Advances in Weed Management
(2015)

SS Rana and MC Rana

Department of Agronomy, Forages and Grassland Management
College of Agriculture, CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur-176062 (India)
Copyright 2014
SS Rana, Sr Scientist, and MC Rana, Prof, Department of Agronomy, Forages and Grassland Management, College of Agriculture, CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur- 176062.

No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise without the written permission of the authors.

Citation
PREFACE

Weeds are probably the most ever-present class of crop pests and are responsible for marked losses in crop yields. Of the total losses caused by pests, weeds have a major share (30%). They reduce the crop yield and deteriorate the quality of produce and hence reduce the market value of the turnout. Therefore, management of weeds in all agro-ecosystems is imperative to sustain our crop productivity and to ensure the food security to the burgeoning population.

There has been a long-felt need for a teaching manual on Advances in Weed Management. This manual is a precise account of various theoretical aspects of weed management presented in a simple language suitable for Agron 606 students. The manual provides useful information on crop-weed competition, weed shifts, mode of action of herbicides, selectivity of herbicides, climate and phytotoxicity of herbicides, fate of herbicides in soil, residue management of herbicides, adjuvants, herbicide application techniques, herbicide resistance, herbicide antidotes, herbicide interactions, transgenic herbicide resistance crops, herbicide development and registration procedures, bioherbicides, allelochemicals and herbicide bioassays.

With all these varied aspects covered in the manual, we hope this will fulfill the requirement of a much needed standard document on Advances in Weed Management not only for the students but also for the teachers, scientists and others involved the field of weed management. The authors would welcome additional information and suggestions from students and teachers to improve the manual.

S.S. Rana
MC Rana
<table>
<thead>
<tr>
<th>SN</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Crop-Weed Competition</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>Weed Shifts</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>Mode of Action of Herbicides</td>
<td>21</td>
</tr>
<tr>
<td>4</td>
<td>Selectivity of Herbicides</td>
<td>38</td>
</tr>
<tr>
<td>5</td>
<td>Climatic Factors and Phytotoxicity of Herbicides</td>
<td>50</td>
</tr>
<tr>
<td>6</td>
<td>Fate of Herbicides in Soil</td>
<td>58</td>
</tr>
<tr>
<td>7</td>
<td>Residue Management of Herbicides</td>
<td>68</td>
</tr>
<tr>
<td>8</td>
<td>Adjuvants</td>
<td>75</td>
</tr>
<tr>
<td>9</td>
<td>Herbicide Application Techniques</td>
<td>90</td>
</tr>
<tr>
<td>10</td>
<td>Herbicide Resistance</td>
<td>100</td>
</tr>
<tr>
<td>11</td>
<td>Herbicide Antidotes</td>
<td>109</td>
</tr>
<tr>
<td>12</td>
<td>Herbicide Interactions</td>
<td>118</td>
</tr>
<tr>
<td>13</td>
<td>Transgenic Herbicide Resistance Crops</td>
<td>126</td>
</tr>
<tr>
<td>14</td>
<td>Herbicide Development</td>
<td>131</td>
</tr>
<tr>
<td>15</td>
<td>Bioherbicides</td>
<td>159</td>
</tr>
<tr>
<td>16</td>
<td>Allelochemicals</td>
<td>163</td>
</tr>
<tr>
<td>17</td>
<td>Herbicide Bioassays</td>
<td>175</td>
</tr>
<tr>
<td></td>
<td>Appendix –I (Structures of Herbicides)</td>
<td>i-xii</td>
</tr>
</tbody>
</table>
Crop-weed competition

Competition is struggle between two or more organisms for a limited resource (Water, nutrient, light and space) that is essential for growth. The Oxford English Dictionary defines competition as “action of endeavouring to gain what another endeavours to gain at the same time; the striving of two or more for the same object; rivalry”. To compete comes from the Latin word competere, which means to ask or sue for the same thing another does. **Competition** in biology, ecology, and sociology, is a contest between organisms, animals, individuals, groups, etc., for territory, a niche, or a location of resources, for resources and goods, mates, for prestige, recognition, awards, or group or social status, for leadership. It is opposite to cooperation. It arises whenever at least two parties strive for a goal which cannot be shared or which is desired individually but not in sharing and cooperation. Competition occurs naturally between living organisms which co-exist in the same environment. For example, animals compete over water supplies, food, mates, and other biological resources. Humans compete usually for food and mates, though when these needs are met deep rivalries often arise over the pursuit of wealth, prestige, and fame. Competition is also a major tenet in market economy and business is often associated with competition as most companies are in competition with at least one other firm over the same group of customers, and also competition inside a company is usually stimulated for meeting and reaching higher quality of services or products that the company produces or develops.

Competition occurs in virtually every ecosystem in nature. This type of relationship develops when more than one organism in an environment requires the same thing in order to survive. When food and shelter are plentiful, there is no competition. It only takes place when there is not enough to go around. Competition often results in the survival of the fittest.

According to Bunting (1960), competition has different shades of meaning for the agronomist and plant physiologist. In his view, physiologists think of competition as for something, usually nutrients, water or light. Agronomists and weed scientists while agreeing, add that competition also exists between plants (Donald 1963). Many definitions were reviewed by Milne (1961).

1. Mather (1961): “Competition implies the presence of one individual as an effective part of other’s environment and a similarity of need or activities so their impact on each other is prospectively detrimental.”
2. Aspinall and Milthorpe (1959): “the interaction between plants and environment. The plants during growth modify the environment around them and the modified environment in turn influences the growth of the constituent plants.”

3. Bleasdale (1960): “Two plants are in competition with each other when the growth of either one or both of them is reduced or their form modified as compared with their growth or form in isolation.”

4. Milne (1961): “Competition is the endeavour of two (more) animals to gain the measure each wants from the supply of a thing when that supply is not sufficient for both (or all).”

5. Birch (1955): “Competition occurs when a number of animals (of the same or of different species) utilize common resources the supply of which is short, or if the resources are not in short supply competition occurs when the animals seeking that resource nevertheless harm one or the other in the process.”

6. Clements et al (1929): “Competition involves the reaction of a plant to the physical factors that encompass it and of the effect of these modified factors upon adjacent plants. For Clements et al., competition is a purely physical process.”

In the exact sense, two plants – no matter how close, do not compete with each other so long as the water content, the nutrient material, the light and heat are in excess of the need of both… Competition occurs when each of two or more organisms seeks the measure they want of any particular factor or things and when the immediate supply of the factor or things is below the combined demand of the organisms.

This definition makes competition different from the broader term interference, which includes competition and allelopathy.

**Crop-weed association**

Association of weeds with a particular crop is due to the following factors

1. Morphological similarities: Morphological similar weeds are difficult to control both by mechanical and chemical methods. For example plants of *Echinochloa crusgalli* which resembles with that of rice are often uprooted with rice nursery and well grown plants of this weed transplanted with rice are not controlled with any herbicide recommended in the crop.
2. Seed shedding behavior: Majority of weed plants complete their life cycle before the harvest/maturity of the crop(s) in which they are associated. The seeds which are shed in the field during one season, become the source of infestation during the coming years. About 90 to 95% seeds of problematic weeds such as *Phalaris minor*, *Avena ludoviciana*, *Echinochloa crusgalli*, *Ischaemum rugosum* and *Trianthema portulacastrum* are shed in the field. This seed shedding behavior is one of the main reasons of association of a particular weed with a specific crop when sown on same field regularly.

3. Escape removal through sieving: The seeds of few weeds are of the same size as of the crop grains/seeds and these cannot be separated out even by thorough sieving. These seeds are sown in the field along with crop seeds. The size of few weed seeds like *Convolvulus arvensis*, *Rumex spinosus*, *Avena ludoviciana* etc are of the same size as of wheat grain and these cannot be removed even with thorough sieving. The crop infested with these weeds should not be kept for seed production purposes.

4. Congenial environment: The association of weeds not only depends upon crop but it is also influenced by cropping sequence. For example field having infestation of wild oats in wheat crop can be eliminated if brought under rice – wheat rotation at least for 2-3 years. The wild oat seeds being porous absorb water and loose viability with continuous standing water. The continuous wet conditions eliminate wild oats but may give favour to *Phalaris* establishment due to congenial environmental conditions created with the cultivation of rice.

5. Germination in flushes: The weed seeds which have the tendency to germinate in many flushes are very difficult to control both with mechanical or chemical methods. The flush of weeds which appear after the adoption of weed control measures, produce seed and such type of weeds are rather permanently associated with the crop in spite of adoption of best weed management practices. For example, *Phalaris minor* seeds germinate in many flushes in wheat crop and due to this character it is very difficult to control this weed.

6. Continuous use of one group of herbicides: With the continuous use of single group of herbicides over years, leads to resistance phenomenon. For example, with continuous use of isoproturon in wheat, *Phalaris minor* has developed resistance to isoproturon in Punjab and Haryana. In spite of use of isoproturon even at higher doses, *Phalaris minor* was not killed. So this weed continued to associate wheat through physiological
modification. Rotational use of herbicides, helps a lot to avoid association of a particular weed with a particular crop.

7. Allelopathic effects: There are few weeds which are only associated with a particular host. In the absence of host plants, these weed seeds show no germination even when the seeds are in viable conditions. For example *Striga densiflora* is a parasitic weed of sugarcane and *Orobanche aegyptica* is of mustard. These host plants (crops) excrete some chemicals in soil and these chemicals acts as stimulants for the germination of seeds of these parasitic weed and are associated with their specific host crops while these do not germinate when other crops are sown in their habitat.

There are at least four reasons why two or more species can coexist in a place. They can have one or more of the following characteristics:

1. Different nutritional requirements, as illustrated by legumes and grasses coexisting in pasture and hay fields.
2. Different causes of mortality, observed in pastures where animals selectively graze.
3. Different sensitivity to toxins, including allelochemicals and herbicides.
4. A different time demand for growth factors. Many plants require the same growth factors to succeed, but they do not demand them at the same time. This may be the most common reason for coexistence.

Competition between crop plants and weeds is most severe when they have similar vegetative habit and common demand for available growth factors.

Weeds appear much more adapted to agro-ecosystems than our crop plants. Without interference by man, weeds would easily wipe out the crop plants. Rr I

**Principle of plant competition** is that the first plants to occupy any area of soil, small or large, tend to exclude others. Practices should be such that crop plants should occupy the soil before weeds. Thus seed bed preparation, depth of seeding, date of sowing and other agronomic manipulations play important part in making it possible for crop plants to avoid competition with weeds. Weeds that appear in the crop after it is well established usually have negligible competing ability. On the other hand weed plants germinated prior or with the crop, show very higher competition.
Weeds compete with the crop for following growth factors

1. **Nutrients:** It is an important aspect of crop weed competition. Weeds usually absorb mineral nutrients faster than crop plants. Usually weeds accumulate relatively larger amounts of nutrients than crop plants. Nutrient removal by weeds leads to huge loss of nutrients in each crop season, which is often twice that of crop plants. *Amaranthus* accumulate over 3% nitrogen in their dry matter and this fall under category of nitrophylls. *Digitaria* sp. accumulates more phosphorous content of over 3.36%. *Chenopodium* and *Portulaca* are potassium lovers, with over 4.0% $K_2O$ in their dry matter. *Setaria lutescens* accumulates as high as 585 ppm of zinc in its dry matter. This is about three times more than by cereal crop.

2. **Moisture:** Crop weed competition becomes critical with increasing soil moisture stress. In general for producing equal amount of dry matter weeds transpire more water than field crops. Therefore, the actual evapotranspiration from the weedy crop fields is much more than the evapotranspiration from a weed free crop field. Consumptive use of *Chenopodium album* is 550 mm as against 479 mm for wheat crop. Further it was noted that weeds remove moisture evenly from up to 90 cm soil depth. While the major uptake of moisture by wheat was limited to top 15 cm of soil depth. Weeds growing in fallow land are found to consume as much as 70-120 ha mm of soil moisture and this moisture is capable of producing 15-20 q of grain per ha in the following season.

3. **Solar energy:** Plant height and vertical leaf area distribution are the important elements of crop weed competition. When moisture and nutrients in soil are plentiful, weeds have an edge over crop plants and grow taller. Competition for light occurs during early crop growth season if a dense weed growth smothers the crop seedlings. Crop plants suffer badly due to shading effect of weeds. Cotton, potato several vegetables and sugarcane are subjected to heavy weed growth during seedling stage. Unlike competition for nutrients and moisture once weeds shade a crop plant, increased light intensity cannot benefit it.

4. **Space/CO\(_2\):** Crop-weed competition for space is the requirement for CO\(_2\) and the competition may occur under extremely crowded plant community condition. A more efficient utilization of CO\(_2\) by $C_4$ type weeds may contribute to their rapid growth over $C_3$ type of crops.
Competition in a community

In agricultural fields, orderly, continuous, natural ecological succession does not occur, but change, due to human manipulation, does. Agricultural fields that are not in a permanent crop such as an orchard or have a semipermanent perennial crop (e.g., alfalfa, where disturbance is still frequent for harvest and the land is rotated to another crop perhaps as often as every three years) are regularly and intentionally disturbed and lack a natural (undisturbed) plant community. Environmental change is a driving force in natural plant succession but annual crop agriculture strives to modify and control the environment through tillage, fertilization, pest (weed, insect, and disease) control, and irrigation.

Because of regular disturbance and other cultural practices, the spatial and temporal variability of agricultural environments is reduced compared to natural plant communities (Radosevich and Roush 1990). Dominance of the planted crop species characterizes agricultural plant communities that also have a few (rarely only one) weed species that occur in cropped fields. Their removal (control) creates open niches into which another weedy species will move, but perhaps not immediately. Therefore, weed management, especially successful weed management, is a never ending process (Zimdahl 1999). One could argue that the best weed management techniques may therefore be those that achieve less than 100 percent control and do not open niches that allow new introductions to succeed and necessitate further weed control.

The three primary processes that control the level of competition from a weed complex in a crop are:

1. Time of emergence of the crop and weed often determine the outcome of interference. When the crop emerges prior to the weeds, it often wins the competitive battle; the reverse is also true. Firbank and Watkinson (1985) reported that emergence time and local crowding accounted for as much as 50 percent of the variation in performance of individual corn cockle plants.

2. A second factor is growth ability and environment (Radosevich and Roush 1990). These are related because growth is surely affected by environment, but they can also be treated separately because growth of some species (e.g., green foxtail and field bindweed) will always be distinctly different, independent of the environment in which they are growing.
together. Clearly, plant growth rate has a strong influence on competitive ability. Species that grow tall rapidly and gain greater ground cover (shading) or spread rapidly laterally will have a competitive advantage over those that do each thing but more slowly. Equally clearly, environments that favour rapid growth will favour the species with greater competitive ability.

3. Finally, Radosevich and Roush (1990) mention the important but often neglected role(s) played by processes other than competition, such as herbivory, density-dependent mortality, predation, senescence, and allelopathy. Aarssen (1989) stated that plant species coexist by avoiding competitive exclusion. He agreed with Sakai (1961) when he proposed that, at the species level, continuing selection results from genetically based differences in competitive abilities in local neighborhoods. Aarssen reported on multigeneration experiments with timothy and common groundsel to show that competitive ability may change as a consequence of selection. He explored the evolutionary consequences of such selection in a community of several species.

Competition from weeds is the most important of all biological factors that reduce agricultural crop yield. This occurs primarily because weeds use resources that would otherwise be available to the crop. If crop plants occupy the soil and are vigorous, weeds are excluded or retarded in their growth. On the other hand if crop stand is thin or lacks vigour, weeds will flourish. Any environmental condition or any intervention that promotes the growth of crop plants tends to diminish the ill effects of weeds. Conversely, conditions or methods unfavourable to the growth of crop plants permit the invasion and development of a weed population. The magnitude of yield loss is affected by many agronomic and environmental factors, but most importantly by the weed density, and time of emergence relative to the crop. Practices that (1) reduce the density of weeds or (2) maximize occupation of space or uptake of resources by the crop or (3) establish an early season size advantage of the crop over the weeds, will minimize the competitive effects of weeds on crops.

There are two types of competition which exist in field crops/weeds viz. **intra-specific competition and inter-specific competition.** In a weed infested crop field in a nutshell, it is possible to identify different components of the overall competitive effect:

- intra-specific competition between plants of the cultivated species;
• inter-specific competition between plants of the cultivated species and weed species;
• inter-specific competition between plants of the different weed species;
• intra-specific competition between plants of the same weed species.

The effect of weed density

Research has shown that, in most cases, the weed density–crop yield relationship is not linear. A few weeds usually do not affect yield in a way that can be detected easily; also, the maximum effect, total crop loss, obviously cannot be exceeded and usually occurs at less than the maximum possible weed density. Based on two assumptions, Zimdahl (1980) proposed that weed competition could be represented by a schematic sigmoidal relationship. The assumptions were: (1) A few (say five in a hectare) weeds might affect crop growth and final yield but the effect could not be measured with any precision, and (2) there is a high density of weeds beyond which no further crop yield loss can be measured. Cousens et al. (1985) point out that the theory of the sigmoidal relationship (Zimdahl 1980) that no competition threshold exists was based on a faulty statistical assumption and not on sound biological principles or on economic rationality.

