exhibit soil activity on emerging broadleaf seedlings.

Table 19. Herbicides (Phenoxys)

<u>Trade Name</u>	<u>Common Name</u>
Several	2,4-DB
Several	2,4-D

Family: Substituted ureas

Mode of Action and Symptoms: These herbicides (Table 20) are classic photosynthetic inhibitor herbicides which cause disruption of cell membranes. They are relatively nonselective at high rates. Most are applied to the soil although a few have foliar activity as well. Substituted urea damage symptoms are generally interveinal chlorosis followed by necrosis. Root growth is not inhibited. Injury from this family of compounds is often difficult to separate from injury caused by compounds within the triazine family.

Table 20. Herbicides (Substituted ureas)

<u>Trade Name</u>	<u>Common Name</u>
Cotoran, Meturon	fluometuron
Spike	tebuthiuron
Karmex, Direx	diuron
Lorox	linuron

Family: Picolinic Acids

Mode of Action and Symptoms: Herbicides in this group (Table 21) are very active auxin-like compounds which have excellent activity on broadleaf weeds. They have been termed "super-phenoxys" by some researchers because they exhibit similar damage symptoms at much lower use rates than phenoxys. Picolinic acids are hormone disruptors which translocate in both the phloem and xylem. They are excellent for the control of perennial broadleaf weeds and brush. Unlike the phenoxys, this group has significant soil activity. Currently, there are no picolinic acid

Table 21. Herbicides (Picolinic Acids)

<u>Trade Name</u>	<u>Common Name</u>
Garlon, Remedy	triclopyr
Stinger, Transline	clopyralid

Family: Pyridines and Pyridazinones

Mode of Action and Symptoms: This herbicide group (Table 22) is active on both grasses and broadleaf weeds and at higher rates may exhibit extended soil activity. These compounds are normally applied preemergence to weeds and are translocated throughout susceptible plants. Pyridazinones and pyridinones are quite compatible with a wide range of PPI, PRE, and POST applied herbicides. These compounds are carotenoid inhibitors and cause bleaching of green tissue. Norflurazon exhibits interveinal bleaching while clomazone exhibits intraveinal bleaching. Lethal doses will first appear a chlorotic white and then progress to total necrosis. Sub-lethal doses may leave plants in the white stage for an extended period of time.

Table 22. Herbicides (Pyridines and Pyridazinones)

<u>Trade Name</u>	<u>Common Name</u>
Solicam, Zorial	norflurazon
Sonar	fluridone
Dimension	dithiopyr

Family: Sulfonylureas

Mode of Action and Symptoms: Sulfonylureas were (Table 23) first reported in the early 1970s. Sulfonylureas are meristemic inhibitors with both foliar and soil activity. Sulfonylureas inhibit acetolactate synthase (ALS), a key enzyme in the production of amino acids leucine, isoleucine, and valine for plant growth (same mode of action as imidazolinones). Sulfonylureas are extremely bioactive with application rates of less than one-half ounce per acre in most cases. In some areas of the country, sulfonylureas persist in the soil long enough to cause damage to sensitive rotational crops. Generally, soils higher in pH and O.M. in cooler climates have shown the most recropping problems. Symptoms of injury include an immediate cessation of growth, shortened internodes, chlorotic yellowing, and a gradual necrosis of leaf and stem tissue. Compounds within this family have extremely low acute and chronic mammalian toxicities and are very safe in the environment.

Table 23. Herbicides (Sulfonylureas)

<u>Trade Name</u>	<u>Common Name</u>
Ally, Escort	metsulfuron-methyl
Classic	chlorimuron-ethyl
Glean, Telar	chlorsulfuron
Oust	sulfometuron-methyl
Sempre, Permit, Manage	halosulfuron
Matrix	rimsulfuron
Accent	nicosulfuron
Londax	bensulfuron
Peak	prosulfuron

Family: Thiocarbamates

Mode of Action and Symptoms: Thiocarbamates (Table 24) also act as a type of meristematic inhibitor but have several different characteristics from

previously discussed families. Thiocarbamates are relatively volatile and all thiocarbamates are soil incorporated to reduce surface loss. These herbicides inhibit both cell division and elongation, fatty acid and lipid biosynthesis, proteins, and also may alter plant hormone distribution within plants. Uptake occurs through seeds, shoots, or roots. Shoots are more affected than roots. Thiocarbamates act primarily by inhibiting growth of shoots of emerging seedlings. The major symptom in grasses is the failure of the leaves to emerge properly from the coleoptile. When this occurs, the growing leaf often forms a loop called "buggy whipping." In broadleaf plants, the seedling leaves often stick together or may be cupped or crinkled with necrotic edges. Thiocarbamates generally display a very strong pungent odor during application and for a period of time in the field thereafter.

