HERBICIDE MECHANISMS OF ACTION
SUMMARY

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Herbicides are substances used to perform weed control. This control occurs through the inhibition of biochemical processes that are essential for the plants to develop.

The herbicides can be classified according to the type of application (pre-emerging, post-emergent or pre-planting incorporated), selectivity (selective or non-selective), Mobility (systemic or contact) and mechanism of action.

Mechanism of action concerns the biochemical process which is disrupted in the presence of the herbicide. Currently there are herbicides that function in 26 distinct sites of action. However, the most commonly used herbicides nowadays compromise 11 mechanisms of action, presented in Table 1.

Most of the interrupted biochemical processes occur in organelles known as chloroplasts, where it occurs the most important process in plants: photosynthesis. Among these herbicides are the ACCase inhibitors, ALS, EPSPS, Protox, PS I, PS II and carotenoids. The other herbicides, including auxin mimics, inhibitors of tubulin polymerization and LCFA, work outside the chloroplast, interrupting processes that aren’t directly related to the photosynthetic reactions.

### Herbicide mechanisms of action

- **ACCase Inhibitors (Acetyl-CoA Carboxylase)**
- **ALS Inhibitors (Acetolactate Synthase)**
- **EPSP Inhibitors (5-enolpyruvylshikimate 3-phosphate synthase)**
- **Auxin Mimics**
- **Protox Inhibitors (Protoporphyrinogen Oxidase)**
- **PS I Inhibitors (Photosystem I)**
- **GS Inhibitors (Glutamine Synthetase)**
- **PS II Inhibitors (Photosystem II)**
- **Carotenoid Biosynthesis Inhibitors**
- **Tubulin polymerization inhibitors**
- **Synthesis Inhibitors (Long Chain Fatty Acid) LCFA**

*Table 1 – Main Herbicide Mechanisms of action*
Enzyme acetyl-CoA carboxylase is responsible for fatty acids production, important components of the cells membranes and organelles. As its name implies, this enzyme is responsible for the carboxylation of acetyl-CoA molecules. After other reactions occur the production of malonyl-CoA, which is a lipids leading which will be a constituents of membranes. In herbicide presence, carboxylation reaction of acetyl-CoA does not occur, interrupting fatty acids production, and consequently membrane production and plant growth.

Herbicides ACCase inhibitors are post-emerging graminicides, selective for eudicotyledons cultures. Therefore they can be used in narrow-leaved weed (sourgrass, annual/Italian ryegrass, southern crabgrass, alexandergrass, among others), broadleaf weeds like soybean, common bean, cotton plantation etc. In some cases they are used in narrow-leaf (winter grains), but selectivity is achieved by safeners (protective compounds that maximize plant defense).

These herbicides can be subdivided into three chemical groups: aryloxyphenoxypropanoates (FOPs), cyclohexanediones (DIMs) and phenylpyrazolines (DENs). Some representatives of each chemical group can be seen in Table 2.

There are in the market some formulations containing two ACCase inhibitors, which usually occur by blending a FOP with a DIM, such as Fenoxaprop-p-ethyl + Clethodim. Although these herbicides inhibit the same site of action, the enzyme points to which they bind are not exactly the same, which results in increased enzyme inhibition, consequently greater control efficiency and increasing action spectrum. Symptoms in treated plants involve initially purpling and/or leaf blade chlorosis of plants of narrow-leaved leaves, followed by necrosis. A typical symptom caused by inhibitors of ACCase is the easy detachment of the central leaf of grasses, known as “dead-heart.” This is due to the lack of fatty acids and new membranes at the growth plant point. In broadleaf and cyperaceous plants named nutgrass, also “tiriricas”, no symptoms are observed due to the presence of an insensitive (eukaryotic) form of ACCase, which is absent in the poaceae (grasses).
## Chemical Groups and main inhibitors of ACCase enzyme