A curvilinear relationship was reported by Roberts and Bond (1975), who described the effect of naturally occurring annual weeds at densities of 65 to 315 plants m\(^{-2}\) on yield of lettuce. Very low densities were not included. Roberts and Bond’s (1975) study clearly shows that marketable lettuce yield sinks to zero at less than maximum weed density. It is now clear that the assumptions of the sigmoidal and curvilinear relationships are incorrect and that neither is capable of accurately describing the relationship between weed density and crop yield (Cousens et al. 1984; Cousens et al. 1985). The relationship is hyperbolic, which means, among other things, that it is best described by a hyperbolic curve that is usually referred to as a rectangular hyperbola. It also means that, even though the effect may not be measurable with precision, there is, in theory and actually, an effect of a few weeds (a low density) on crop yield and growth. Finally, the hyperbolic relationship means that the weeds’ effect cannot exceed total (100 percent) crop loss.

Threshold

Farmers cannot tolerate excessive yield losses from weeds. To avoid such losses, the determination of two distinct weed threshold concepts can be useful. First, a competitive
threshold can be defined as the weed density and duration of interference above which crop yield is reduced significantly, generally from 10-20%. Second, an economic threshold occurs where the monetary yield loss exceeds cost of control. The producer usually is most concerned about the economic threshold because of its immediate role in production decisions. However, along with the impact of weeds on the current crop, the producer should consider the complete crop rotation cycle, the future weed infestation, and even the future generations who will farm the land. The three additive research techniques to determine competitive weed thresholds are removal or plant-back, paired plot and area of influence. Each method has advantages and disadvantages and must be conducted in a specific manner in the field. The area of influence technique is the most accurate for determining the influence of a single weed. Data must be reported in a usable form such as percent yield reduction. Generally the economic threshold is one-half to one-third the competitive threshold.

The economic threshold (=economic injury levels), the weed density at which the cost of treatment equals the economic benefit obtained from that treatment, may be calculated after modifying the formula presented by Uygur & Mennan (1995) as well as those given by Stone and Pedigo (1972) as below:

Uygur & Mennan:

\[ Y = \left\{ \frac{(100/He*Hc)+Ac}{(Gp*Yg)} \right\} \times 100 \]

Where, \( Y \) is percent yield losses at a different weed density; \( He \), herbicide efficiency; \( Hc \), herbicide cost; \( Ac \), application cost of herbicide; \( Gp \), grain price and \( Ywf \), yield of weed free.

Stone and Pedigo:

Economic threshold = Gain threshold/Regression coefficient

Where, gain threshold = Cost of weed control \((Hc+Ac)/Price\) of produce \((Gp)\), and regression coefficient \((b)\) is the outcome of simple linear relationship between yield \((Y)\) and weed density/biomass \((x)\), \( Y = a + bx \).

Several papers report specific effects of specific weeds on corn yield reduction. More than 13 species have been studied and the papers are summarized below:
**Effect of several weeds on corn yield**

<table>
<thead>
<tr>
<th>Weed species</th>
<th>Density</th>
<th>Yield reduction</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barnyard grass</td>
<td>100 m⁻²</td>
<td>18%</td>
<td>Kropff et al. 1984</td>
</tr>
<tr>
<td></td>
<td>200 m⁻² concurrent</td>
<td>26-35%</td>
<td>Bosnic et al 1997</td>
</tr>
<tr>
<td></td>
<td>emergence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Common milkweed</td>
<td>11000-45200 m⁻²</td>
<td>10%</td>
<td>Cramer and burnside</td>
</tr>
<tr>
<td></td>
<td>Emergence when corn had 4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>leaves</td>
<td>6%</td>
<td></td>
</tr>
<tr>
<td>Giant ragweed</td>
<td>1.7, 6.9 or 13.8 m⁻²</td>
<td>10%</td>
<td>Harrison et al 2001</td>
</tr>
<tr>
<td>Giant foxtail</td>
<td>0, 29, 56, 89 or 129 m⁻²</td>
<td>20-56%</td>
<td>Sibuga and bandeen 1980</td>
</tr>
<tr>
<td>Hemp dogbane</td>
<td>Natural</td>
<td>0-10%</td>
<td>Schultz 1979</td>
</tr>
<tr>
<td>Itchgrass</td>
<td>2, 4, upto 14 wk</td>
<td>125 kg ha⁻¹ for each week</td>
<td>Strahan et al 2000</td>
</tr>
<tr>
<td></td>
<td>of presence</td>
<td>13%</td>
<td></td>
</tr>
<tr>
<td>Jimsonweed</td>
<td>8.3 or 16.7</td>
<td>14-63% plants m⁻²</td>
<td>Cavero et al 1999</td>
</tr>
<tr>
<td>Quackgrass</td>
<td>65-390 shoots m⁻²</td>
<td>12-16%</td>
<td>Young et al 1984</td>
</tr>
<tr>
<td></td>
<td>745 shoots m⁻²</td>
<td>37%</td>
<td></td>
</tr>
<tr>
<td>Palmer amaranth</td>
<td>0.5 to 8 m⁻²</td>
<td>11-74%</td>
<td>Massinga et al 2002</td>
</tr>
<tr>
<td>Redroot pigweed</td>
<td>0.5 m⁻³ with concurrent</td>
<td>5%</td>
<td>Knezevic et al 1994</td>
</tr>
<tr>
<td></td>
<td>planting or 4 m⁻³ with</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>planting at corn’s 3-5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>leaf stage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wild proso millet</td>
<td>10 m⁻²</td>
<td>13-22%</td>
<td>Wilson and Westra 1991</td>
</tr>
<tr>
<td>Yellow nutsedge</td>
<td>100 shoots m⁻²</td>
<td>8%</td>
<td>Stoller et al 1979</td>
</tr>
<tr>
<td></td>
<td>300 tubers m⁻²</td>
<td>17%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>700 tubers m⁻²</td>
<td>41%</td>
<td></td>
</tr>
</tbody>
</table>

**Effect of competition duration**

It seems obvious that if one weed is, or several weeds are, present for 1 day in the life of a crop, it will have no measurable effect on final yield. But what if the weed(s) is present for 2, 10, or 100 days? The question of duration of competition has been addressed in two ways. The first kind of study asks, what is the effect when weeds emerge with the crop and are allowed to grow for defined periods of time? After each of these times, the crop is then kept weed free for the rest of the growing season. The second, frequently complementary, study asks, what is the effect when the crop is kept weed free after crop emergence for certain periods of time and then weeds are allowed to grow for the rest of the growing season? These studies, when combined, can be used to define what is usually called the critical weed-free period.
Minotti and Sweet (1981) said, “weed scientists have conducted a substantial number of so-called ‘critical period’ studies.” The period at which maximum crop weed competition occurs is called critical period. It is the shortest time span in the ontogeny of crop when weeding results in highest economic returns. Minotti and Sweet (1981) note that relatively noncompetitive crops such as onion or garlic require a weed-free period of three months or more. More competitive crops such as corn or soybeans require only three to four weed-free weeks.

**Critical period of crop-weed competition and losses in yield in some crops**

<table>
<thead>
<tr>
<th>Crop</th>
<th>Critical period (days)</th>
<th>% reduction in grain yield</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cereals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transplanted Rice</td>
<td>30-45</td>
<td>15-45</td>
</tr>
<tr>
<td>Direct seeded rice</td>
<td>15-45</td>
<td>15-90</td>
</tr>
<tr>
<td>Wheat</td>
<td>30-45</td>
<td>20-40</td>
</tr>
<tr>
<td>Maize</td>
<td>15-45</td>
<td>40-60</td>
</tr>
<tr>
<td>Bajra</td>
<td>30-45</td>
<td>15-60</td>
</tr>
<tr>
<td>Jowar</td>
<td>15-45</td>
<td>15-40</td>
</tr>
<tr>
<td>Ragi</td>
<td></td>
<td>35</td>
</tr>
<tr>
<td><strong>Legumes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Green gram and Black gram</td>
<td>15-30</td>
<td>25-50</td>
</tr>
<tr>
<td>Red gram/pigeonpea</td>
<td>15-60</td>
<td>20-40</td>
</tr>
<tr>
<td>Horse gram</td>
<td>20-45</td>
<td>-</td>
</tr>
<tr>
<td>Cowpea</td>
<td>15-30</td>
<td>15-30</td>
</tr>
<tr>
<td>Chickpea</td>
<td>30-60</td>
<td>15-25</td>
</tr>
<tr>
<td>Peas</td>
<td>30-45</td>
<td>20-30</td>
</tr>
<tr>
<td>Lentil</td>
<td>30-60</td>
<td>20-30</td>
</tr>
<tr>
<td>Soybean</td>
<td>20-45</td>
<td>40-60</td>
</tr>
<tr>
<td><strong>Oilseeds</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sesamum</td>
<td>15-45</td>
<td>15-40</td>
</tr>
<tr>
<td>Groundnut</td>
<td>40-60</td>
<td>40-50</td>
</tr>
<tr>
<td>Sunflower</td>
<td>30-45</td>
<td>30-60</td>
</tr>
<tr>
<td>Castor</td>
<td>30-50</td>
<td>30-35</td>
</tr>
<tr>
<td>Safflower</td>
<td>15-45</td>
<td>15-40</td>
</tr>
<tr>
<td>Rapeseed mustard</td>
<td>15-40</td>
<td>15-30</td>
</tr>
<tr>
<td>Linseed</td>
<td>20-45</td>
<td>30-40</td>
</tr>
<tr>
<td><strong>Commercial crops</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sugarcane</td>
<td>30-120</td>
<td>20-30</td>
</tr>
<tr>
<td>Cotton</td>
<td>15-60</td>
<td>40-50</td>
</tr>
<tr>
<td>Potato</td>
<td>20-40</td>
<td>30-60</td>
</tr>
<tr>
<td>Tobacco</td>
<td>40-65</td>
<td>-</td>
</tr>
<tr>
<td>Jute</td>
<td>30-45</td>
<td>50-80</td>
</tr>
<tr>
<td><strong>Vegetables</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Factors affecting weed-crop interference

1. Period of weed growth

Weeds interfere with crops at anytime they are present in the crop. Thus weeds that germinate along with crops are more competitive. Sugarcane takes about one month to complete its germination phase while weeds require very less time to complete their germination. By that time crop plants are usually smothered by the weeds completely. First 1/4 - 1/3 of the growing period of many crops is critical period. However in a situation, where weeds germinate late, as in dry land wheat and sorghum, the late stage weeding is more useful than their early weeding. In general for most of the annual crops first 20-30 days weed free period is very important.

2. Weeds / crop density

Increasing weed density decreases the crop yield. The relationship between yield and weed competition is sigmoidal. In rice, density of Joint vetch (*Aschynomene virginica*) and barnyard grass, if it is > 10 plants/m² rice yields were reduced by 20 and 11 q/ha, respectively. Crop density also effect the weed biomass production. Increase in plant population decreases weed growth and reduce competition until they are self competitive for soil moisture and other nutrients. In wheat, reduced row spacing from 20 to 15 cm reduced the dry matter yield of *Lolium* and *Phalaris* sp. by 11.8% and 18.3%, respectively.

3. Plant species effects
   a) Weed species

Weeds differ in their ability to compete with crops at similar density levels. This is because of differences in their growth habits and to some extent due to allelopathic effects. At early stage of growth, cocklebur (*Xanthium strumarium*) and wild mustard (*Brassica* spp.) are better competitors for crops than many grasses because of their fast growing leaves that shade the ground heavily. In dry areas perennial weeds like Canada thistle (*Cirsium arvense*) and field bind weed (*Convolvulus arvensis*) were more competitive than annual weeds because of their deep

<table>
<thead>
<tr>
<th>Crop</th>
<th>Lower PP</th>
<th>Upper PP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Okra</td>
<td>15-30</td>
<td>40-50</td>
</tr>
<tr>
<td>Tomato</td>
<td>30-45</td>
<td>40-70</td>
</tr>
<tr>
<td>Onion</td>
<td>30-75</td>
<td>60-70</td>
</tr>
<tr>
<td>Cauliflower</td>
<td>30-45</td>
<td>50-60</td>
</tr>
<tr>
<td>Cabbage</td>
<td>30-45</td>
<td>50-60</td>
</tr>
</tbody>
</table>
roots and early heavy shoot growth. Composite stand of weed sp. is always more competitive than a solid stand of single weed sp.

b) Crop species and varietal effects
They differ in their competing ability with weeds. Among winter grains the decreasing order of weed competing ability is barley > rye > wheat > oat. In Barley it may be due to more extensive root growth during the initial three weeks. Fast canopy forming and tall crops are more competitive than slow growing short stature crops (sorghum, maize, soybean, cowpea). Because of their slow initial growth. Late sown dwarf wheat is affected by the late germinating weeds like Canada thistle and wild safflower (Carthamus oxyacantha) and Phalaris minor even though they escape an initial flush of weeds.

Varieties
Smothering crops grow very fast during early stages. Cowpea and horse gram are tolerant to weed competition. When we compare the crop-weed competition between two varieties of groundnut, in spreading groundnut (TMV-3) the yield loss is 15% in weedy plots compared to bunch groundnut (TMV-2) where yield loss is 30%. This is due to smothering effect of spreading groundnut. Likewise long duration rice is more competitive than short duration rice varieties. Wild oat growth increase with increase dwarfness of wheat plant.

4) Soil and climatic influence
a) Soil fertility
Under limited nutrient conditions, competition exists between the crop and the weed. Soil type, soil fertility, soil moisture and soil reaction influences the crop weed competition. Elevated soil fertility usually stimulates weeds more than the crop, reducing thus crop yields. Method and time of application of fertilizers to crop determining whether added fertilizer will suppress or invigorate weed growth in fields. Application of fertilizers during early crop growth season when weed growth is negligible was more beneficial. Band application of fertilizers to the crop will be inaccessible to inter row weeds.

b) Soil moisture status
Weeds differ in their response to available moisture in soil. Russian thistle Salsola kali showed similar growth in both dry soils and wet soils; whereas large crab grass Digitaria
sanguinalis produce more growth on wet soil. When fields are irrigated immediately after planting then weeds attain more competitive advantage over crops. If the weeds were already present at the time of irrigation, they would grow so luxuriantly as to completely over cover the crops. If the crop in irrigated after it has grown 15 cm or more in a weed free environment irrigation could hasten closing in of crop rows, thus suppressing weeds. In water logged soils weeds are more competitive than crop plants. In submerged conditions in rice, weeds are put to disadvantage to start with. But if there is a break in submergence, the weeds may germinate and grow more vigorously than the crop, even if fields were submerged later.

c) Soil reaction

Abnormal soil reactions (very high or very low pH) often aggravate weed competition. Weeds offer intense competition to crops on abnormal pH soils than on normal pH soils. In acid soils Rumex acetosella and Pteridium spp, saline alkaline soils Taraxacum stricta, Agropyron repens are the dominant weeds.

d) Climatic influences

Adverse weather conditions such as drought, floods and extreme of temperature intensify weed-crop interference since most of our crop varieties are highly susceptible to such climatic influences where as the weeds are tolerant to their stresses.

5) Cropping practices

a) Time of planting crops

If the time of planting of a crop is such that its germination coincides with the emergence of first flush of weeds, it leads to intense weed-crop interference. Usually longer the interval between emergence of crop and weeds, lesser will be the weed-crop interference.

b) Method of planting of crops

Weed seeds germinate most readily from top 1.25 cm of soil, though it is considered up to 2.5 cm depth. Avena, barnyard grass, Xanthium and Vicia spp may germinate even from 15 cm depth. Therefore, planting method that dries up the top 3-5 cm of soil rapidly to deny weed seeds opportunity to absorb moisture for their germination and usually postpone weed emergence until first irrigation. By that time crop establishes well and competes with weeds. Weed seeds are
classified as i) Deep germinating (15 cm), ii) Shallow germinating (up to 5 cm), and iii) Surface germinating (0.25 cm)

c) Crop density and rectangularity

It determines the quantity and quality of crop environment available to the growth of weeds. Wide row spacing with simultaneous high intra row plant population may induce dense weed growth. But square method of planting is ideal to reduce intra row competition. (from the point of weed–crop competition).

Weed shifts

Bruckner et al. (1997) surveyed weeds in maize in the Szigetkoz area between 1 July and 31 September 1996, and the findings compared with those of surveys conducted in 1990. An increase in the average cover and frequency of occurrence of *Panicum miliaceum*, *Mercurialis annua* and *Ambrosia artemisiifolia* was observed. Many species not recorded in 1990 were registered (*Eragrostis minor*, *Amaranthus graecizans*, *Digitaria sanguinalis* and *Geranium pusillum*). Such changes in weed flora composition over time are referred to as weed shifts. A weed shift may be defined as ‘the change in the composition or relative frequencies of weeds in a weed population (all individuals of a single species in a defined area) or community (all plant populations in a defined area) in response to natural or human-made environmental changes in an agricultural system’. Shifts in weeds are not new. Weed shifts have happened as long as humans have cultivated crops. Weedy and invasive species can easily adapt to changes in production practices in order to take advantage of the available niches.