Table 24. Herbicides (Thiocarbamates)

<u>Trade Name</u>	<u>Common Name</u>
Bolero	thiobencarb
Eptam	EPTC
Sutan + Tillam	butylate pebulate

Family: Triazines

Mode of Action and Symptoms: Triazines (Table 25) were first tested for weed control in the early 1950s and are used primarily as preemergence soil-applied treatments. Some products in this family are also used widely for postemergence applications. Triazines are photosynthetic inhibitors and symptoms are generally leaf chlorosis followed by necrosis. Generally, outer leaf margins of lower leaves are most affected and if the entire leaf turns yellow some of the veins may remain somewhat green. Soil-applied triazines are taken up by the roots and move within the transpiration stream of the plant. Triazines applied postemergence exhibit an initial contact burn and are very effective on small broadleaves and some weedy grasses.

Table 25. Herbicides (Triazines)

<u>Trade Name</u>	<u>Common Name</u>
AAtrex	atrazine
Caparol	prometryn
Evik	ametryn
Sencor	metribuzin
Pramitol	prometon
Princep	simazine
Velpar	hexazinone

Family: Triazolopyrimidines

Mode of Action and Symptoms: The triazolopyrimidines (Table 26) inhibit acetolactate synthase (ALS), also called acetoxyhydroxylated synthase (AHAS), a key enzyme in the biosynthesis of the branched-chain amino acids isoleucine, leucine, and valine. Plant death results from events occurring in response to ALS inhibition, but the actual sequence of phytotoxic processes is unclear. Most sensitive weed species are killed before emergence following soil applications of the triazolopyrimidines, but weeds may die after emergence under some conditions. Emerged sensitive species exhibit stunting, growing point effects, interveinal discoloration (purpling) and necrosis within 1-3 weeks. Postemergence symptoms following **chloransulam** applications usually occur within 3-10 days. These symptoms include stunting, growing point inhibition, and chlorosis followed by necrosis. Complete death of susceptible weeds occurs within 2-3 weeks.

Table 26. Triazolopyrimidines

<u>Trade Name</u>	<u>Common Name</u>
First Rate	chloransulam
Python	flumetsulam
Strongarm	diclosulam

Family: Uracils

Mode of Action and Symptoms: Uracils (Table 27) are also photosynthetic inhibitors which, like the triazines and phenylureas, block the Hill reaction. These herbicides are usually soil-applied and move

within the transpiration stream in plants. Uracil herbicides are used extensively in citrus and probably more total pounds of this family are used in Florida than any of the other herbicide families. Citrus is very tolerant to the uracils and, at rates of 3 to 5 pounds per application, good residual weed control can be obtained. Being photosynthetic inhibitors, symptoms of this group are generally leaf chlorosis followed by necrosis. Chlorosis is often first noticed in the leaf veins and later spreads to the interveinal areas.

Table 27. Herbicides (Uracils)

<u>Trade Name</u>	<u>Common Name</u>
Hyvar X, Hyvar X-L	bromacil
Sinbar	terbacil

Other Herbicides - No Family

Mode of Action and Symptoms: Bensulide (Table 29) inhibits root elongation or partially inhibits cell division (mitosis). The exact mechanism of action is not known. The mechanism of action of clomazone (Table 29) is not completely understood, but apparently inhibits an enzyme in the isoprenoid pathway after farnesyl pyrophosphate. Susceptible seedlings usually emerge from soils treated with clomazone, but are bleached white and become necrotic after several days. Susceptible species in later growth stages may exhibit foliar bleaching when treated postemergence or when exposed to clomazone vapor, drifting from nearby treated areas. The mechanism of action for quinclorac (Table 29) is not completely understood. In susceptible broadleaves, quinclorac action appears to be similar to that of native auxin (IAA). In susceptible grasses, however, quinclorac may inhibit an enzyme associated with cellulose biosynthesis. Its effect on grasses may also be due to increases in ethylene and cyanide production. In certain susceptible broadleaf plants, symptoms of quinclorac may resemble those of auxin-type herbicides, including mild epinastic bending of stems and petioles, stem swelling (particularly at nodes) and elongation, and leaf cupping or curling. This is followed by growth inhibition, chlorosis at the growing points, wilting, and necrosis. In susceptible grasses, rapid chlorosis begins in a band at the zone of elongation in newly

expanding leaves, followed by general foliar chlorosis and necrosis.