<table>
<thead>
<tr>
<th>Chemical group</th>
<th>Active Ingredient</th>
<th>Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aryloxyphenoxypropionate (FOPs)</td>
<td>Cyhalofop-butyl</td>
<td>Clincher</td>
</tr>
<tr>
<td></td>
<td>Clodinafop-propargyl</td>
<td>Topik 240 EC</td>
</tr>
<tr>
<td></td>
<td>diclofop-methyl</td>
<td>Iloxan CE</td>
</tr>
<tr>
<td></td>
<td>Fenoxaprop-ethyl</td>
<td>Podium EW</td>
</tr>
<tr>
<td></td>
<td>Fluazifop-p-butyl</td>
<td>Fusilade 250 EW</td>
</tr>
<tr>
<td></td>
<td>Haloxyfop methyl</td>
<td>Verdict R</td>
</tr>
<tr>
<td></td>
<td>Quizalofop-p-ethyl</td>
<td>Targa 50 EC</td>
</tr>
<tr>
<td></td>
<td>Butroxydim</td>
<td></td>
</tr>
<tr>
<td></td>
<td>clethodim</td>
<td></td>
</tr>
<tr>
<td>Cyclohexanedione (DIMs)</td>
<td>Profoxydim</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sethoxydim</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tepraloxydim</td>
<td></td>
</tr>
<tr>
<td>Phenylpyrazolin (DENs)</td>
<td>Pinoxaden</td>
<td>Axial</td>
</tr>
</tbody>
</table>

*Table 2*
ALS enzyme inhibitors herbicides interrupt two reactions that result in the production of valine amino acids, leucine and isoleucine, from molecules of pyruvate and Ketobutyrate. Inhibition occurs by herbicide binding to the channel that gives access to the catalytic site of the enzyme, interrupting the entrance of substrates used in the reactions. These amino acids are essential in the synthesis of proteins and inhibition of its production results in plants death.

ALS enzyme Inhibitors herbicides establish the mechanism of action with the largest available number of active ingredients, which are subdivided into five chemical groups: sulfonylureas (SU), Imidazolinones (IMI), pyrimidylthiobenzoates (PTB), triazolopyrimidines (TPD) and sulfonlamine carbonyl triazolinones (SCT). Some herbicides belonging to these chemical groups are represented in table 3.

Even as to ACCase inhibitors, there are formulated mixtures of active ingredients. In case of ALS inhibitors, the mixture of two imidazolinones, imazapique + imazethapyr (Only), for example, increases the control spectrum and efficiency. Although they inhibit the same enzyme and belong to the same chemical group, the herbicide-enzyme links are not exactly the same in all species, justifying these types of mixtures.

ALS-inhibitors herbicides selectivity is difficult to predict only by the chemical group. This means that within the same group there are selective herbicides for various crop species, which is due to the different ability of each crop in metabolizing a herbicide in particular. For example sulfonylureas chlorimuron (Classic) and nicosulfuron (Sanson) are post-emergence selective of soybean and corn/maize crops, respectively.

ALS inhibitor herbicides are for the most part systemic post-emergent. However some present a prolonged residual effect period (greater than 30 days) and others are pre-emergent with little effect on foliar applications such as the diclosulam. ALS-inhibiting herbicides control both broadleaf and narrow-leaved weeds depending of the herbicide. The symptoms begin with chlorosis in the narrow-leafed the between the veins from the first week after the application, followed by necrosis. Purpling vein is common in broadleaf, along with chlorosis and necrosis of the leaves.
## Chemical Groups and main ALS enzyme inhibitors herbicide