The behavior of a weed population depends on the nature of the environment experienced by individual plants. Increase in the size of a population is achieved through reproduction of the individuals that survive to maturity and by gains from immigration. Survival may occur by persistence in a dormant state (as seeds in the soil) or by escape from control as seedlings or plants (through chance or due to genotype, as in herbicide resistance). It is therefore, the reproductive contribution of these survivors that is important in the growth of the population.

Plant species may be pre-adapted to be weeds in the sense that species possesses a suite of a life history characteristics which enables rapid population growth in the particular habitat conditions created and maintained by human activity. Pre-adapted weeds have been defined as those species that
• are resident in a natural plant community within dispersal distance of the crop (or other habitat)
• come to predominate within the crop as a consequence of a change in crop and weed management practices.

The successful invasion of a crop by a species from natural habitat, wasteland or hedgerow therefore depends on the match of life history characteristics of the weed to the habitat template provided by the cropping system.

Examples of weed shifts that have occurred in recent history include the following:

**Community shift in response to herbicide use.** With the use of clodinafop in wheat weed flora was mainly composed of *Poa annua*. Similarly in maize continuous use of pre-emergence atrazine give subsequent flushes of *Commelina banghalensis, Bracharia ramosa* and *Ageratum conyzoides*. In the Corn Belt and winter wheat areas of the western United States, changes in weed communities were noted within 10 years of the introduction of 2,4-D for the control of broadleaf weeds. In corn, summer annual grass species increased as broadleaf species were controlled. In wheat, winter annual grass species replaced broadleaf species as the predominant troublesome species.

**Community shift in response to tillage change.** Changes from conventional to reduced tillage systems often cause weed community shifts that include increases in summer annual grasses and small-seeded summer annual broadleaves, winter annual, biennial, and perennial species, and decreases in large-seeded summer annual species.

**Community shift as a result of new localized or long-distance introductions.** Common lambsquarters, a weed believed to be native to Europe and Asia, is now found throughout much of the United States. In much of Pennsylvania, common lambsquarters has become predominant in the weed community. The shift occurred because the species grows aggressively, is difficult to control, and is a prolific seed producer. Long-distance dispersal has also resulted in the introduction of many noxious weeds to the United States, some of which have caused weed community shifts (including field bindweed in the western plains, leafy spurge in rangeland, and multiflora rose in pasture). Under Indian conditions, *Phalaris minor* in wheat and invasion of
Advances in Weed Management

Grasslands and pastures and other non-cropped areas with *Lantana* and *Parthenium* are typical examples.

**Population biotype shifts in response to herbicide use (herbicide-resistant populations).** In the mid-west, in many populations of common waterhemp (pigweed species), biotypes differed in susceptibility to ALS-inhibiting herbicides. With recurrent spraying of ALS-inhibiting herbicides, populations shifted from susceptible to highly resistant biotypes. (Note: A concurrent shift in many weed communities also occurred. Other species were controlled with these highly effective herbicides, and the waterhemp numbers increased as a proportion of the entire weed community). Other ALS-resistant pigweed species have also developed in several areas of the United States, including the northeast. Most recently, glyphosate resistant weeds such as horseweed (marestail) and pigweed species are a problem in different regions of the U.S. as a direct result of glyphosate use in herbicide resistant crops.

**Why Do Weed Spectrums Change?**

Weeds are well equipped to flourish in disturbed agricultural systems. Weeds are genetically diverse and can readily take advantage of the variety of conditions created by any given crop production system. Many common weed species also have the ability to rapidly establish themselves in a field in just a couple of year’s time. This is primarily due to some weeds ability to produce a large quantity of viable seeds (if it is an annual) or vegetative tissues such as rhizomes (if it is a perennial) in a single growing season. Most weed species also have the attribute of seed or bud (if it is a perennial) dormancy. This allows a diversity of weed species to exist for long periods of time in the soil. Thus, when changes in the cropping system occur that are favorable for their germination and development, a particular weed species is able to respond fairly quickly and rapidly (often within three to five growing seasons) and establish itself in the cropping system. Therefore, one key to reducing the predominance of any given weed species is to increase the diversity of crops within the cropping system, or at least the diversity of weed management practices within the cropping system.

There are many factors that interact and influence the weed population dynamics in cropping system. Changes in tillage practices, cultural practices, row spacing and planting date, and weed management practices have all had an impact on the weed spectrum. Further, the ever
changing weather patterns only complicate matters. Weed shifts have occurred in response to changes in tillage (Ball 1992; Tuesca et al. 2001), irrigation, fertility (Davis et al. 2005), crop rotation (Davis et al. 2005; Takim et al. 2014; Ball 1992) and herbicide use practices (Davis et al 2005; Sakia and Pandey 1999).

Weed shifts occur when weed management practices do not control an entire weed community or population. The management practice could be herbicide use or any other practice such as tillage, manure application, or harvest schedule that brings about a change in weed species composition.

Some species or biotypes are killed by (or susceptible to) the weed management practice, others are not affected by the management practice (tolerant or resistant), and still others do not encounter the management practice (dormant at application). Those species that are not controlled can grow, reproduce, and increase in the community; resulting in a weed shift. Any cultural, physiological, biological, or chemical practice that modifies the growing environment without controlling all species equally can result in a weed shift.

WEED RESISTANCE

In contrast to weed shift, weed resistance is a change in the population of weeds that were previously susceptible to an herbicide, turning them into a population of the same species that is no longer controlled by that herbicide. While weed shifts occur with any agronomic practice (crop rotation, tillage, frequent harvest or use of particular herbicide), the evolution of weed resistance is only the result of continued herbicide application. The use of a single class herbicide application continuously over time creates selection pressure so that resistant individuals of a species survive and reproduce, while susceptible ones are killed.

A weed shift is far more common than weed resistance, and ordinarily take less time to develop. If an herbicide does not control all the weeds, the tendency is to quickly jump to the conclusion that resistance has occurred.

A common misconception is that weed resistance is intrinsically linked to genetically engineered crops. However, this is not correct. The occurrence of weed shifts and weed resistance is not unique to genetically engineered crops. Weed shifts and resistance are caused by
the practices (for example repeated use of single herbicide) that may accompany a genetically engineered crop and not the genetically engineered crop itself.

Transgenic herbicide resistance crops have greater potential to foster weed shifts and resistant weeds since a grower is more likely to use single herbicide in transgenic herbicide resistance crops. The increase in acreage of these crops could increase the potential for weed shifts and weed resistance in the cropping systems utilising transgenic herbicide resistance crops. Weed species shifts and herbicide resistant weeds are the direct result of a lack of diversification in weed management systems.

**Production Practices That Influence Shifts**

**Effectiveness of weed management program.** The effectiveness of the overall weed management program will be the single most important factor affecting weed shifts. If the program is not managing all weed species, species that are not effectively controlled will increase. Farmer diligence and ability to make adjustments in a weed management program will directly influence the occurrence of weed shifts.

**Speed of weed shift occurrence.** The speed at which weed shifts occur will depend on various factors. The ability to prevent the introduction of new weeds to an area, the buffering capacity of the soil seed bank, weed characteristics described previously, and the farmer’s ability to quickly adjust a weed management strategy when a shift is first observed will all influence the speed at which a shift occurs—or if a shift occurs at all. With poor management, a field could shift predominant weed species very quickly. Weed species shifts are a long-range risk, generally taking 5 to 7 years for significant weed species shifts to occur. The temptation of the short-term gains of using the Roundup Ready technology across all corn and soybean acres is strong and short-term gains are often adopted because "a Rupee today is worth more than a Rupee tomorrow".

**Interface areas.** A cropped field will likely include places where no herbicide is applied (skips) or where reduced rates are applied (field edges, places with a lack of overlap). These areas may or may not influence weed shifts. In some cases, these areas may allow genotypes that were not
subjected to the selection pressure to survive and reproduce. However, these areas may be so small that they have a negligible effect on overall field effects.

**Preventing weed species shifts and herbicide resistant weeds:**
Weed species shifts, whether it is to a resistant weed, or a new biotype, complicate weed management decisions. The best solution is to develop integrated weed management systems that prevent the problem from occurring.

- Use mixtures or sequential treatments of herbicides that each control the weeds in question, but have a different site of action.
- Scout fields after application to detect weed escapes or shifts. If a potentially resistant weed or weed population has been detected, use available control methods to avoid seed deposition in the field.
- Rotate herbicides (sites or modes of action) so you make no more than two consecutive applications with the same site of action against the same weed unless you use other effective control practices.
- Rotate crops, particularly those with different life cycles (for example, winter annuals such as winter wheat, perennials such as alfalfa, summer annuals such as corn or soybeans).
- Clean equipment before leaving fields infested with or suspected to have resistant weeds.
Mode of Action of Herbicides

Herbicide effectiveness depends on 1) adequate plant contact; 2) absorption by plants; 3) translocation (i.e. movement) within the plants to the site of action, without deactivation; and 4) toxic levels at the site of action. The application method, whether pre-plant incorporated, pre-emergence, or post-emergence, largely determines when the herbicide will contact plants and the portion of the plant contacted. Site of action or mechanism of action refers to the primary biochemical or biophysical reactions which bring about the ultimate herbicidal effects. Mode of action refers to the sequence of events from absorption into plants to plant death. The mode of herbicide action may determine the application method needed for best results. For example, herbicides which affect photosynthesis but have little soil residual, such as desmedipham (Betanex) or paraquat (Gramoxone Extra), need to be applied post-emergence to leaf tissue. Seedling growth inhibitors, such as trifluralin (Treflan) and EPTC (Eptam), need to be applied to the soil to effectively control newly germinating seedlings.

An understanding of how herbicides kill weeds (herbicide mode of action) is useful in selecting and applying the proper herbicide. Herbicide mode of action information also is useful in diagnoses of injury from herbicides. Knowing the way herbicides act inside plants helps the person to apply them properly and to explain the results obtained. To kill weeds, herbicides must reach the appropriate site or sites of action within the weeds where a lethal reaction will occur. A herbicide must enter into the weed plant, move by diffusion or other means within the plant, escape being detoxified by the weed and finally attack some vital plant process important to the weed’s survival.

Herbicides enter plants through roots, stems and leaves. They exert their toxicity in many different ways and can be broadly divided into leaf-acting and soil-acting herbicides. Leaf-acting herbicides may be further sub-divided into contact and translocated herbicides, according to whether they act by contact only or are absorbed and moved (translocated) within the plant. Contact herbicides e.g. paraquat kill only those parts of the plant that they touch. They are used mainly as directed sprays (i.e. onto the weeds) for controlling seedling or annual weeds and do not eradicate deep-rooted perennial weeds unless used repeatedly. Leaf-acting, translocated herbicides e.g. glyphosate, move through the sap conducting tissue (phloem) and are carried with
the sap stream from the leaves to regions of active growth. Soil-acting herbicides are absorbed by roots and move through the water conducting tissue (xylem) to the actively growing parts of the plant.

Because leaf-acting herbicides move from the site of application to other parts of the plant they are often effective against weeds with deep roots. Weed foliage must be present if these herbicides are to be effective and it is futile to apply glyphosate or paraquat to bare soil. Similarly, it is bad practice to apply leaf acting herbicides at very high volumes (weak concentration) which result in excessive run-off of the spray. Many leaf acting herbicides have no action through the soil and any chemical reaching the soil is wasted. Thorough wetting of all foliage with a translocated herbicide is unnecessary because the herbicide will move within the plant. With contact herbicides good spray coverage is essential for effective control.

**Plant physiological processes**

Some of the most Important physiological processes that are affected by herbicides are listed below:-

- Photosynthesis - the synthesis of carbohydrate by which the chlorophyll in green plants provides the basic food material for other processes.
- Nitrogen metabolism - the incorporation of inorganic N into organic compounds, making possible the synthesis of proteins and protoplasm itself.
- Respiration - the oxidation of food in living cells, releasing the energy used in assimilation, mineral absorption and other energy using processes.
- Translocation - movement of water, minerals, food and hormones from place to place.
- Growth - permanent increase in size from the interaction of the above processes.
- Growth regulation - the complex interaction of hormones and nutritional balance that controls the growth of plants.

**Penetration and movement of herbicides within plants**

A series of barriers have to be overcome between the time the herbicide is applied and the weeds are killed.
Shoot penetration

Plants absorb herbicides both through their upper and lower surfaces. The lower surface (epidermis) is usually penetrated most easily and particularly through open stomata (leaf pores), natural fissures or other breaks in the cuticle (the waxy protective layer on the outside of the leaf). Sprays with the addition of suitable wetters readily enter plant leaves through open stomata.

If a herbicide enters a leaf through the cuticle, it enters by diffusion. When weeds are under pressure, e.g. through age, drought or poor growing conditions, the cuticle tends to be thicker and herbicides may enter the leaf less readily. This helps to explain why glyphosate works better when weeds are growing well and why some surfactants may be beneficial as they improve penetration through the waxy covering. Not all surfactants are useful with particular herbicides as some may damage cell tissue and this could impede absorption of the herbicide and translocation from the leaf.

Root penetration

Herbicides enter plants via the roots just the same way as nutrients. This may either be by passive or active mechanisms (requiring energy). If passive, the herbicide enters with absorbed water and may continue to move with water through the plant mainly in the water conducting tissue (the xylem). Active uptake involves entrance into the protoplasm and movement via the symplast system.

Light rainfall usually improves the effect of root absorbed herbicides as the chemical is incorporated more effectively through the root absorbing zone of the weeds. Moist soil is essential for good results with simazine and, more particularly, isoxaben. On the other hand, heavy and prolonged rainfall may leach herbicides, such as propyzamide, from the weed germinating zone, especially on light soil and reduce its herbicidal effect.

Movement from site of uptake to site of action

Herbicides applied to weed foliage can be divided into those that mainly affect sprayed leaves e.g. paraquat and those that are translocated (moved) to other parts of the plant e.g.
glyphosate. Once inside the leaf, further movement of a translocated herbicide must occur. This usually takes place in the water conducting stream (the xylem) or in the sap conducting stream (the phloem). A few herbicides, such as fluazifop - butyl move in both streams - in the water conducting tissue when absorbed by the roots and in the sap conducting tissue when absorbed by the leaves. Contact herbicides do not usually translocate from treated leaves but simple diffusion can account for limited herbicide movement in some cases, e.g. paraquat moves a short distance in the xylem stream.

A number of factors can affect a herbicide during translocation from the point of absorption to the site of phytotoxic action in sensitive tissue. For example, simazine is converted to an inactive form in the leaves of some woody plants which accounts for the tolerance of these plants to simazine. The simazine molecule is dechlorinated (dehalogenation reaction) and the chlorine is replaced with a hydroxyl group. The end product of the inactivation reaction is hydroxysimazine, which is 1000 times less toxic than simazine. In the majority of cases selectivity is due to differences in metabolism between the crop and weed i.e. the crop unlike the weed is able to metabolize the herbicide to an inactive form. Atrazine is rapidly degraded by corn but not by weeds. Hence the corn is tolerant and the weeds are susceptible. This makes atrazine a selective herbicide.

Specific biochemical or chemical reactions are

- decarboxylation - removal of -COOH group from the herbicide; part of the degradation sequence for many phenoxy, benzoic acid, and substituted urea herbicides
- hydroxylation - the addition of a -OH group to the molecule; often accompanied by removal or shifting of another atom such as chlorine; common among phenoxy, benzoic acid, and triazine herbicides
- hydrolysis - splitting of a molecule through the addition of water; involved in the degradation of carbamate, thiocarbamate, and substituted urea herbicides.
- dealkylation - removal of alkyl side chains from the herbicide; examples include the triazine, substituted urea, carbamate, thiocarbamate, and dinitroaniline herbicides
- deamination - removal of an amine group (NH2) from the herbicide
- ring hydroxylation - the addition of a -OH group to the ring structure of the molecule
- dealkyloxylolation - removal of an alkyl group with an attached “O”
beta-oxidation - pathway in plants whereby long chain carbon segments are degraded by removing 2 carbons for each cycle of the pathway

Although a large number of herbicides are available, they can be divided into groups with similar chemical and phytotoxic properties. Herbicides with a common chemistry have been conveniently organized into "families". In addition, two or more herbicide families may have the same mode of phytotoxic action and thus express the same injury symptoms.

The following sections describe the characteristics of widely used herbicide families grouped by mode of action. These seven major modes of action are: growth regulation, amino acid synthesis inhibition, lipid synthesis inhibition, seedling growth inhibition, photosynthesis inhibition, cell membrane disruption, and pigment inhibition.