Table 28. Unclassified

<u>Trade Name</u>	<u>Common Trade</u>
Prograss	ethofumesate

Table 29. None

<u>Trade Name</u>	<u>Common Name</u>
several	bensulide
Command	clomazone
Drive	quinclorac

Table 30. Herbicides and associated families

Herbicide	Family	Herbicide	Family
A			
AAtrex	triazine	Accent	sulfonylurea
Acclaim	aryloxyphenoxy propionate	Aim	aryl triazinone
acetochlor + safener	chloroacetamide	acifluorfen	diphenylether
Alanap	amide	Ally	sulfonylurea
Ally	sulfonylurea	ametryn	triazine
Arsenal	imidazolinone	Assert	imidazolinone
Assure II	aryloxyphenoxy propionate	asulam	carbamate
Asulox	carbamate	atrazine	triazine
Authority	aryl triazinone		
B			
Balan	dinitroaniline	Banvel	benzoic acid
Basagran	benzothiadiazole	Beacon	sulfonylurea
benefin	dinitroaniline	bensulide	none
bentazon	benzothiadiazole	Bolero	thiocarbamate
bromacil	uracil	bromoxynil	nitrile
Buctril	nitrile	Butoxone	phenoxy
butylate	thiocarbamate	Butyrac	phenoxy
Ultra Blazer	diphenylether	Boa	bipyridilium
C			

Table 30. Herbicides and associated families

Herbicide	Family	Herbicide	Family
Cadre	imidazolinone	Caparol	triazine
Casoron	nitrile	chlorimuron-ethyl	sulfonylurea
chlorsulfuron	sulfonylurea	Chopper	imidazolinone
chloransulam	triazolopyrimidine	Classic	sulfonylurea
clethodim	cyclohexanedione	clomazone	pyridazinone, pyridinone
clopyralid	picolinic acid	Cobra	diphenylether
Command	pyridazinone, pyridinone	Cotoran	substituted urea
Clarity	benzoic acid	cycloate	thiocarbamate
D			
DCPA	phthalic acid	dacthal	phthalic acid
DSMA	organic arsenical	Dimension	pyridazinone
desmedipham	carbanilate, phenyl carbamate	Devrinol	amide
dicamba	benzoic acid	dichlobenil	nitrile
diclofop-methyl	aryloxyphenoxy propionate	diquat	bipyridilium
diuron	substituted urea	diclosulam	triazolopyrimidine
Dual Magnum	chloroacetamide	Dual II Magnum	chloroacetamide
Drive	none	2,4-D	phenoxy
2,4-DB	phenoxy		
E			
Envoy	cyclohexanedione	endothall	phthalic acids
EPTC	thiocarbamate	Eptam	thiocarbamate
Eradicane	thiocarbamate	ethalfuralin	dinitroaniline
Evik	triazine	Escort	sulfonylurea
ethofumesate	unclassified		
F			

Table 30. Herbicides and associated families

Herbicide	Family	Herbicide	Family
fenoxaprop-ethyl	aryloxyphenoxy propionate	Finale	amino acid derivative
fluazifop-P	aryloxyphenoxy propionate	fluometuron	substituted urea
fluridone	pyridazinone, pyridinone	fomesafen	diphenyl ether
Fusilade DX	aryloxyphenoxy propionate	Fusilade II	aryloxyphenoxy propionate
flumioxazin	N-phenylphtalimide	flumichlorac	N-phenylphtalimide
flumetsulam	triazolopyrimidine	First Rate	triazolopyrimidines
G			
Garlon	picolinic acid	Glean	sulfonylurea
glufosinate	amino acid derivative	glyphosate	amino acid derivative
Goal	diphenylether	Gramoxone Extra	bipyridilium
Gramoxone Max	bipyridilium	Gallery	amide
H			
halosulfuron	sulfonylurea	haloxyfop-methyl	aryloxyphenoxy propionate
Harmony Extra	sulfonylurea	hexazinone	triazine
Hoelon	aryloxyphenoxy propionate	Hyvar X, Hyvar XL	uracil
I			
Ignite	amino acid derivative	imazamethabenz	imidiazolinone
imazamox	imidiazolinone	imazapic	imidiazolinone
imazapyr	imidazolinone	imazaquin	imidazolinone
imazethapyr	imidazolinone	isopropalin	dinitroaniline
isoxaben	amide		
K			
Karmex	substituted urea	Kerb	amide
L			
lactofen	diphenylether	Liberty	amino acid derivative