<table>
<thead>
<tr>
<th>Chemical Group</th>
<th>Active Ingredient</th>
<th>Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphonylureas (SU)</td>
<td>Chlorimuron-ethyl</td>
<td>Classic</td>
</tr>
<tr>
<td></td>
<td>Ethoxysulfuron</td>
<td>Gladium</td>
</tr>
<tr>
<td></td>
<td>Iodosulfuron-methyl</td>
<td>Hussar</td>
</tr>
<tr>
<td></td>
<td>Metsulfuron-methyl nicosulfuron</td>
<td>Ally</td>
</tr>
<tr>
<td></td>
<td>pyrazosulfuron-ethyl trifloxsulfuron-sodic</td>
<td>Sanson 40 SC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sirius</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Envoke</td>
</tr>
<tr>
<td>Imidazolinones (IMI)</td>
<td>Imazapyr</td>
<td>Contain</td>
</tr>
<tr>
<td></td>
<td>imazapic</td>
<td>Plateau</td>
</tr>
<tr>
<td></td>
<td>Imazaquin</td>
<td>Topgan</td>
</tr>
<tr>
<td></td>
<td>Imazethapyr</td>
<td>Pivot</td>
</tr>
<tr>
<td>Pyrimidylthiobenzoates (PTB)</td>
<td>Bispiribaque-sodic</td>
<td>Nominne 400 SC</td>
</tr>
<tr>
<td></td>
<td>Piritiobaque</td>
<td>Staple 280 CS</td>
</tr>
<tr>
<td>Triazolopyrimidines (TPD)</td>
<td>Cloransulam-methyl</td>
<td>Pacto</td>
</tr>
<tr>
<td></td>
<td>Diclosulam</td>
<td>Spider 840 WG</td>
</tr>
<tr>
<td></td>
<td>Flumetsulam</td>
<td>Scorpion</td>
</tr>
<tr>
<td></td>
<td>Penoxsulam</td>
<td>Ricer</td>
</tr>
<tr>
<td>Sulphonylamine carbonyl triazolinones (TCS)</td>
<td>Flucarbazone-sodic</td>
<td>Not Available in BR</td>
</tr>
</tbody>
</table>

*Table 3*
This mechanism of action is represented by glyphosate, systemic and non-selective herbicide; post-emergent with no residual remaining in the soil.

In the absence of the herbicide, EPSPS enzyme catalyzes the reaction between shikimate and phosphoenol-pyruvate which culminates in precursors of the amino acids tyrosine production, phenylalanine and tryptophan which are essential for plants. When sprayed on plants, glyphosate binds to the EPSPS enzyme, occupying the binding site where phosphoenol-pyruvate molecule should bind, interrupting the reaction.

For being non-selective, glyphosate is used in desiccations in pre-sowing cultivation in order to eliminate all weeds present in the area. Further, it can be used in post-emergence of resistant crops (RR), which carry a gene that produces EPSPS insensitive to the herbicide. There are soybean, corn/maize, cotton and canola cultivars that have this technology.

Symptoms appear after a week, with chlorosis followed by necrosis. The total death of plants is variable, and may take from 10 to more than 30 days. Due to the emergence of some species resistant to glyphosate, it is recommended to include other mechanisms of action in weed control planning. Even with cases of resistance, due to the broad spectrum of action, Glyphosate is still the most used killer-weed control herbicide.
Auxin mimics were the first synthetic herbicides introduced to the market in the late 1940s, represented by 2,4-D. Its importance is not only related to the weed control provided at that time. Due to the stimulus generated by the success of the 2, 4-D, the industry has invested in research that reflects to date the availability of various herbicides molecules. Auxins such as idoleacetic acid (IAA) are hormones found naturally in plants. The Auxinic herbicides mimics (imitate) the effects of natural auxins. However, when in excess they cause physiological disorders that lead the plants to death.

Although they are first herbicides developed, elucidation of the mechanism of action is not yet complete. However, it is known that these herbicides act in various processes, once they unsettle the hormonal plant balance. Among the main effects caused by synthetic auxins are their interference in cell division elongation, due mainly to the large ethylene production. Other hormones, such as gibberellins, cytokinins and abscisic acid also have their balance altered accordingly to these herbicides.

Symptoms caused in plants treated with auxin mimics include epithelial (twisting) of stems, leaves and petioles right after the first days of application. Then, chlorosis and necrosis of the leaves occur, and the death of plants is delayed, occurring from the third week after application. Epithelial occurs in response to increased ethylene production. The selectivity in monocotyledons is due to the vascular bundles organization limiting the translocation besides the occurrence of herbicides metabolism.