**Growth Regulators**

Benzoic acids, phenoxyacetic acids, pyridine carboxylic acids and quinoline carboxylic acids are herbicides that act similar to that of endogenous auxin (IAA) although the true mechanism is not well understood. Growth regulator herbicides can act at multiple sites in a plant to disrupt hormone balance and protein synthesis and thereby cause a variety of plant growth abnormalities. The specific cellular or molecular binding site relevant to the action of IAA and the auxin-mimicking herbicides has not been identified. Nevertheless, the primary action of these compounds appears to affect cell wall plasticity and nucleic acid metabolism. These compounds are thought to acidify the cell wall by stimulating the activity of a membrane-bound ATPase proton pump. The reduction in apoplastic pH induces cell elongation by increasing the activity of enzymes responsible for cell wall loosening. Low concentrations of auxin-mimicking herbicides also stimulate RNA polymerase, resulting in subsequent increases in RNA, DNA, and protein biosynthesis. Abnormal increases in these processes presumably lead to uncontrolled cell division and growth, which results in vascular tissue destruction. In contrast, high concentrations of these herbicides inhibit cell division and growth, usually in meristematic regions that accumulate photosynthate assimilates and herbicide from the phloem. Auxin-mimicking herbicides stimulate ethylene evolution which may in some cases produce the characteristic epinastic symptoms associated with exposure to these herbicides. Growth regulator
herbicides control broadleaf weeds and can injure broad-leaf crops. Herbicides in this group can move in both the xylem and the phloem to areas of new plant growth. As a result, many herbicides in this group are effective on perennial and annual broadleaf weeds. Herbicide uptake is primarily through the foliage but root uptake may also occur.

1. Phenoxy Acetic Acids

a. Use: 2,4-D ((2,4-dichlorophenoxy) acetic acid) for small grains, corn, grass pastures, and non—cropland; MCPA (2-methyl-4-chlorophenoxy acetic acid) for small grains and grass establishment; 2,4-DB (4-(2,4-dichlorophenoxy) butyric acid) for alfalfa and soybean.

b. Injury Symptoms: All phenoxy acetic acids produce identical symptoms in sugarbeet. Sugarbeet leaves will be flat on the ground within a few hours after exposure and leaves may remain more prostrate than normal for the rest of the growing season if injury is severe. Leaf petioles exhibit twisting, also called epinasty. Sugarbeet exposed to phenoxy acetic acids in the cotyledon to early four-leaf stage may develop fused petioles and a symptom called "celery stalking" or "trumpeting". Phenoxy acetic acid herbicides on larger sugarbeet will not produce "celery stalking." New leaf growth generally will resume at about six to 10 days after exposure to phenoxy acetic acids. New leaves often will be malformed with crinkled leaf margins, parallel veins, or leaf strapping. Rapid initiation of new leaf growth suggests less sugarbeet injury and less sugarbeet yield loss than a delayed initiation of new leaf growth. Sugarbeet injured with phenoxy acetic acids should be processed immediately after harvest because injured sugarbeet roots do not store as well as non-injured roots.

c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action. Sugarbeet exposed to phenoxy acetic acids in the cotyledon to early four-leaf stage may develop fused petioles and a symptom called "celery stalking" or "trumpeting". Phenoxy acetic acid herbicides on larger sugarbeet will not produce "celery stalking." New leaf growth generally will resume at about six to 10 days after exposure to phenoxy acetic acids. New leaves often will be malformed with crinkled leaf margins, parallel veins, or leaf strapping. Rapid initiation of new leaf growth suggests less sugarbeet injury and less sugarbeet yield loss than a delayed initiation of new leaf growth. Sugarbeet injured with phenoxyacetic acids should be processed immediately after harvest because injured sugarbeet roots do not store as well as non-injured roots.
2. Benzoic Acids

a. Use: **Dicamba (Banvel)** for corn, wheat, oats, sorghum, pastures, and noncropland.

b. Injury Symptoms: Banvel causes sugarbeet injury that is visually indistinguishable from phenoxy acetic acid injury. Banvel may have sufficient residual in the soil to reduce emergence and to injure emerging sugarbeet. Emerging seedlings may be twisted and development of the first true leaves may be inhibited. Banvel residual in soil can cause the "trumpeting" symptom which also can be caused by early postemergence exposure to phenoxy acetic acid herbicides.

c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

3. Pyridines

a. Use: **Clopyralid (Stinger)** for small grains, sugarbeet, corn and grass pastures. **Picloram (Tordon)** for non-cropland, small grains, and grass pastures. **Triclopyr** for non-cropland and grass pasture.

b. Injury Symptoms: Pyridine injury symptoms are very similar to the phenoxy acetic acids or Banvel. Stinger is registered on sugarbeet but can injured sugarbeet at high rates with a warm, moist environment that favours phytotoxicity. The leaves lay flat and the petioles exhibit epinasty from Stinger just as from phenoxy acetic acids or Banvel. Also, leaves may become more strap-shaped than normal. However, Stinger injury often causes leaves to roll upward from the edges. Leaf rolling is caused to a greater extent by clopyralid than by phenoxy acetic acids or Banvel.

c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

Pyrimidinloxybenzoic acids are a group of benzoic acid herbicides including bispyribac and pyriminobac.
Amino Acid Synthesis Inhibitors

The amino acid synthesis inhibitors include the herbicide families: sulfonylurea, imidazolinone, sulfonamide and amino acid derivatives. Amino acid synthesis inhibitors act on a specific enzyme to prevent the production of specific amino acids, key building blocks for normal plant growth and development. Sulfonylurea, imidazolinone and sulfonamide herbicides prevent the production of three essential branch-chain amino acids by inhibiting one key plant enzyme. The amino acid derivative herbicides inhibit the production of three essential aromatic amino acids by inhibiting another key plant enzyme. In general, injury symptoms are slow to develop (one to two weeks) and include stunting or slowing of plant growth and a slow plant death. Herbicides in the sulfonylurea, imidazolinone and sulfonamide families can move in both the xylem and phloem to areas of new growth and can be taken up through plant foliage and roots. Herbicides in these families vary greatly in selectivity and may control annual and perennial broadleaf or grass weeds and may be soil or foliar applied.

Glyphosate, an amino acid derivative herbicide, is non-selective and the site of uptake is the plant foliage. Glyphosate moves via the phloem to all parts of the plant and is an excellent perennial weed control herbicide and is active on annual weeds as well.

1. Imidazolinones
   a. Use: Imazamethabenz (Assert) for wheat, barley and sunflower. Imazaquin (Scepter) for soybean and non-cropland. Imazethapyr (Pursuit) for soybean, dry bean, alfalfa, noncropland and pea.
   b. Injury Symptoms: Same as for sulfonylureas, see next section.
   c. Site of Action: Acetolactate synthase (ALS) or acetohydroxy acid synthase (AHAS) enzyme.

2. Triazolopyrimidine sulfonanilide or sulfonamide
   a. Use: Flumetsulam (Broadstrike) for corn and soybean.
   b. Injury Symptoms: Same as for sulfonylureas, (see next section).
   c. Site of Action: Acetolactate synthase (ALS) or acetohydroxy acid synthase (AHAS) enzyme.
3. Sulfonylureas


b. Injury Symptoms from Soil Residual: Symptoms from imidazolinone, sulfonylurea and sulfonamide herbicides are identical. Not all injured plants will exhibit all symptoms, and symptoms may vary from field to field. Sugarbeet plants may be stunted and the leaves usually become a bright yellow with first yellowing on young leaves. Relatively high levels of herbicide residual in soil may cause the plants to form a rosette rather than a normal sugarbeet plant. The total root and hypocotyl of sugarbeet seedlings may turn brown and shrivel or the root may turn brown and die starting at the point where the root joins the hypocotyl, about 1 to 1.5 inches below the soil surface. Nearly identical symptoms on roots of seedling sugarbeet also can be caused by dinitroaniline herbicides and *Aphanomyces cochlioides*, a fungal disease. Plants that survive and grow may produce new leaves that are more strap-shaped than normal.

c. Injury Symptoms from Postemergence Exposure: Symptoms from imidazolinone and sulfonylurea herbicides are identical. Not all injured plants will exhibit all symptoms and symptoms may vary from field to field. Plant leaves will become prostrate a few hours after exposure similar to the effect from phenoxy acetic acids, Banvel or pyridines. Old leaves may remain prostrate for several weeks. However, the petiole epinasty from imidazolinones or sulfonylureas is less than from phenoxy acetic acids, Banvel or pyridines. Yellowing of the youngest leaves begins about four to five days after exposure and the yellowing intensifies and spreads to the older leaves with time. Severely affected leaves or whole plants may die and turn brown. Petioles may turn black or have black streaks as symptoms worsen. The color contrast between affected and normal plants can become quite evident. The yellow may disappear later in the season as affected plants recover and begin to produce new leaves. Some affected plants may develop brown rings in the roots within five to seven days after exposure. These rings may still be present at harvest. Plants injured by imidazolinones or sulfonylureas often produce new leaves...
in clusters rather than in pairs. This can result in more than one crown per root. These plants may be more difficult to defoliate than normal plants. Young seedling exposure to imidazolinone or sulfonylurea herbicides can cause root symptoms similar to those from soil residual.

d. Site of Action: Acetolactate synthase (ALS) enzyme. Also referred to as acetohydroxy acid synthase (AHAS).

4. Amino Acid Derivatives

a. Use: Glyphosate (Several trade names) non-selective weed control before crop emergence, for spot treatments in some crops, in pasture and in non-cropland.

b. Injury Symptoms: Sugarbeet injury from glyphosate is quite similar to injury from imidazolinones or sulfonylureas. However, the yellowing from exposure to glyphosate starts with the older leaves and moves toward the younger leaves while injury from imidazolinones or sulfonylureas starts with the younger leaves and moves toward the older leaves. Glyphosate can cause brown coloring in the roots similar to the imidazolinones or sulfonylureas.

c. Site of Action: 5-enolpyruvyl-shikimate-3 phosphate synthase (EPSP synthase) enzyme.

Lipid Inhibitors

The lipid inhibitors include the aryloxyphenoxypropionate and cyclohexanedione herbicide families: These herbicides prevent the formation of fatty acids, components essential for the production of plant lipids. Lipids are vital to the integrity of cell membranes and to new plant growth. The lipid inhibitor herbicides inhibit a single key enzyme involved in fatty acid biogenesis. Broad-leaf plants are tolerant to these herbicide families, but almost all perennial and annual grasses are susceptible. Injury symptoms are slow to develop (seven to 10 days) and appear first on new leaves emerging from the whorl of the grass plant. These herbicides are taken up by the foliage and move in the phloem to areas of new growth.

1. Cyclohexanediones

a. Use: Sethoxydim (Poast Plus) for soybean and alfalfa. (Poast) for soybean, alfalfa, sugarbeet, flax, sunflower, lentil, pea and potato. Clethodim (Select) for soybean.
b. Injury Symptoms: Sugarbeet is not injured by cyclohex-anediones even at high rates.

c. Site of Action: Acetyl-CoA carboxylase enzyme.

2. Aryloxyphenoxypropionates

a. Use: Diclofop (Hoelon) for small grain. Fluazifop (Fusilade 2000) for soybean. Fenoxaprop (Whip, Option II) for soybean and (Dakota, Tiller, Cheyenne) on small grain. Quizalofop (Assure II) for soybean.

b. Injury Symptoms: Sugarbeet is not injured by aryloxyphenoxypropionates used alone but would be injured by mixtures with other herbicides.

c. Site of Action: Acetyl-CoA carboxylase enzyme.

**Seedling Growth Inhibitors**

The seedling growth inhibitors include the dinitroanilines, acetanilides and thiocarbamates. All of these herbicides must be used pre-plant incorporated or pre-emergence to the weeds to be effective. In some uses, the herbicides are applied after crop emergence but before weed emergence. Seedling growth inhibitors interfere with new plant growth, thereby reducing the ability of seedlings to develop normally in the soil. Plants can take up these herbicides after germinating, until the seedling emerges from the soil. Therefore, these herbicides are only effective on seedling annual or perennial weeds. Plants that have emerged from the soil uninjured are likely to remain unaffected.

Seedling growth inhibitors are active at two main sites, the developing shoot and the root. Much more is known about the action of seedling root inhibiting herbicides than seedling shoot inhibitor herbicides. The root inhibitors stop plant cells from dividing, which inhibits shoot elongation and lateral root formation. Uptake is through developing roots and shoots. Because herbicide movement within the plant is limited, herbicide injury is confined primarily to plant roots and shoots. Shoot inhibiting herbicides are taken up by developing roots and shoots and can move via the xylem to areas of new growth. Present evidence suggests that these herbicides can affect multiple sites within a plant, primarily interfering with lipid and protein synthesis.
A. Root Inhibitors

1. Dinitroanilines
   
a. Use: Ethalfuralin (Sonalan) for soybean, sunflower and dry bean. Pendimethalin (Prowl) soybean, dry bean, and sunflower. Trifluralin (Treflan) for soybean, dry bean, sunflower, alfalfa, small grain, Canola (rapeseed), mustard and pea.
   
b. Injury Symptoms: Dinitroaniline residual in soil may cause sugarbeet to be severely stunted with small leaves that are more erect than normal. The roots of damaged sugarbeet seedlings may turn brown and die starting at the point where the root joins the hypocotyl, about 1 to 1.5 inches below the soil surface. Plants with dead roots may die or they may survive by producing secondary roots from the hypocotyl. Identical symptoms on roots of seedling sugarbeet also can be caused by imidazolinone, sulfonylurea or sulfonamide herbicides and by Aphanomyces, a fungal disease. Affected plants will be smaller than unaffected plants. Plant regrowth by secondary root production would be prevented by drought in the surface 2 inches of soil and injured plants would die. Imidazolinone, sulfonylurea and sulfonamide herbicides also can cause similar seedling root death.
   
c. Site of Action: Tubulin protein involved in cell division.

B. Shoot Inhibitors

1. Acetanilides
   
a. Use: Alachlor (Lasso) for corn, dry bean, sorghum, sunflower and soybean. Acetochlor (Harness Plus, Surpass) for corn. Metolachlor (Dual) for corn, dry bean, sorghum, potato and soybean. Propachlor (Ramrod) for corn, flax, and sorghum. Dimethenamid (Frontier) for corn.
   
b. Injury Symptoms: Acetanilides cause no distinctive symptoms on sugarbeet. Some plants may die before emergence. Emerged but injured plants are normal in appearance but stunted.
   
c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

2. Thiocarbamates
   
a. Use: EPTC (Eptam) for alfalfa, potato, dry bean, flax, safflower, sugarbeet and sunflower. EPTC plus safener (Eradlcane) for corn. Butylate plus safener (Susan+) for corn. Triallate (Far-Go) for wheat barley, lentils and pea. Cycloate (Ro-Neet) for sugarbeet.
b. Injury Symptoms: Thiocarbamates reduce the formation of epicuticular wax on leaves, which can cause leaves to stick together rather than unfold normally. Affected plants may be stunted, leaves may be shortened and thickened, or true leaf development may be inhibited. Some severely stunted plants may die while others will start producing new leaves and will produce a nearly normal-sized root at harvest. Severely stunted plants may grow very little for two or more weeks after emergence and then make a complete or nearly complete recovery.

c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

**Photosynthesis Inhibitors**

The photosynthesis inhibitors include triazines, phenylureas, uracils, benzothiadiazoles, nitriles, carbamate dicarboxylic acid, phenylcarbamates, pyridazinones, triazinones, phenylpyridazines and amides. They inhibit photosynthesis by binding to the QB-binding niche on the D1 protein of the photosystem II complex in chloroplast thylakoid membranes. Herbicide binding at this protein location blocks electron transport from QA to QB and stops CO2 fixation and production of ATP and NADPH2 which are all needed for plant growth. However, plant death occurs by other processes in most cases. Inability to reoxidize QA promotes the formation of triplet state chlorophyll which interacts with ground state oxygen to form singlet oxygen. Both triplet chlorophyll and singlet oxygen can abstract hydrogen from unsaturated lipids, producing a lipid radical and initiating a chain reaction of lipid peroxidation. Lipids and proteins are attacked and oxidized, resulting in loss of chlorophyll and carotenoids and in leaky membranes which allow cells and cell organelles to dry and disintegrate rapidly. Some compounds in this group may also inhibit carotenoid biosynthesis (fluometuron) or synthesis of anthocyanin, RNA, and proteins (propanil), as well as effects on the plasmalemma (propanil) (Devine et al. 1993).

Inhibition of photosynthesis could result in a slow starvation of the plant; however, in many situations rapid death occurs perhaps from the production of secondary toxic substances.

Injury symptoms include yellowing (chlorosis) of leaf tissue followed by death (necrosis) of the tissue. Three of the herbicide families (triazines, phenylureas and uracils) are taken up into the plant via the roots or foliage and move in the xylem to plant leaves. As a result, injury symptoms will first appear on the older leaves, along the leaf margin. Foliar applied photosynthetic
inhibitors generally remain in the foliar portions of the treated plant and movement from foliage to roots is negligible.