Table 30. Herbicides and associated families

Herbicide	Family	Herbicide	Family
linuron	sub. urea	Lorox	substituted urea
Londax	sulfonylurea		
M			
MCPA	phenoxy	MCPB	phenoxy
MCPP	phenoxy	MSMA	organic arsenical
Matrix	sulfonylurea	mecoprop	phenoxy
metribuzin	triazine	metsulfuron-methyl	sulfonylurea
Milestone	triazolone	Milogard	triazine
Meturon	substituted urea	molinate	thiocarbamate
S-metolachlor	chloroacetamide	Manage	sulfonylurea
N			
napropamide	amide	naptalam	amide
nicosulfuron	sulfonylurea	norflurazon	pyridazinone, pyridinone
O			
Ordram	thiocarbamate	oryzalin	dinitroaniline
Oust	sulfonylurea	oxyfluorfen	diphenylether
P			
Paarlan	dinitroaniline	paraquat	bipyridilium
pebulate	thiocarbamates	pendimethalin	dinitroaniline
Peak	sulfonylurea	Poast, Poast Plus	cyclohexanedione
Pramitol	triazine	Prefar	amide
primisulfuron	sulfonylurea	Princep	triazine
prometon	triazine	prometryn	triazine
pronamide	amide	propachlor	chloroacetamide

Table 30. Herbicides and associated families

Herbicide	Family	Herbicide	Family
propanil	amide	propazine	triazine
Prowl	dinitroaniline	Pursuit, Pursuit Plus	imidiazolinone
Permit	halosulfuron	Plateau	imidiazolinone
Prograss	unclassified	prodiamine	dinitroaniline
Pennant	chloroacetamide	prosulfuron	sulfonylurea
Pendimax 3.3	dinitroaniline	pyrithiobac	
Python	triazolopyrimidine		
Q			
quizalofop-P	arylxyphenoxy propionate	quinclorac	none
R			
Ramrod	chloroacetamide	Raptor	imidazolinone
Reclaim	pyridinoxy, picolinic acid	Reflex	diphenyl ether
Remedy	picolinic acid	Reward	bipyridilium
Resource	N-phenylphtalimide	Ro-Neet	thiocarbamate
Rhomene	phenoxy	rimsulfuron	sulfonylurea
Rodeo	amino acid derivative	Roundup Ultra Max	amino acid derivative
Roundup Ultra	amino acid derivative		
S			
Scepter	imidazolinone	Select	cyclohexanedione
Sempra	sulfonylurea	Sencor	triazine
sethoxydim	cyclohexanedione	siduron	phenylurea, substituted urea
simazine	triazine	Sinbar	uracil
Sodium TCA	chlorinated aliphatic acid	Solicam	pyridazinone, pyridinone
Sonalan	dinitroaniline	Sonar	pyridazinone, pyridinone
Spartan	aryl triazinone	Spike	phenylurea, substituted urea

Table 30. Herbicides and associated families

Herbicide	Family	Herbicide	Family
Stam	amide	Strongarm	triazolopyrimidine
Stinger	picolinic acid	sulfentrazone	aryl triazinone
sulfentrazone + chlorimuron-ethyl	aryl triazinone	sulfometuron-methyl	sulfonylurea
sulfosate	amino acid derivative	Surflan	dinitroaniline
Surpass+	acetochlor + safener	Sutan+	thiocarbamate
Staple	benzoate		
T			
tebuthiuron	substituted urea	terbacil	uracil
thiobencarb	thiocarbamate	Tillam	thiocarbamate
Telar	chlorsulfuron	Transline	picolinic acid
Touchdown	amino acid derivative	Treflan	dinitroaniline
tribenuron +thifensulfuron	sulfonylurea	triclopyr	picolinic acid
trifluralin	dinitroaniline	Triflusulfuron	sulfonylurea
Tupersan	phenylurea, substituted urea	Touchdown 5	amino acid derivative
U			
Upbeet	sulfonylurea		
V			
Valor	N-phenylphthalimide	Vanquish	benzoic acid
Velpar	triazine	Verdict	aryloxyphenoxy propionate
Vantage	cyclohexanediones		
W			
Whip	aryloxyphenoxy propionate		
Z			
Zorial	pyridazinone, pyridinone		