Auxin Mimics (Table 4) are selective post-emergent herbicides, and it is common to observe residual effect, although short in most cases. However, herbicides such as picloran can act for several months in the soil. They are selective herbicides to paeceous crops (cereals and pastures) and are indicated for the broadleaf weed control. There are some exceptions, like barnyardgrass control with quinclorac.
### Chemical Groups and main auxin mimics herbicides

<table>
<thead>
<tr>
<th>Chemical group</th>
<th>Active Ingredient</th>
<th>Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzoic Acids</td>
<td>Dicamba</td>
<td>Alectra</td>
</tr>
<tr>
<td>Phenoxyaliphatic Acid</td>
<td>2,4-D</td>
<td>DMA 806 BR</td>
</tr>
<tr>
<td>Carboxylic acid</td>
<td>Fluroxypyr-metyl</td>
<td>Starane 200</td>
</tr>
<tr>
<td></td>
<td>Picloran</td>
<td>Padron</td>
</tr>
<tr>
<td></td>
<td>Triclopyr</td>
<td>Garlon 480 BR</td>
</tr>
<tr>
<td>Quinolone carboxylic acid</td>
<td>Quinclorac</td>
<td>Facet</td>
</tr>
</tbody>
</table>

*Table 4*
PROTOX enzyme is responsible for enzymatic oxidation of protoporphyrinogen IX which results in the protoporphyrin IX production though is precursor of chlorophyll and heme compounds. In herbicides PROTOX inhibitors presence this reaction is inhibited. However, it is not the lack of chlorophyll that causes plant death since treated plants do not present chlorosis.

With inhibition of the reaction occurs protoporphyrinogen IX accumulation in the chloroplast, which exudes to the cytoplasm. In the cytoplasm this compound is converted to protoporphyrin IX through non-enzymatic reactions. Protoporphyrin IX then accumulates in the cytoplasm and in the presence of light and O2 occurs reactive oxygen species production (ROS), such as singlet oxygen (1 O2). ROS is highly toxic to the cell membranes causing them to collapse by oxidative stress. Thus, the best herbicide efficiency occurs after exposure of treated plants to light.

PROTOX inhibitors herbicide are selective and contact herbicides (except saflufenacil, which features mobility by phloem), with action predominantly on broadleaf weeds. In Brazil there are 10 active ingredients subdivided into five chemical groups (Table 5). Due to oxidative stress and cellular extravasation the symptoms can be noticed few hours after absorption which also occurs rapidly. Symptoms include oily spots presence, bleaching, desiccation and leaf necrosis.
## Chemical groups and main PROTOX inhibitors herbicide

<table>
<thead>
<tr>
<th>Chemical group</th>
<th>Active Ingredient</th>
<th>Commercial Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphenyl ethers</td>
<td>Acifluorfen-sodium</td>
<td>Blazer Sol</td>
</tr>
<tr>
<td></td>
<td>Fomesafen</td>
<td>Flex</td>
</tr>
<tr>
<td></td>
<td>Lactofen</td>
<td>Cobra</td>
</tr>
<tr>
<td></td>
<td>Oxyfluorfen</td>
<td>Galigan 240 EC</td>
</tr>
<tr>
<td>Phthalimide</td>
<td>Flumicorac</td>
<td>Radiant 100</td>
</tr>
<tr>
<td></td>
<td>Flumioxazin</td>
<td>Flumyzin 500</td>
</tr>
<tr>
<td>Triazolinone</td>
<td>Carfentrazone-ethyl</td>
<td>Aurora</td>
</tr>
<tr>
<td></td>
<td>Sulfentrazone</td>
<td>Boral 500 SC</td>
</tr>
<tr>
<td>Oxadiazoles</td>
<td>Oxadiazon</td>
<td>Ronstar SC</td>
</tr>
<tr>
<td>Pyrimidinedione</td>
<td>Saflufenacil</td>
<td>Heat</td>
</tr>
</tbody>
</table>

*Table 5*
Photosystems are present in membranes of thylakoids in chloroplasts and comprise a series of proteins and molecules that act as electron carriers (e-), such as quinones A and B (Qa and Qb). PS II e-originated from the breakdown of water molecules are excited by sunlight energy captured by chlorophyll molecules and transported toward PS I by chain carrier e- for energy production (NADPH) that will be used in other reactions of photosynthesis. In the presence of PS II Inhibitors herbicides this transport is interrupted exactly in the region where the quinones are found and not occurring transition of e- to PS I.