1. Triazines

a: Use: Ametryn (Evik) for corn. Atrazine for corn and sorghum. Cyanazine (Bladex) for corn. Simazine (Princep) for corn. Metribuzin (Lexone, Sencor) for alfalfa, soybean, potato, pea and lentil. Hexazinone (Velpar) for alfalfa.

b. Injury Symptoms: Residual of photosynthesis inhibitors in soil does not prevent seedlings from germinating or emerging. Injury symptoms occur after emergence and the speed of appearance of symptoms will be more rapid with sunny days than with cloudy weather. Also, symptoms will be more severe and more rapid as the level of herbicide in the soil increases. Sugarbeet plants may be in the two- to four-leaf stage before symptoms become noticeable but plants can die in the early two-leaf stage. Initial symptoms include browning of the cotyledonary leaves and yellowing of the true leaf margins. Browning of leaves will increase with time and total desiccation may result. Older and larger leaves are affected before younger leaves. Postemergence triazines cause an initial yellowing followed by desiccation and leaf browning.


2. Phenylureas

a: Use: Linuron (Lorox) for soybean and corn. Tebuthiuron (Spike) for grass pasture and noncropland.

b. Injury Symptoms: Same as for the triazine herbicides.


3. Uracils

a. Use: Terbacil (Sinbar) for alfalfa.

b. Injury Symptoms: Same as for triazine herbicides.

4. Benzothiadiazoles

a. Use: Bentazon (Basagran) for soybean, corn, dry bean and grain sorghum.

b. Injury Symptoms: Leaves become chlorotic and later turn brown and die. The older leaves die first. All the older leaves can turn brown while the growing point remains green. Sugarbeet can recover, produce new leaves and produce a nearly normal-size root at harvest if the growing point survives.


5. Nitriles

a. Use: Bromoxynil (Buctril) for wheat, barley, oats, rye, flax, corn and alfalfa.

b. Injury Symptoms: Leaves become chlorotic and later turn brown and die. Contact with isolated spray droplets may cause a spotting or speckling of the leaves. The older sugarbeet leaves will be affected more than the young leaves. Sugarbeet can produce new leaves and a harvestable root if the growing point survives.


6. Carbamate

a. Use: Desmedipham (Betanex) for sugarbeet. Desmedipham + Phenmedipham (Betamix) for sugarbeet.

b. Injury Symptoms: Desmedipham and phenmedipham are registered for sugarbeet but injury sometimes occurs, most often in a hot and moist environment. Symptoms from des-medipham and phenmediham are very similar to symptoms from bentazon and bromoxynil. Injured leaves may turn brown and die. The older leaves die first and the growing point may remain green and alive even when most leaves are dead. Sugarbeet plants with a surviving growing point will produce new leaves and a nearly normal size root at harvest.

7. Dicarboxylic Acid

a. Use: Endothall (H-273) for sugarbeet.

b. Injury Symptoms: Endothall is registered for sugarbeet but injury sometimes occurs, most often in a hot and moist environment. Symptoms from endothall are very similar to symptoms from bentazon, bromoxynil, desmedipham and phenmedipham. Injured leaves turn brown and die. The older leaves die first and the growing point may remain green and alive even when most leaves are dead. Sugarbeet plants with a surviving growing point will produce new leaves and a nearly normal size root at harvest.


Cell Membrane Disrupters

The cell membrane disrupters include the diphenylether and bipyridylium herbicide families. These herbicides are postemergence contact herbicides that are activated by exposure to sunlight to form oxygen compounds such as hydrogen peroxide. These oxygen compounds destroy plant tissue by rupturing plant cell membranes. Destruction of cell membranes results in a rapid browning (necrosis) of plant tissue. On a bright and sunny day, herbicide injury symptoms can occur in one to two hours. Because these are contact herbicides, they are excellent for burn down of existing foliage and postemergence control of annual weeds. Perennial weeds usually regrow because the herbicides do not move to underground root or shoot systems. These herbicides have little phytotoxicity through the soil.

1. Bipyridyliums

a. Use: Paraquat (Gramoxone Extra) for non-selective weed control in corn, soybean, dry bean, sunflower, sugarbeet, small grains and dormant alfalfa and for desiccation of potato and sunflower.

Difenzoquat (Avenge) for barley, winter wheat and some spring and durum wheat varieties.

b. Injury Symptoms: Drift on sugarbeet often will appear as spotting of leaf tissue. High amounts of drift or an accidental application may cause patches of brown tissue on leaves. Spots from
bipyridylium drift have been confused with foliar diseases such as Cercospora or bacterial blight. Generally the pattern of injury in a field can be used to distinguish between disease and drift. If in doubt, samples should be taken to a diagnostic laboratory for disease identification.

c. Site of Action: Activated by photosystem I (PSI).

2. Diphenylethers

a. Use: Acifluorfen (Blazer), Lactofen (Cobra) and Fomesafen (Reflex) for soybean.

b. Injury Symptoms: Affected leaves will exhibit dessication where the herbicide contacted the plant. Drift generally will not kill sugarbeet but the plants may be severely stunted. New leaf growth will appear normal.

c. Site of Action: Inhibition of photoporphyrinogen oxidase (Protox).

Pigment Inhibitors

Pigment inhibitors prevent plants from forming photosynthetic pigments. As a result, the affected plant parts become white to translucent. Clomazone (Command), a soil-applied herbicide, is the only member of the isoxazolidinone family in use at this time. Clomazone is taken up by plant roots and shoots and can move in the xylem to plant leaves. The newly developed foliage of many plant species is very susceptible to Clomazone and very small amounts can whiten new plant growth. Susceptible weeds will emerge as white plants before dying.

1. Isoxazolidinones

a. Use: Clomazone (Command) for soybean.

b. Injury Symptoms: All or portions of the true leaves will turn white. Sugarbeet is relatively tolerant of Clomazone residual in soil and generally plants with symptoms will live, turn green, and produce a nearly normal-size root at harvest.

c. Site of Action: Specific sites unknown.
Selectivity of herbicides

Different plant species respond differently to same herbicide and same plant species respond differently to different herbicides. The susceptibility or tolerance of different plants to herbicide is called herbicide selectivity. It is the phenomenon/mechanism by which in a mixed stand of plants, some plant species (e.g. weeds) are preferentially controlled or killed, while others (e.g. crop plants) remain unaffected or negligibly affected when a herbicide is applied to them. It is an important factor towards weed control through herbicides and crop safety. Selectivity is a complex phenomenon achieved by complex interaction between plant, herbicide and environment (climate and soil).

Many plants can deactivate or metabolize herbicide before plant injury occurs. The genetics of a plant also affect a plant's tolerance to herbicide. Many plant-related factors determine herbicide selectivity. Other factors include differences in herbicide retention, penetration, translocation and metabolism. It is also important to remember that selectivity depends upon herbicide dosage.

Kinds of selectivity

There are four types of selectivity viz., physical, chemical, biological/biochemical and chronological. One or more than one kind of selectivity operate/work under a given situation.

1. Physical selectivity: Selectivity achieved due to manipulation of physical or environment factors is called physical selectivity.
   a) Depth protection selectivity
      Pre-emergence herbicides inhibit germination of surface lain or shallow-placed (0-3.0 cm depth) weed seeds, whereas most crop seeds (except a few like pearl/finger millet, teff having very small seeds), which are sown little deeper in soil are not attacked by these herbicides and their germination is not inhibited. Thus the selectivity achieved due to protection in depth of sowing of crops is called “depth protection selectivity”.
   b) Externally-working antidote-mediated selectivity
      Externally-working antidotes such as activated carbon/charcoal pose physical barriers to herbicide uptake to the lethal level. They adsorb/absorb certain amount of herbicide applied to soil and interfere directly with uptake and indirectly with the metabolism of herbicide. Some antidotes may even compete for site of action.
c) **Application device protection selectivity of non-selective herbicides**

Non-selective herbicides are made selective to some standing crops by modifying the application technique or device. For example, by using a spray hood 2,4-D is made selective to jute, a broad-leaved crop. Similarly, paraquat and glyphosate are made selective in broadly spaced crops by directing their spray only to weeds, or by using herbicide gloves, rope-wick applicator etc.

**d) **Selectivity to non-selective herbicides achieved due to other means**

Non-selective herbicides could be made selective by modifying the crop environment. For example, at the early stages of sapling growth of plantation, fruit and orchard crops, the rows of saplings planted may be covered by polythene sheet during spray of non-selective herbicides like glyphosate and paraquat. The polythene sheet is removed after spray. Even in tall maize crop, smearing weeds by a cloth wet with non-selective herbicide solution could be of enough use towards weed control at the later stages.

2. **Chemical selectivity:** Selectivity achieved due to structural changes (change in functional group or side chains of the phenyl/benzene ring) of the herbicides is called chemical selectivity. Chlorotoluron has “-CH₃” group, while diuron has “-Cl” group on the 4th carbon (4C) position of the ethyl/benzene ring. Otherwise, they both have similar structures and belong to phenylurea group. Due to this difference, diuron is selective to cotton, while chlorotoluron is selective to wheat.

3. **Biological/biochemical selectivity:** Selectivity achieved due to metabolism or reverse metabolism of herbicides inside plants is called biological/biochemical selectivity. This an example of true selectivity, which can hardly be manipulated by extraneous factors at least at the recommended dose of herbicide. However, due to continuous use of herbicide, plants may undergo certain internal physiological changes towards developing resistance in course of time. Then also selectivity may operate in plants, but may be dose-dependent or shift in selectivity may take place between plant species. Tank-mix or ready-mix internally-active antidotes are directly involved with herbicide translocation and metabolism inside plants. They offer selectivity of certain herbicides to some crops.

4. **Chronological selectivity:** Selectivity achieved due to manipulation of the time of application of an herbicide such as pre-planting, pre-emergence or post-emergence is called chronological selectivity. Pendimethalin is selective to crops, when applied as pre-
emergence, but not as post-emergence. Metribuzin is selective to potato as pre-emergence, but not as post-emergence.

**Selective Mechanism of Herbicides May Occur Due To These Aspects**

1) Differential rate of absorption of herbicides
2) Differential rate of translocation of herbicides
3) Differential rate of deactivation of applied herbicides
4) Protoplasmic resistance to the specific herbicide

1. *Differential absorption of herbicides:* Absorption of 2, 4-D in wild cucumber (*Sicyos angulatus*) is so slow that it keep pace with its metabolism easily thus the plant proved tolerant while the cultivated cucumber (*Cucumis sativus*) succumb to death due to faster absorption. Similarly, bigleaf maple (*Acer macrophyllum*) are tolerant to amitrole due to its slower absorption while, bean and lucerne plants are susceptible due to fast absorption of the herbicide by them. Under field conditions, differential absorption of herbicides may occur due to many reasons

   A. Plant species may differ in their morphology and growth habits.
   B. Herbicides may be applied at different times by different methods.
   C. Use of antidotes and adsorbents to prevent herbicide absorption by non target plants.
   D. Herbicide formulations may differ in their ability to contact with non target plants.

The selectivity may be due to one or combination of processes.

A. *Differences in morphology of plants:* Certain morphological features allow limited retention of aqueous herbicides on their foliage. They are

   i. Narrow upright leaves.
   ii. Corrugated (or) finely ridged leaf surfaces.
   iii. Waxy leaf surface.
   iv. Pubescent leaves.

Pea, onion, sugarcane, cabbage and colacasia posses the above morphological features. The herbicide bounce off as droplets from their foliage or small area may be wetted. Crops like wheat and sugarcane are protected by herbicide sprays by covered growing point. The limited spray retention provides resistance against selective contact herbicides without any wetting agents.
With translocated herbicides, limited spray retention is not of much help in protecting the non-target plants from herbicide injury. Post emergence application of bromoxynil and ioxynil controlled many broad leaved weeds in wheat crop due to limited spray retention. Likewise selectivity of nitrofen in case of rice and brassica is due to differential wetting. In recent years importance of limited wetting of crop plants as a factor in herbicide selectivity has diminished.

**B. Differences in growth habit of plants**

**I. Shoot growth difference:** When crop rows have a clear advantage in height over the inter row weeds, directed spraying of herbicides is a common method of achieving selective control of weeds. Herbicide mulches are used in standing crop rows for affecting selective control of germinating weeds. In slow germinating crops like potato and sugarcane, weeds often establish themselves even before crop emergence; hence they are controlled selectively by spraying a contact herbicide before more than 10% of crop plants are seen over the ground.

In more advanced stages of crop growth, sometimes specific weed sp may grow much above the crop height. In crops like spinach and Egyptian clover, these tall weeds may often completely hide the crop plants. In USA, herbicide-laden wax bars have been employed successfully for the control of tall weeds in wide row crops. In the closely sown crops a low volume application of a contact herbicide can be used. In lawns and gardens shoots of nutsedge and other erect weeds can be selectively wiped with herbicides from either with herbicide- laden wax bars (or) clothed stick dipped in concentrated herbicide solution.

**II. Root growth differences:** When herbicides are applied to soil, differences in the growth habit of underground parts of weeds and crop plants become important in determining their selective absorption. In general weed seeds germinate from top 1.25-1.5 cm of soil, whereas many crop seeds are planted 5 to 7.5 cm deep. When a recommended pre-emergence herbicide is applied on the soil surface and the soil moisture conditions are suitable to leach it to about 2.5 – 3 cm soil depth, it is readily available for absorption to the germinating weeds. Crop plants that grow their roots beyond 5 cm depth obviously avoid herbicide absorption and escape phytotoxicity. This is the basic principle of selectivity of most of the pre-emergence herbicides. Basic principle of selectivity of pre emergence herbicide is a function of herbicide structure, formulation and rate besides soil texture, organisms and inorganic colloids and rain fall. When
any of these factors is unfavorable herbicide may either injure the crops or poor weed control is seen. Sometimes, both these adverse effects may occur together then the phenomenon is called ‘reverse selectivity’.

Selectivity of mollinate between rice and *E. colonum* is due to differences in crown root initiation levels of two grasses. CRI is close to surface in *E. Colonum* but in rice CRI is below the soils surface in herbicide free zone. Selectivity of Trifluralin in wheat and green foxtail (*Setaria viridis*) is due to differences in coleoptilar nodes, irrespective of depth from which they are germinated. In *S. viridis* it is within 1 cm of the soil surface whereas in wheat it is much deeper.

### III. Use of adsorbents and antidotes (Induced selectivity)

**(a) Adsorbents:** These are the materials having ability to adsorb herbicides which are placed near crop seed. Activated charcoal is strong absorbent of 2,4-D, EPTC, 2, 4, 5-T, propanam, propachlor, pyrazon, trifluralin, chloramben, diuron, butachlor, simazine etc. When a germinating seed is surrounded by a layer of activated charcoal, then seed is prevented from absorption of soil applied pre-emergence herbicides. Mostly in horticultural crops activated charcoal is placed over the crop seeds. Activated charcoal is first used as an adsorbent of 2,4-D. In transplanted horticultural crops, roots of seedlings are dipped in a charcoal before transplanting. Seed pelleting with charcoal has been developed in recent years using gum/ PVA (poly vinyl acetate) for increasing the selectivity of ETPC to maize and cowpea, and of chloramben, butachlor and EPTC to rice.

**(b) Antidote (safener):** Safeners are chemicals discovered to antagonize phytotoxicity of specific herbicides to specific plant species. Safeners prove successful against herbicides which inhibit cell division. Otto L Hoffman – father of safeners, as early as in 1948 observed antagonism of 2,4-D to 2,4,6-T on tomato plants. By 1969 he discovered and reported NA (1, 8 Naphthalic anhydride) as highly successful safener of EPTC and butylate in maize. Effective dose is 0.5 g per kg seed. It should be applied as seed dress. Later maize specific safener of EPTC and butylate, namely R-25788 (N, N - dially 1-2, 2, dichloroacetamide) was discovered. The dose of the soil applied R-25788 is 0.6 kg/ha. It has further been found an antidote of metachlor and alachlor in protecting maize seedlings. A seed coating has been found to provide
protection to cultivated oat against pre-emergence alachlor and maize and sorghum against perfluidone and diclofop. CGA-43089 provide safety to sorghum against metalachlor by seed treatment at 1-1.5 kg per ha.

The granules filters through crop foliage leaving very little for absorption, then settle over ground where the weeds will absorb. The important desirable character of herbicide granules is, low leach ability in soils. e.g. Chlorpropham, Dinoeb, diuron and nitrofen.

2. Differential rates of translocation of herbicides: Plants can translocate herbicide through the plant as much herbicide it absorbs. Equal amounts of herbicides may be absorbed by plants and weeds but translocation rates may be different. For example 2,4,5-T is more toxic to Cucumis trigonus than 2,4-D because it was translocated much more rapidly than the latter compound inside plants. Likewise differences in the selectivity between sugarcane (tolerant) and beans (susceptible) to 2,4-D is on the basis of its slow translocation in sugarcane and rapid translocation in beans. Always faster translocation does not mean quick killing. In certain cases it will help the plants in escaping specific herbicide action. For instance, diphenamide is selective to Convolvulus arvensis because the herbicide is translocated very rapidly from shoots to the roots where it gets metabolized very rapidly than in Avena sativa (it fails to transmit very rapidly from roots to shoots). Soybean has tolerance to oxyflourfen due to its slow absorption.