Although blocking occurs in e- transport between photosystems, this is not the primary cause of plants treated death. With interruption of electron transport chain the molecules of chlorophyll of PS I become excessively energized, reacting with molecular oxygen (O2), producing reactive oxygen species. In this way plants die from oxidative stress. Herbicides PS II inhibitors can be subdivided into three chemical groups as shown in table 6.

PS II inhibitors herbicides are pre-emergent selective, although they also present post-initial emergency action. Preferentially they control broadleaf weeds, although they have effects in some poaceae species. Symptoms show chlorosis and necrosis. In some cases, due to oxidative stress caused by ROS, there is tissues necrosis without chlorosis.
Chemical Groups and main Inhibitors PS II herbicides (Photosystem II)

<table>
<thead>
<tr>
<th>Chemical group</th>
<th>Active Ingredient</th>
<th>Commercial Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triazine</td>
<td>Ametryn</td>
<td>Ametrina Alta 500 SC</td>
</tr>
<tr>
<td></td>
<td>Amicarbazone</td>
<td>Dinamic</td>
</tr>
<tr>
<td></td>
<td>Atrazine</td>
<td>Gesaprim GrDa</td>
</tr>
<tr>
<td></td>
<td>Metribuzin</td>
<td>Sencor 480</td>
</tr>
<tr>
<td></td>
<td>Simazine</td>
<td>Herbazin 500 BR</td>
</tr>
<tr>
<td>Urea</td>
<td>Diurom</td>
<td>Karmex 800</td>
</tr>
<tr>
<td></td>
<td>Linuron</td>
<td>Alafon SC</td>
</tr>
<tr>
<td></td>
<td>Tebuthiuron</td>
<td>Combine 500 SC</td>
</tr>
<tr>
<td>Uracil</td>
<td>Bromacil</td>
<td>Uragan 800 WP</td>
</tr>
</tbody>
</table>

*Table 6*
Photosystem I is responsible for important reactions in photochemical phase of photosynthesis, more precisely in the final stages of electron chain transport (e-). It is from the PS I that NADPH molecules are produced which serve as energy for the reduction of carbon in biochemical phase of photosynthesis. PS I inhibitors herbicides are strong cations that divert electrons in the electron transport chain in the PS I forming radicals bipyridyls (herbicide + e-). These compounds are unstable and in light presence they react with molecules of O2 forming ROS which cause lipid peroxidation present in the membranes of cells and organelles.

PS I inhibitors herbicides are non-selective post-emergent, with no residual action. They are immobile herbicides in plants, requiring good spray coverage. Its efficiency is greater in small plants or in sequential applications to systemic herbicides. These herbicides uptake is very fast; taking less than an hour. Symptoms appear quickly, with striking tissue necrosis due to oxidative stress and extravasation of the cellular contents caused by these herbicides.
Carotenoids are protective pigments responsible for dissipating the excess energy in photosystems, protecting chlorophylls against photodegradation. Its production is higher in hours of greater light intensity close to midday. These pigments are produced by a route involving several enzymes, including DXS enzymes (1-deoxy-d-xylulose-5 phosphate synthase), HPPD (94-hydroxyphenyl-pyruvate dioxygenase) and FDS (phytoene desaturase) which are inhibited by carotenoids inhibitors herbicides. With the inhibition of these enzymes there is no carotenoid biosynthesis in young tissues leading to the chlorophyll photodegradation.

Carotenoid inhibitors are pre-emergent herbicides that act mainly in broadleaf weeds. They can be classified accordingly to the enzyme that is inhibited, as shown in table 8.

Symptoms in plants treated with carotenoid inhibitors occur after germination of seedlings being one of the most striking among all herbicides. Due to chlorophyll photodegradation young tissues become extremely albinos. Following, it occurs the tissue necrosis and plants death.
## Chemical Groups and main Carotenoid Biosynthesis Inhibitors Herbicides

<table>
<thead>
<tr>
<th>Inhibited Enzyme</th>
<th>Active Ingredient</th>
<th>Commercial Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>DXS</td>
<td>Clomazone</td>
<td>Gamit</td>
</tr>
<tr>
<td>HPPD</td>
<td>Isoxaflutole</td>
<td>Provence 750 WG</td>
</tr>
<tr>
<td></td>
<td>Mesotrione</td>
<td>Callisto</td>
</tr>
<tr>
<td></td>
<td>Tembotrione</td>
<td>Soberan</td>
</tr>
<tr>
<td>FDS</td>
<td>(unavailable in BR)</td>
<td></td>
</tr>
</tbody>
</table>