3. Differential rates of deactivation of herbicides: Selectivity is primarily a function of differential rate of deactivation. A tolerant plant species deactivates the herbicide molecule rapidly, whereas a susceptible species deactivates it slowly. This deactivation may be a process of i) metabolism, ii) reverse metabolism and iii) conjugation. Reverse metabolism is important mode of herbicide dissipation. Conversion of active herbicide to inactive form is metabolism where as conversion of inactive to active herbicide form is reverse metabolism.

a. Metabolism: It involves a change in molecular structure of applied herbicides inside the plants. Ribes nigrum is susceptible to 2,4-D. It metabolises the 2% of herbicide applied in 96 hours. Whereas Ribes sativum is tolerant to 2,4-D. It metabolizes 50% of applied amount within 96 hours. Mentha piperata (tolerant) metabolised terbacil rapidly and shown temporary fall in photosynthesis but in Ipomea hederaceae (susceptible) terbacil persisted for long time to inhibit photosynthesis. Rice is tolerant to bensulfuron due to rapid metabolism inside the plant.
b. **Reverse Metabolism (inactive to active):** This is an enzymatic beta oxidation process. Intermediate chemical compounds are more phytotoxic than original compounds (parent compounds). Even number carbon $ω$ Phenoxy Alkanoic acid compounds like 2,4-DB and MCPB are non toxic but in plants they are converted to 2,4-D and MCPA (these are more toxic). This is due to enzymatic oxidation in non-leguminous plants. But legumes like lucerne, berseem, peas and clovers lack B-oxidation and are tolerant to 2,4-DB and MPCB.

c. **Conjugation:** Coupling of intact herbicide molecule with some plant cell constituents in living plants. Tolerance of grasses and *Convolvulus arvensis* to 2,4-D, this conjugate with glucose and form glucoside, $β$- D glucose ester of 2,4-D. Binding of 2,4-D on certain protein films in tolerant graminaceous members e.g. sugarcane. It takes toxic herbicide concentration out of the main stream and makes tolerant. In soybean chloramben- translocate rapidly to roots and conjugated with glucose molecules forming N-glucosyl chloramben and an unknown compound Chloroamiben –X. In apple, maize and certain millets atrazine and simazine are deactivated by conjugation. Enzyme responsible for conjugation in maize is glutathione–S-Transferase. This catalyses conjugation of simazine with reduced glutathione to form S- Glutathion and chloride ion released during this process. Likewise propanil selective to rice (tolerant); phytotoxic to *Echinocloa colonum* (susceptible) due to an enzyme called arylacylamidine amidohydrolase content in leaves. In Barnyard grass the enzyme is less by 1/60 th as that in rice. In rice, leaves able to hydrolyze propanil to non phytotoxic compounds 3,4-dichloro aniline and propionic acid.

**4. Differential protoplasmic resistance**

Protoplasm of different plant species differing in withstanding abnormal deficiencies or excess constituents, that may be induced in the presence of some specific herbicide molecules. e.g. plant show tolerance to dalapon can withstand pantothenic acid deficiency and resist precipitation of cell protein. Buffering mechanism of protoplasm of plants is affected differently by different herbicides. Eg. Tolerance of mustard, groundnut and cotton to trifluralin and nitriles is due to their inherent protoplasmic resistance. Tolerance of rice plants to molinate is due to protoplasmic tolerance.

**Factors affecting herbicide effectiveness**

**A. Plant factors**
1. Morphology of plants

   i) Leaf/foliage/canopy

      a) Nature and orientation of leaf: The nature and orientation of leaves directly affect the interception and retention of a herbicide and indirectly its subsequent penetration and translocation. Higher the interception and retention, greater is the penetration and translocation. Broad-leaved plants having bigger size, greater area and horizontal orientation of leaves are likely to intercept more amount of herbicide than grasses which have narrow, erect and upright leaves of smaller size and area. As a result, broad-leaved plants are more prone to be killed by a herbicide than grasses.

      b) Pubescence and waxiness of leaf/canopy: A waxy surface or one that has a lot of hairs will reduce amount of herbicide contacting plant/leaf surface. If hairs are less on the leaf surface, the droplets retained in between hairs may require more time to be absorbed. The more hairy/pubescent plant surface will have less contact and retention of spray-droplet and hence will absorb smaller amount of spray than smooth and less hairy surface.

      c) Roughness and corrugation of leaf: A rough and highly corrugated surface will reduce amount of herbicide contacting leaf surface and hence affect penetration. The distribution of spray droplets is generally non-uniform on the rough and corrugated leaves and there are chances of spray droplets bouncing back from leaves.

      d) Composition and thickness of cuticle: The thickness of cuticle directly affects the penetration of a herbicide and indirectly its translocation through plant system. The thicker the cuticle, lesser is the penetration.

   ii) Root system: Herbicides like atrazine remains mainly in the upper layers of soil and affect the plant roots lying in that zone of soil. Plants with roots below this layer will be hardly affected. The pre-emergence application of herbicides is based on this philosophy, where shallow-rooted annual weeds are controlled, while deep-rooted crops mostly remain safe.

   iii) Age/stage of the plants: Generally younger are the plants, greater are the susceptibility. Younger plants usually have tender less-developed roots, more meristematic tissues and
Advances in Weed Management

growing activities, elongate very rapidly and less thick cuticle than older ones and, therefore, are likely to be more prone to herbicide.

2. Physiology of plants

i) **Uptake/absorption:** The availability of herbicide may be same on the site of application (e.g. leaves, stems, roots), but uptake may vary due to genetic factors. Herbicide absorption by roots in most cases is passive and therefore, factors affecting transpiration may affect absorption of herbicide by roots. These are the inherent characteristics of plants and not under the control of man or applicator. Resistant or tolerant plants usually absorb and translocate less amounts of herbicides compared to susceptible ones.

ii) **Translocation:** Differential absorption of herbicides by plants results differences in the translocation of herbicides inside plants. Susceptible plants translocate more amount of herbicide compared to resistant or tolerant ones. Translocation may be apoplastic, symplastic or both based on herbicide. Translocated/systemic herbicides may move from site of application to site of action via the phloem (symplast), xylem (apoplast) or both, but contact herbicide have very little or no movement from the site of application.

iii) **Metabolism of herbicide:** Metabolism is the most dominant hypothesis/mechanism in support of herbicides selectivity to plants. Plants differ in their ability to metabolize herbicides. Once a herbicide has entered into the plants, metabolism is faster than accumulation in some plants. As a result, lethal herbicide amount at the site of action hardly build up and plants show tolerance, whereas reverse is true for the plants showing susceptibility or mortality. For example, isoproturon, a substituted phenylurea herbicide and photosynthesis (PS II) inhibitor, is selective to wheat due to its increased detoxification inside wheat plants, whereas grass weeds, namely *Phalaris minor* and *Avena fatua* are killed because of the decomposition of herbicide is slower.

B. Herbicide factors

i) **Chemical family:** Herbicides are synthesized with chemical modification of a ‘base compound’ and are grouped under a chemical family/class named after the base compound, e.g. dinitrophenols, dinitroanilines etc. Herbicides of one chemical class differ from those of another class. Herbicides within a class differ from each other as well as may have some
common physical and chemical properties (toxicities, adsorption, water solubility, vapour pressure), which render them selective or non-selective to group of plants.

ii) **Molecular make-up of herbicides:** A particular molecular structure is required for a herbicide to become selective to crops. Herbicides even within a group show different degree of activity and selectivity mainly due to different molecular structure. Diuron, a substituted phenylurea herbicide, is selective to cotton and sugarcane, but phytotoxic to wheat. When “para chlorine atom” (p-Cl) of the phenyl ring of diuron is changed by a “methyl” (-CH$_3$) group, the compound formed is chlortoluron, another herbicide of substituted phenylureas. Chlortoluron is selective to wheat unlike diuron.

iii) **Formulation of herbicide:** In granular formulation, the herbicide/toxicant remains impregnated inside the granules and does not come in direct contact of crops and weeds when spread in the field. Granular formulation thus offers a general selectivity to established crop plants and associated germinating weeds. Granular ensure slow release and thereby lower leaching of the herbicide in soil, which provides greater opportunity of selective absorption of herbicide by crops and weeds emerging from different stratum of soil. Herbicides applied on the soil surface hardly reach to crop roots lying deeper in soil and thus crops remain safe.

iv) **Time of herbicide application:** Pre-plant incorporation (PPI) and pre-emergence applications offer more selectivity to a crop than a post-emergence application. In their case, selectivity to crops is achieved mainly due to depth protection. However, plant morphology, physiology/metabolism and age/stage of plants play a dominant role towards selectivity under post-emergence application.

v) **Dosages of herbicide:** A herbicide is selective to a crop at a particular dose called ‘recommended dose’ and therefore, selectivity is dose-dependent. For example, atrazine is selective to maize when it is applied at the recommended dose (1.0-1.5 kg/ha) or little higher dose (2.0-3.0 kg/ha) than recommended depending on organic matter status of soil. At higher doses, the conducting tissues of plants may be damaged and further translocation of herbicide may be cut-off.
vi) **Adjuvants:** Adjuvants such as surfactants (surface active agents), spreaders (help the herbicide to spread out on the leaf rather than in one droplet) and stickers (help the herbicide to stay on the leaf and to penetrate) are added with herbicides to prepare formulations/commercial products. They are generally non-toxic, but increase the potency of a herbicide by increasing the availability of herbicide at the site of action.

vii) **Safeners/antidotes/crop protectants:** Safeners are used where weeds and crops are closely related morphologically or when herbicide is less selective or non-selective. Safeners may make some physical barrier on crops roots or foliage or have chemical reaction with herbicide and thus render non-selective herbicide to become selective. Several antidotes viz. NA (1,8-napthalic anhydride), R 25788, CGA 43089, CGA 92194, MON 4604 have been used against a number of herbicides to make those selective to a number of crops.

C. **Environmental factors**

**Climatic factors**

a) **Temperature:** If there is an increase in temperature, greater uptake and translocation of herbicides are expected to occur because of higher transpiration. Metabolism may also take place at higher rate. For example, isoproturon and barben have faster metabolism at higher temperatures and thereby have less activity.

b) **Relative humidity:** The lower leaf surface is usually more permeable to a herbicide. At higher humidity, the foliage-applied herbicide droplets will be retained for more time on the leaf/canopy due to slower evaporation and hence more penetration and absorption of herbicide through the leaf cuticle are expected.

c) **Sunlight:** Light helps herbicide penetration by stimulating stomatal opening and movement from the leaf to other parts of the plants by activating photosynthesis leading to greater movement of organic solutes. Light is a pre-requisite for the activity of all the herbicides since nearly 80% of the herbicides are affecting photosynthesis directly or indirectly.

d) **Rainfall:** Rain soon after application washes of the spray deposits off the foliage and therefore, reduces herbicide effectiveness. However, the nature, amount, duration and distribution of rainfall may play a great role towards herbicides activity and selectivity.
Soil factors

a) **Organic matter content**: Organic matter having huge surface area and negative charges influences herbicide availability to plants. Higher the organic matter content, higher is the dose of herbicide required to achieve the same level of efficiency of a herbicide. Weak base herbicides (triazines, triazinones, trizoles, pyridinones), cationic herbicides (bipyridiliums) and non-ionic polar and apolar herbicides undergo sorption with soil colloids and become unavailable to plants. This results in decreased activity of the herbicides.

b) **Texture and structure of soil**: Heavily textured clayey soil requires more dose of herbicide compared to light-textured sandy soil for the same degree of weed control. Leaching of herbicide is more in light soil, whereas adsorption is more in heavy soil. In light soil, leaching, therefore, may cause phytotoxicity and affect selectivity by reaching to the crop root zone even though the dose of herbicide is less.

c) **Soil temperature**: Soil temperature affects microbial growth and activity, photo- and chemical decomposition and thereby plays a role in herbicide metabolism. Metabolism of herbicide increases in warmer soil than in cold soil. For example, the half life ($T_{1/2}$) of isoproturon is 24 days at 30°C, whereas 12 day at 40°C.

d) **Soil moisture**: Microbial degradation of herbicide is higher at higher soil moisture. This results in the lower persistence of herbicide in soil. The half life of isoproturon is 40 days in dry soil, 25 days at field capacity, whereas 10 days in flooded soil.

e) **Soil pH**: Generally, herbicides exist in ionic form between pH 4.3-7.5 with an increase in ionic concentration being proportional to an increase on pH. The negatively charged functional groups on the soil predominate at pH above 5.3 and repel anionic forms of herbicides such as 2,4-D, dicamba. Thus increase in CEC of a soil in moderate to alkaline pH due to increase in pH-dependent site of the colloids affects adsorption of ionic/polar herbicides. However, non-polar/apolar herbicides are hardly affected by soil pH.
Climatic factors and phytotoxicity of herbicides

Phytotoxic means harmful or lethal to plants. Phytotoxicity is the degree to which a chemical or other compound is toxic to plants. All types of pesticides can injure or kill plants. Herbicides are especially hazardous to plants because they are designed to kill or suppress plants. Some insecticides and fungicides can also harm plants. Phytotoxic effects caused by herbicides can be from spray droplets, soil residues or vapours contacting sensitive plants. Plants can also be harmed by herbicides which move-off target in water or soil or when sensitive crops are planted in fields too soon after a herbicide treatment was applied for a previous crop.

Phytotoxic properties of pesticides are usually associated with specific formulations (wettable powder, emulsifiable concentrate, granule, etc) or specific plants rather than groups of pesticides or plants. Phytotoxic effects can range from slight burning or browning of leaves to death of the plant. Sometimes the damage appears as distorted leaves, fruit, flowers or stems. Damage symptoms vary with the pesticide and the type of plant that has been affected.

Phytotoxicity is not necessarily caused by the active ingredient. Plant damage can also be caused by: the solvents in a formulation, impurities in spray water, using more pesticide than listed on the label, or poorly mixing of the spray solutions. Condition of the plant at the time of treatment can affect phytotoxicity; stressed plants may be more susceptible. The variability of the weather is a major cause of unreliable herbicide performance resulting in either inadequate control of weeds or crop damage. In order to meet the most economic use of herbicides, with minimal adverse effect on the environment, it is important to know how light, temperature, humidity and rain affect herbicide performance. Environmental conditions such the temperature, humidity, and light can influence phytotoxicity. High temperatures can speed up pesticide degradation and

<table>
<thead>
<tr>
<th>All herbicides are affected by weather conditions. Some generalisations are:-</th>
</tr>
</thead>
<tbody>
<tr>
<td>• <strong>High temperature and low humidity</strong> - poor weed control as plants produce thicker, less penetrable cuticle, spray droplets dry faster and stomata are closed to prevent water loss.</td>
</tr>
<tr>
<td>• <strong>High relative humidity</strong> - good weed control. Stomata remain open and spray droplets dry slowly.</td>
</tr>
<tr>
<td>• <strong>Warm temperatures but not too hot</strong> - usually improve weed control as these conditions promote plant growth and the entry and movement of herbicides inside the plant.</td>
</tr>
<tr>
<td>• <strong>Rainfall</strong> - effect depends on the herbicide; - glyphosate is less effective as this herbicide enters the plant slowly and so is washed off the leaves; paraquat is not affected as this herbicide enters foliage very rapidly.</td>
</tr>
</tbody>
</table>
volatilization, but may also result in increased phytotoxicity for some products. UV light rapidly breaks down many pesticides. Soil properties such as texture, temperature, moisture, microbial activity and pH also influence phytotoxicity. Higher pH soils are less binding and may increase photoxicity. High microbial activity can reduce phytotoxicity.

Certain weather criteria must be met for successful application of pesticides and wide use is made of weather forecasts in decision making for spray application. It is less widely appreciated that, even when these criteria for spraying are met, environmental conditions before and after application exert a major influence on herbicide activity which may result in either inadequate of weeds or crop damage. Field studies provide an overall view of herbicide performance under contrasting soil and weather conditions, but usually the role of individual environmental factors cannot be established. However, the importance of individual factors can be ranked in controlled environment experiments. This approach allows the conditions for optimum herbicide performance to be defined in environment/herbicide performance profiles (EHPPs).

The pre-spraying environment

During this period the weather affects the size, form, habit and cuticular characteristics of shoots, all factors of particular importance in relation to the efficacy of post-emergence herbicides. Similarly root development and position in the soil profile are affected, which is of importance for herbicides with soil activity. The steps in herbicide activity that are involved include interception, retention, foliar penetration and to a lesser extent, movement of the herbicide. The pre-spraying environment will also affect the development of storage and regeneration organs in perennials to which herbicide must be transported for effective control. The long and short-term pre-spraying environment affects the morphology and physiology of the plant and may subsequently alter the performance of herbicides.