*Table 8*
GS INHIBITORS

GS enzyme is involved in the production of forms of nitrogen (N) usable by plants. This process involves the production of glutamine from ammonia (NH4 +) and glutamate. Glutamine is understandable by plants and used in amino acids synthesis. In this way when it occurs GS inhibition there is a reduction in the synthesis of amino acids (glutamine, glutamate, aspartate, alanine, glycine and serine) and the accumulation of NH4 +, which is toxic to plants. Also Associated with this is the reduction of photosynthetic activity in plants. With photosynthesis inhibition the excess energy produces ROS, which cause lipid peroxidation.

Glufosinate-ammonium is a post-emergent herbicide with no residual effect. Still, it is considered a contact and non-selective herbicide, except in cases of crop tolerance introduced via biotechnology (Liberty Link crops). Symptoms involve chlorosis followed by leaf necrosis within the first days after application. As well as the other contact herbicides, they are more efficient in plants at the initial stage of growth.

<table>
<thead>
<tr>
<th>Chemical Group</th>
<th>Active Ingredient</th>
<th>Commercial Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphinic acid</td>
<td>Glufosinate-ammonium</td>
<td>Finale Liberty</td>
</tr>
</tbody>
</table>

Table 9
Tubulin is a protein that is found in two forms in cells: α-tubulin and β-tubulin. Its importance is related to the organization of the cytoskeleton and especially in the cell division process, during chromosomes separation and formation of new cell wall. Tubulin polymers are hollow circular filaments formed from interlayers of α-tubulin and β-tubulin. Herbicides act by inhibiting the formation of filament occupying the place of connection between the forms of tubulin. With this the division of the genetic material of cells occurs unevenly and mitosis process is interrupted.

Dinitroanilines Herbicides are absorbed by the roots and because they are immobile, they act inhibiting cell division in the meristem, preventing root growth. As a result, tips of roots swells up a symptom known as “chicken-thigh”, due to the form shape taken by roots. With root system restriction, shoot growth is also impaired in sensitive plants.

Tubulin polymerization inhibitors are selective pre-emergent herbicide that acts in narrow-leafed weed control. Due to high vapor pressure these herbicides need to be incorporated into the soil right after spraying.

### Chemical Group and main Tubulin polymerization inhibitors Herbicides

<table>
<thead>
<tr>
<th>Chemical Group</th>
<th>Active Ingredient</th>
<th>Commercial Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dinitroanilines</td>
<td>Trifluralin</td>
<td>Premerlin 600 EC</td>
</tr>
<tr>
<td></td>
<td>Pendimethalin</td>
<td>Herbadox</td>
</tr>
</tbody>
</table>

Table 10
LCFA SYNTHESIS INHIBITORS

Long chain Fatty acids (LCFA) are produced at the start of enzyme activity of acyl-CoA elongase, from fatty acids up to 18 carbons (18C) produced by ACCase enzyme. The enzyme acyl-CoA elongase as its name suggests is responsible for elongation of fatty acids through addition of carbons. LCFA are was precursors, cutin and suberin besides being membranes components.

Chloroacetamides are pre-selective emerging herbicides that control both broadleaf weeds and narrow-leafed ones. In sensitive monocotyledonous plants that are able to emerge, symptoms are characterized by difficulty in shedding off the leaves of the whorl. In dicotyledons the most remarkable symptom is the shortening of midribs giving a heart shape in these leaves.

### Chemical Group and main LCFA synthesis Inhibitors Herbicides

<table>
<thead>
<tr>
<th>Chemical Group</th>
<th>Active Ingredient</th>
<th>Commercial Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroacetamide</td>
<td>S-metolachlor</td>
<td>Dual Gold</td>
</tr>
<tr>
<td></td>
<td>Acetochlor</td>
<td>Fist CE</td>
</tr>
<tr>
<td></td>
<td>alachlor</td>
<td>Laço EC</td>
</tr>
</tbody>
</table>

*Table 11*
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