Constraints on spraying

Herbicides should be applied when the tolerance of the crop and susceptibility of the weed are both optimal. The duration of this period depends on the specific herbicide/crop/weed/weather situation. Environmental factors affect the rate of development of the crop and weed, and adverse conditions such as rain and wind limit the opportunities for spraying. It was found that spray application of phenoxy herbicides to cereals was limited to around 20 days by the growth stages.
of the crop and weeds. Levels of rain and wind which prevented spraying reduced this period to between 4 and 8 days. To some extent the flexibility of spraying programmes has been increased by newer herbicides that can be applied to a wider range of growth stages and during longer periods of the growing season. In addition use of controlled drop application and low ground pressure spray vehicles make it possible to spray herbicides under less favourable environment conditions. In spite of these advances, herbicide applications are frequently made under suboptimal conditions due to lack of ‘good spray days’.

**The post application environment**

The hours immediately following application are of paramount importance to post-emergence treatments. Herbicide deposits on the surface of the plant are vulnerable to removal by, for example, heavy rain, and the success of a treatment is not assured until a lethal quantity has penetrated the cuticle.

**PPI AND PRE HERBICIDES**

**Incorporation of herbicides**

Good weed control with PPI and PRE herbicides depends on many factors, including rainfall after application, soil moisture, soil temperature, soil type and weed species. For these reasons, PRE herbicides applied to the soil surface sometimes fail to control weeds. Herbicides that are incorporated into the soil surface usually require less rainfall after application for effective weed control than unincorporated herbicides. Small weeds just emerging through a PRE herbicide may be controlled by a rotary hoe or harrow, which may also help activate the herbicide under dry conditions.

Many factors influence the activity and performance of soil-applied herbicides. Factors that should be considered are: rate too low for soil type, high weed pressure, weeds not listed on label, poor control in wheel tracks, cloddy soil, wet soil, amount of previous crop residue, dry weather, poor incorporation, improper setting of incorporation implement, herbicide resistant weeds, incorporation too shallow or deep, incorporation speed too slow, worn sweeps on cultivator, single pass instead of two pass incorporation, and second incorporation deeper than first. Consider these possibilities, before poor weed control is attributed only to the herbicide.

Buckle, Eptam, Far-Go, Ro-Neet, Sonalan, and Treflan* require incorporation. Eptam, Far-Go, and Ro-Neet must be incorporated immediately (within minutes) after application. Treflan
incorporation may be delayed up to 24 hours if applied to a cool, dry soil and if wind velocity is less than 10 mph. Sonalan incorporation may be delayed up to 48 hours. Prowl* is labeled only PPI in soybean, dry beans, and pulse crops and labeled PRE, not PPI, on corn. Dual*, Harness/Surpass*, IntRRo*, and Outlook* may be used PRE but shallow PPI improves weed control, particularly on fine textured soils. Incorporation of Dual*, Intrro*, and Nortron* may be delayed several days. Incorporation of Eradicane and Eptam can be delayed up to 4 hours when applied with liquid fertilizer and the same day when impregnated on dry bulk fertilizer. Ro-Neet can be incorporated up to 4 hours after application and up to 8 hours when impregnated on dry fertilizer.

Second tillage at right angles to the initial incorporation is needed if a disk or field cultivator is used. The second incorporation will incorporate any herbicide remaining on the soil surface and provide more uniform distribution in the soil, thereby improving weed control and reducing crop injury.

Soil-applied herbicides are adsorbed and inactivated by the clay component in soil but more by organic matter. Adjust herbicide rates for soil type and organic matter content. Most soil-applied herbicides require higher rates to be effective in high organic matter soils, but crop safety may be marginal on low organic matter soils. Some herbicides (linuron) give good weed control only when organic matter levels are low.

Far-Go, Treflan* and most POST herbicides are affected only slightly by organic matter levels. Organic matter levels should be determined on each field where organic-matter-sensitive herbicides are to be used. Organic matter levels change very slowly, and testing once every 5 years should be adequate.

POST APPLIED HERBICIDES

Weed control from POST herbicides is influenced by rate, weed species, weed size, and climatic conditions. Low labeled rates will be effective under favorable conditions and when weeds are small and actively growing. Use the highest labeled rates under adverse conditions and for well established weeds.

Sunlight inactivates some herbicides by the ultraviolet (UV) spectrum of light. Treflan* and Eptam degradation is minimal when incorporation is soon after application. “Dim” herbicides (Achieve, Select*, and Poast) are highly susceptible to UV light and will degrade rapidly if left in nonmetal spray tanks for an extended period of time or if applied during mid-day. To avoid
UV breakdown, apply soon after mixing and add an effective oil adjuvant which speeds absorption. Increased **light intensity** has been found to enhance the uptake and translocation of many herbicides and to accelerate the development of herbicide damage. In general, glyphosate treatment applied in the middle of the day or night period did not differ significantly from those made at the beginning of the day regime. In contrast, the performance of the foliage applied herbicide paraquat is markedly increased when several hours ‘light’ follow its application as in the mid-morning treatments i.e. mid morning applications are better than evening applications.

**Ideal temperatures** for applying most POST herbicides are between 65 and 85 °F. Speed of kill may be slow when temperatures remain below 60 °F. Some herbicides may injure crops if applied above 85 °F or below 40 °F. Avoid applying volatile herbicides under conditions where vapors and particle drift may injure susceptible crops, shelterbelt trees, or farmsteads.

Temperatures following herbicide application influence crop safety and weed control. Crops metabolize herbicides but metabolism slows during cool or cold conditions, which extends the amount of time required for plants to degrade herbicides. Rapid degradation under warm conditions allows plants to escape herbicide injury. Herbicides may be sprayed following cold night-time temperatures if day-time temperatures warm to at least 60 degrees.

**Temperature** influences plant growth and development. Respiration, transpiration and other metabolic processes such as membrane permeability, ion uptake and water uptake also are affected by temperature. Factors which contribute to efficacy of soil applied herbicides such as herbicide volatility, solubility and adsorption and desorption from soil have been influenced by temperature. Cucumber, oats and wheat were most susceptible to soil applied terbutryn with temperature of 22 to 29 °C which also favour rapid growth. Phytotoxicity of atrazine and several methyl-urea herbicides increased as temperature increased within the range of 10-30 °C. Similar temperature effects have been reported for EPTC and 2,4-D. It was reported that alachlor injured navy beans at 20 and 25, but not at 30 °C. Temperate between 20 and 30 °C did not affect the phytotoxicity of trifluralin. In general high temperature during or after herbicide application increased weed susceptibility, probably because of increased herbicide uptake and translocation.

Based on experiments in controlled environmental chambers it was revealed that Diclofop soil incorporated or surface applied, was more toxic to wild oat (**Avena fatua** L.) shoots at 10 and 17 °C than at 24 °C. Efficacy of diclofop was enhanced with soil incorporation. Diclofop toxicity to wild oat roots was not influenced by a change in temperature. EPTC (S-ethyl
dipropylthiocarbamate) stimulated sugar-beet (Beta vulgaris L. 'American Crystal Hybrid B') shoot dry weight production at 10 °C and caused dry weight reduction at 24 °C. Atrazine toxicity to barley and alachlor toxicity to oats (Avena sativa L. 'Chief') increased with increased temperature from 10 to 17 °C. Temperatures within the range of 10 to 24 °C did not affect trifluralin toxicity to barley or BAY-5653 [N-(2-benzothiazolyl)-N-methylurea] or chloamben toxicity to oats.

Some “Fop” ACCase herbicides are more effective during cold/cool temperatures and are much less effective when grass weeds are drought stressed. Other ACCase herbicides, such as Assure II*, Poast, and Select* control grasses best in warm weather when grasses are actively growing. ALS grass herbicides in wheat generally provide more consistent and greater grass control in warm, dry conditions compared with cool, wet conditions. Cool or cold conditions at or following application of ACCase herbicides may increase injury to wheat. Wild oat is a cool season grass but green and yellow foxtail are warm season grasses which may stop growing under cold conditions, resulting in poor control. Weeds are controlled most effectively when plants are actively growing.

Cold temperatures and freezing conditions following application of ALS herbicides, Buctril*, and Sencor* may increase crop injury with little effect on weed control. Delay applying fenoxaprop, ALS herbicides, and Sencor* until daytime temperatures exceed 60 °F and after active plant growth resumes.

Basagran*, Cobra, Flexstar, Ignite, paraquat*, Reflex, and Ultra Blazer are less likely to cause crop injury when cold temperatures follow application but less weed control may result. 2,4-D, MCPA, Banvel*, Starane*, Stinger*, and Glyphosate (resistant crops) have adequate crop safety and provide similar weed control across a wide range of temperatures, but weed death is slowed when cold temperatures follow application.

In relation to crop tolerance, Papalia and Blacklow observed that the wheat cultivar Sonora was more susceptible to chlorsulfuron when night soil temperature were 5 °C and 0 °C compared to 13 °C, and this may well be related to the temperature dependent rate of metabolism of the herbicide to non-phytotoxic metabolites.

A number of processes of importance to herbicide performance are affected by humidity including spray drop drying. A precise correlation between these and RH can only be expected at a given temperature, thus both these factors should be recorded in environmental studies. A fully
hydrated cuticle favours the uptake of foliage applied herbicides, particularly water soluble compounds such as glyphosate, which are believed to enter the plant via a hydrophilic pathway. Transpiration is increased under low humidity regimes, and if adequate water is available from the soil enhanced acropetal movement of foliage and soil applied herbicides will result. **Dew** may increase absorption and weed control by hydrating leaf cuticle but may reduce weed control if spray run-off occurs. **Rainfall** shortly after POST herbicide application reduces weed control because herbicide is washed off the leaves before absorption is complete (See the rainfall interval chart on the next page). The significance of rainfall within this period however, depends upon many factors: characteristics of the herbicide, its dose, formulation, period between spraying and the onset of rain, type and intensity of rain, temperature, humidity and other factors.

 Minimum Interval Between Application and Rain for Maximum POST Weed Control

<table>
<thead>
<tr>
<th>Herbcide</th>
<th>Time interval (hour)</th>
<th>Herbcide</th>
<th>Time interval (hour)</th>
<th>Herbcide</th>
<th>Time interval (hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accent</td>
<td>4-6</td>
<td>Fusilade DX</td>
<td>1</td>
<td>Realm Q</td>
<td>4</td>
</tr>
<tr>
<td>Aim</td>
<td>6-8</td>
<td>Fusion</td>
<td>1</td>
<td>Redeem</td>
<td>2</td>
</tr>
<tr>
<td>Ally/Escort</td>
<td>4</td>
<td>Goldsky</td>
<td>4</td>
<td>Reflex</td>
<td>1</td>
</tr>
<tr>
<td>Armazon/impact</td>
<td>1</td>
<td>Halex GT</td>
<td>4</td>
<td>Reglone</td>
<td>0.5</td>
</tr>
<tr>
<td>Assure II/Targa</td>
<td>1</td>
<td>Harmony</td>
<td>4</td>
<td>Remedy</td>
<td>6-8</td>
</tr>
<tr>
<td>Atrazine</td>
<td>4</td>
<td>Hornet</td>
<td>2</td>
<td>Require Q</td>
<td>4</td>
</tr>
<tr>
<td>Axial XL</td>
<td>0.5</td>
<td>Huskie</td>
<td>4</td>
<td>Resolve/Q</td>
<td>4</td>
</tr>
<tr>
<td>Axial Star</td>
<td>1</td>
<td>Huskie/Complete</td>
<td>4</td>
<td>Resource</td>
<td>1</td>
</tr>
<tr>
<td>Banvel/Clarity</td>
<td>6-8</td>
<td>Instigate</td>
<td>4</td>
<td>Rezult</td>
<td>4</td>
</tr>
<tr>
<td>Basagran</td>
<td>4-8</td>
<td>Laudis</td>
<td>4</td>
<td>Rimfire Max</td>
<td>4</td>
</tr>
<tr>
<td>Betamix</td>
<td>6</td>
<td>Liberty 280</td>
<td>4</td>
<td>Roundup</td>
<td>6-12</td>
</tr>
<tr>
<td>Bronate</td>
<td>1</td>
<td>Lumax</td>
<td>4</td>
<td>Roundup</td>
<td>6-12</td>
</tr>
<tr>
<td>Herbicide</td>
<td>Time interval (hour)</td>
<td>Herbicide</td>
<td>Time interval (hour)</td>
<td>Herbicide</td>
<td>Time interval (hour)</td>
</tr>
<tr>
<td>--------------------</td>
<td>---------------------</td>
<td>--------------------</td>
<td>---------------------</td>
<td>--------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Partial adjuvant)</td>
<td></td>
</tr>
<tr>
<td>Buctril</td>
<td>1</td>
<td>Manvel</td>
<td>1</td>
<td>Roundup (No adjuvant)</td>
<td>6-12</td>
</tr>
<tr>
<td>Cadet</td>
<td>1</td>
<td>Maverick</td>
<td>4</td>
<td>Select/Max</td>
<td>1</td>
</tr>
<tr>
<td>Callisto/GT</td>
<td>1</td>
<td>MCPA amine</td>
<td>4-6</td>
<td>Sharpen</td>
<td>1</td>
</tr>
<tr>
<td>Capreno</td>
<td>1</td>
<td>MCPA ester</td>
<td>1</td>
<td>Spartan Charge</td>
<td>6-8</td>
</tr>
<tr>
<td>Cimarron X-tra</td>
<td>4</td>
<td>Milestone</td>
<td>4</td>
<td>Starane/Flex</td>
<td>4</td>
</tr>
<tr>
<td>ClearMax</td>
<td>1</td>
<td>Olympus</td>
<td>4</td>
<td>Starane NXT</td>
<td>1</td>
</tr>
<tr>
<td>Cobra</td>
<td>0.5</td>
<td>Orion</td>
<td>4</td>
<td>Status</td>
<td>4</td>
</tr>
<tr>
<td>Curtail/M</td>
<td>6-8</td>
<td>Osprey</td>
<td>4</td>
<td>Steadfast</td>
<td>4</td>
</tr>
<tr>
<td>Defol 750</td>
<td>24</td>
<td>Paramount</td>
<td>6</td>
<td>Stinger</td>
<td>6-8</td>
</tr>
<tr>
<td>Diquat</td>
<td>0.5</td>
<td>Paraquat</td>
<td>0.5</td>
<td>SU herbicides</td>
<td>4</td>
</tr>
<tr>
<td>Discover NG</td>
<td>0.5</td>
<td>Permit</td>
<td>4</td>
<td>Supremacy</td>
<td>2</td>
</tr>
<tr>
<td>Express</td>
<td>4</td>
<td>Perspective</td>
<td>6-8</td>
<td>Tordon 22K</td>
<td>6-8</td>
</tr>
<tr>
<td>Everest 2.0</td>
<td>1</td>
<td>Plateau</td>
<td>1</td>
<td>Ultra Blazer</td>
<td>4</td>
</tr>
<tr>
<td>Extreme</td>
<td>1</td>
<td>Poast</td>
<td>1</td>
<td>UpBeet</td>
<td>6</td>
</tr>
<tr>
<td>Fenoxaprop</td>
<td>1</td>
<td>PowerFlax</td>
<td>4</td>
<td>Weedmaster</td>
<td>6-8</td>
</tr>
<tr>
<td>First Rate</td>
<td>2</td>
<td>Pulsar</td>
<td>4</td>
<td>WideMatch</td>
<td>6</td>
</tr>
<tr>
<td>Flexstar</td>
<td>1</td>
<td>Pursuit</td>
<td>1</td>
<td>Wolverine</td>
<td>1</td>
</tr>
<tr>
<td>Flexstar GT 3.5</td>
<td>6-12</td>
<td>Raptor</td>
<td>1</td>
<td>2,4-D amine</td>
<td>4-8</td>
</tr>
<tr>
<td>Foxfire</td>
<td>1</td>
<td>Rase</td>
<td>1</td>
<td>2,4-D ester</td>
<td>1</td>
</tr>
</tbody>
</table>
Minimize the risk of phytotoxic effects by:

- reading and following label directions, especially the correct rates and timing, and being aware of potential weather effects,
- avoiding application of pesticides when drift is likely to happen,
- waiting for the correct planting times,
- if unsure, conduct a simple field bioassay by treating only a few plants, before the treating whole block to check for phytotoxic effects, especially when growing new cultivars.

Information on the influence of environmental factors on herbicide performance before, during and after application of the spray may assist in the choice of spray periods that not only favour spraying but will also enhance herbicide activity. When the target weed has grown under conditions conductive to herbicide action and the weather forecast is favourable, a reduced dose of active ingredient be used. If weather conditions are sub-optimum the EHPP may help in the choice of an adjuvant to overcome adverse environmental conditions. For example weed has developed under cold or water stress conditions, addition of surfactant or appropriate adjuvant oil may enhance retention and penetration. If rain is forecast, a polymer adjuvant may improve adhesion and resistance to wash-off. When environmental conditions have predisposed the weeds to herbicide tolerance, synergists may be employed. Thus tridiphane reduced the metabolism and increases the phytotoxicity of atrazine in target weeds, but not maize. To conclude, the most effective use of EHPPs requires access to weather records, reliable forecasts and for greatest precision ‘on farm’ weather monitoring.

**Fate of Herbicide in Soil**

Herbicides are either applied directly to the soil (pre-emergence) or on to the foliage of weeds and crops (post-emergence). A part of the post-emergence herbicide also reaches the soil. Upon contact with the soil, herbicides are subjected to various processes.

The various processes and reactions that take place between herbicides and soil are important because they can affect the activity and behavior of the herbicide. It is important to understand herbicide behavior in the soil because it:
1. determines success or failure in weed control
2. determines whether or not crop injury occurs
3. affects persistence of the herbicide, which in turn determines the length of weed control and whether or not there is carryover to the following crop, and
4. ultimately determine the environmental fate of the herbicide.

Soil has no impact on efficacy of non-residual postemergence herbicides. With residual postemergence herbicides [Classic (chlorimuron) and Scepter (imazaquin) applied postemergence to sicklepod], the soil may affect efficacy.

**Environmental fate:** It refers to different interacting processes that lead to removal of a herbicide from its original site of application. There are two general categories of processes affecting environmental fate (Fig 1).

<table>
<thead>
<tr>
<th>Transport processes</th>
<th>Adsorption, Leaching, Volatilization, Plant-uptake and Runoff</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degradation processes</td>
<td>Microbial, Chemical and Photo-degradation</td>
</tr>
</tbody>
</table>

**A. ADSORPTION:** Binding of herbicide to soil colloids (the clay and organic matter fraction of the soil). The extent of adsorption determines how much herbicide is available to plants (available to control weeds or injure crops). Adsorption also affects other environmental processes, such as leaching, volatilization, and microbial degradation.

1. **Factors affecting herbicide adsorption**
   a. **Soil organic matter content**
      i. Soil organic matter (humic matter) has a high capacity to adsorb herbicides, and it is the single greatest factor affecting herbicide adsorption. Herbicde adsorption increases as the organic
matter content increases. Application rate of most soil-applied herbicides increases, as the organic matter content increases. Organic matter (humic matter) has many negatively charged sites that can attract positively charged herbicides. That is referred to as ionic bonding. Organic matter (humic matter) also has many organophillic, non-charged sites on the surface that lead to non-ionic bonding of herbicides.

b. Soil clay type and content

Clay carries a negative charge, which contributes to the soils CEC. Herbicides with a positive charge are ionically bound to clay particles. 2:1 type clays (such as montmorillonite), which have expanding lattices, have more surface area and more negatively charged sites than 1:1 type, or non-expanding, clays (such as kaolinite). Hence, soils with 2:1 clays will adsorb more herbicide than soils with 1:1 type clays. Herbicide adsorption and therefore application rates of most soil-applied herbicides increases with increase in the clay content of a soil.

c. Soil moisture

Herbicides are more tightly bound to drier soil due to less competition with water for binding sites under dry conditions. Plants absorb herbicide that is dissolved in the aqueous phase, or the soil solution. With good soil moisture, some of the binding sites on colloids are occupied by water and more of the herbicide is in the soil solution. As the soil becomes drier, there is less water present to compete with the herbicide for binding sites on the colloid, hence more of the binding sites are occupied by herbicide. More herbicide is bound to the drier soil, meaning there is less herbicide in the aqueous phase available for uptake by plants. Hence, the herbicide is less effective on weeds under dry conditions.

d. Herbicide chemistry and soil pH

The chemical properties of a given herbicide have a major influence on the amount of adsorption. Simply stated, some herbicides are naturally bound to the soil more tightly than other herbicides. And, because of the chemical characteristics of certain herbicides, soil pH can affect the amount of adsorption. The term sorption coefficient, or KOC, is used to describe the tendency for herbicides to adsorb to soil. Partition coefficient, Kd, is the ratio of herbicide bound to soil particles compared to the amount left in the soil solution.
Advances in Weed Management

\[ \text{Kd} = \frac{\text{amount adsorbed by soil}}{\text{amount in soil solution}} \]

\[ \text{KOC} = \frac{\text{Kd}}{\text{organic carbon content}} \]

Sorption coefficient, KOC, measures the tendency for adsorption adjusted for organic carbon content. The smaller the KOC value, the smaller the amount of herbicide bound to the soil. Paraquat is very tightly and irreversibly bound to soil. Alachlor and imazaquin are weakly bound to soil.

The impact of soil pH is herbicide-specific; adsorption of some herbicides is affected by soil pH, whereas pH has no effect on adsorption of other herbicides.

Nonionizable herbicides: These remain uncharged regardless of soil pH. Hence, soil pH does not affect adsorption of such herbicides. An example is trifluralin (Treflan).

Cationic herbicides: Paraquat exists only in the cationic form (positively charged). It is strongly (irreversibly) bound to negatively charged clay particles.

Basic herbicides: Basic herbicides can exist in a neutral form or an ionized form, depending upon soil pH. Basic herbicides (those which can accept a proton) become positively charged as the pH is decreased. The cationic form of these herbicides, which exists at lower pH values, is strongly adsorbed to negatively charged clay. Atrazine is an example of a basic herbicide.

Acidic herbicides: Acidic herbicides also can exist in a neutral form or an ionized form, depending upon soil pH. Acidic herbicides (those which can give up a proton) are neutral in charge at low pH and become negatively charged as the pH is increases. Soil pH has little effect on adsorption of acidic herbicides because negatively charged herbicide molecules are not attracted to negatively charged sites on clay. 2,4-D is an example of an acidic herbicide. In some cases, the charge on a molecule can affect its solubility. Sulfentrazone, the active ingredient in Spartan herbicide, behaves as an acidic herbicide. Although soil pH has little effect on adsorption, pH does affect the solubility of sulfentrazone. The herbicide is more water soluble at lower pH values, and plants may take up more under lower pH conditions.

<table>
<thead>
<tr>
<th>Herbicide</th>
<th>KOC (mL/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paraquat</td>
<td>1,000000</td>
</tr>
<tr>
<td>Glyphosate</td>
<td>24,000</td>
</tr>
<tr>
<td>Trifluralin</td>
<td>7,000</td>
</tr>
<tr>
<td>Alachlor</td>
<td>124</td>
</tr>
<tr>
<td>Imazaquin</td>
<td>20</td>
</tr>
</tbody>
</table>
B. LEACHING:

Leaching is the downward movement of a substance (herbicide) by water in the soil.

1. Importance of leaching:
   a. Activation of preemergence herbicides

   Soil-applied herbicides are typically placed on the soil surface. To be effective, the herbicides must be in the weed seed germination zone. Herbicides can get into this zone by either being mechanically mixed into the soil (incorporation) or they can be leached into the zone by rainfall or irrigation water. The amount of rainfall or irrigation necessary to activate a herbicide depends upon its water solubility and the degree to which it is adsorbed to soil.

   
   Lower solubility = greater amount of water to activate.

   Greater adsorption = greater amount of water to activate.

   b. Leaching can also explain selectivity. Crop tolerance of a herbicide may be very good as long as the herbicide is not deep enough to be in the crops root zone. However, heavy leaching may move the herbicide down into the crop root zone and cause injury or death to the crop. For example metribuzin (Sencor) tolerance on soybeans is good as long as the herbicide remains above the root zone, but soybeans can be severely injured if the herbicide is leached into the root zone. This is sometimes referred to as ‘profile tolerance’, meaning the crop tolerance is dependent upon location of the herbicide in the soil profile.

   c. Herbicides that are not bound to the soil especially very light sandy soils may leach into ground water. It may also occur on lighter soils where the water table is close to the surface.
2. Factors affecting herbicide leaching:

a. Chemical properties of the herbicide:

i. Water solubility: Assuming that two herbicides are equally bound to soil, the herbicide with the greater water solubility is more prone to leaching.

ii. Adsorption: Leaching is inversely related to adsorption. Greater adsorption = less leaching.

iii. Water solubility and the tendency for adsorption together form the pesticide leaching potential (PLP). Herbicides (and other pesticides) can be ranked according to their potential to leach based upon chemical properties.

<table>
<thead>
<tr>
<th>Herbicide</th>
<th>PLP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pendimethalin (Prowl)</td>
<td>very low</td>
</tr>
<tr>
<td>Imazethapyr (Pursuit)</td>
<td>low</td>
</tr>
<tr>
<td>Atrazine</td>
<td>medium</td>
</tr>
<tr>
<td>Prometone (Pramitol)</td>
<td>very high</td>
</tr>
</tbody>
</table>

From: NC Agricultural Chemicals Manual, Chapter 1.

b. Soil characteristics:

The primary soil characteristics affecting herbicide leaching are soil texture and organic matter content. The potential for leaching is inversely related to adsorption. Greater adsorption = less leaching. Soils can be categorized by their potential to allow pesticide leaching (soil leaching potential, or SLP). Well-drained, sandy, low organic matter soils have the greatest SLP. The SLP for selected soils is listed below.

<table>
<thead>
<tr>
<th>Soil Series</th>
<th>SLP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cape Fear</td>
<td>very low</td>
</tr>
<tr>
<td>Alamance</td>
<td>medium</td>
</tr>
<tr>
<td>Tarboro</td>
<td>very high</td>
</tr>
</tbody>
</table>

From: NC Agricultural Chemicals Manual, Chapter 1.

The PLP and SLP together form the ground water contamination potential (GWCP).

c. Volume of water flow

Leaching is affected by the volume of water flow through the soil profile. This is determined by the water infiltration rate of the soil and by amount and intensity of rainfall.

C. RUNOFF AND EROSION:

Runoff and erosion are transport processes affecting environmental fate of a herbicide, usually in a negative manner. A herbicide may leave the application site in surface water running off the site. The herbicide may be dissolved or suspended in the runoff water or it may be adsorbed to
soil carried by runoff water. Herbicide runoff is a serious concern because runoff water ultimately makes it way into rivers and surface reservoirs used for drinking water.

1. Factors affecting herbicide loss via runoff
   a. Herbicide application rate: Higher application rates mean more herbicide is likely to be transported in runoff.
   b. Time of occurrence, intensity, and amount of rainfall: Highest concentrations of herbicides occur in the first significant runoff event after application. Concentrations will decrease with successive runoff events as the herbicide is leached into the soil or dissipates by other mechanisms. Higher amounts of rainfall and greater intensity of the rainfall lead to greater runoff from the target site and, hence, greater transport of herbicide.
   c. Soil texture and slope: Soil texture affects water infiltration rate, thereby affecting the amount of runoff. There tends to be greater runoff from finer-textured soils. Organic matter affects infiltration rate; there tends to be less water runoff as organic matter content increases. Organic matter also adsorbs herbicide, thus reducing amount of dissolved herbicide leaving the field.
   d. Chemical properties of the herbicide: Herbicides with higher water solubility and weak adsorption to colloids are more likely to be transported in runoff water as dissolved chemicals; herbicides with lower water solubility and stronger adsorption to colloids are more likely to be transported on soil particles suspended in runoff water. However, herbicides with higher water solubility and weak adsorption are more likely to leach, which may reduce transport in runoff.

2. Best management practices (BMPs) to reduce herbicide runoff:
Practices that reduce soil erosion will reduce runoff of herbicides adsorbed to suspended soil particles. However, erosion control per se will have a limited effect on transport of compounds dissolved in water unless the erosion control also reduces the volume of runoff water. Use of vegetative buffer strips around application areas can help intercept herbicide runoff loses through sediment deposition.
   Examples of BMPs to reduce runoff:
   a. No-till planting
   b. Surface residue
c. Vegetative buffer strips
d. Others, such as containment ponds around nurseries
e. Timing of herbicide application; where practical, avoid spraying ahead of anticipated heavy rain

D. VOLATILIZATION:

The change from a solid or liquid phase to a gaseous phase and subsequent dissipation to the atmosphere

1. Factors affecting extent of loss by volatilization

a. Vapor pressure of the herbicide: Higher vapor pressures mean greater potential for volatilization. Butylate has a very high vapor pressure and is readily subject to loss by volatilization. Atrazine has a low vapor pressure, and volatilization losses are minimal.

b. Soil moisture content: volatilization losses are greater from a wet soil as compared with a dry soil. Due to effect of soil moisture on herbicide adsorption. Herbicides bound tighter to dry soil, hence less volatilization.

c. Soil temperature: volatilization losses increase as soil temperature increases.

d. Herbicide adsorption: Greater adsorption = less volatilization.

e. Herbicide application method: Primarily referring to preplant incorporated vs preemergence. Incorporation greatly reduces volatilization losses. One of the primary reasons to incorporate certain soil-applied herbicides is to reduce volatilization losses.

2. Importance of volatilization

a. Excessive volatilization losses can adversely impact weed control.
b. Volatilization can cause injury to off-target vegetation.

E. PLANT UPTAKE:

The amount of herbicide absorbed by plants and subsequently metabolized (degraded to inactive form) is usually a minuscule amount of what was applied. Hence, plant uptake typically has little

<table>
<thead>
<tr>
<th>Herbicide</th>
<th>VP (mm Hg @ 25C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>butylate</td>
<td>$1.3 \times 10^{-2}$ or $130,000 \times 10^{-7}$</td>
</tr>
<tr>
<td>trifluralin</td>
<td>$1.1 \times 10^{-7}$ or $1,100 \times 10^{-13}$</td>
</tr>
<tr>
<td>pendimethalin</td>
<td>$9.4 \times 10^{-6}$ or $94 \times 10^{-12}$</td>
</tr>
<tr>
<td>atrazine</td>
<td>$2.9 \times 10^{-7}$ or $2.9 \times 10^{-13}$</td>
</tr>
</tbody>
</table>
effect on environmental fate of herbicides. One exception to that is corn and atrazine. Corn can absorb and degrade significant amounts of atrazine.

**F. MICROBIAL DEGRADATION:**

Microbial degradation is a major means of dissipation for many herbicides. Herbicides can serve as a substrate for soil microorganisms (basically a food source). Various species of bacteria are the primary soil microorganisms degrading herbicides.

Microbial degradation results in a change from an active herbicide molecule to one with no herbicidal activity. See the example below for 2,4-D.

Aerobic degradation (by microorganisms requiring oxygen) is by far the most common. However, certain herbicides are readily subject to degradation by anaerobic microorganisms, and they can be rapidly lost in saturated soils.

The rate of microbial degradation is highly correlated with the population and activity of microorganisms. Condition that favor high microbial activity lead to more rapid microbial degradation of herbicides.

The population and activity of soil microorganisms are affected by:

1. Soil temperature: 80 to 90°F is generally best. Microbial activity is greatly diminished in cool temperatures.
2. Soil moisture: 50 to 100% field capacity is best for aerobic degradation. Dry soil retards microbial degradation.
3. Soil aeration: poor aeration (and flooding) retard aerobic degradation.

**G. CHEMICAL DEGRADATION (ABIOTIC OR NON-BIOLOGICAL):**

This is basically chemical reactions, such as hydrolysis (decomposition involving the splitting of a bond and
the addition of the elements of water), reduction (addition of hydrogen), and photolysis (decomposition by sunlight). Rates of chemical degradation are affected by temperature (greater at higher temperature) and, for some herbicides, the rate is greatly affected by soil pH. An example of chemical degradation of a triazine is shown below.

**H. PHOTODEGRADATION** (photolysis):

Some soil-applied dinitroanilines are subject to photodegradation (A chemical degradation by sunlight). This can be avoided by mechanically incorporating the herbicide into the soil. Sethoxydim, a postemergence herbicide, is also highly subject to photodegradation. Sethoxydim still works (avoids photodegradation) by being very rapidly absorbed by plants.

**III. HERBICIDE PERSISTENCE:**

Persistence refers to the length of time a herbicide remains phytotoxic (or herbicidally active) in the soil. Persistence is important for the following reasons:

A. Determines length of weed control
B. Determines toxicity to following crop (carryover)
C. Possibly illegal residues in succeeding crop
D. Impacts environmental fate

Ideally, a herbicide should persist long enough beyond the critical period of interference but then dissipate rapidly thereafter to avoid damage to succeeding crops. With a soil-applied herbicide, there is a minimum concentration necessary to control weeds. Recommended application rates typically provide more than the minimal amount so that growers will get weed control over some length of time before the herbicide dissipates to an ineffective concentration. There is also a maximum concentration that is safe to any given rotational crop. Herbicide persistence is typically expressed as half-life ($T_{1/2}$), meaning the time it takes 50% of the herbicide to breakdown to an inactive form. Half lives vary greatly, depending upon the conditions under which the determination was made. Soil properties (texture and organic matter, pH, and microbial populations), herbicide properties (water solubility, vapor pressure, binding characteristics, susceptibility to chemical and microbial degradation), and climatic conditions (temperature and moisture) all interact to determine persistence.
Residue Management of Herbicides

When applied at recommended rates most herbicides breakdown within a few days or weeks after application and impose no restrictions on cropping options the next year. Some herbicides however do not degrade quickly and can persist in the soil for weeks, months or years following application. The use of residual herbicides can be beneficial as the residues prevent growth of sensitive weed species throughout the season. These residues however can restrict the crops that can be grown in rotation. Understanding the factors that influence carryover and breakdown are key to assessing risk and the appropriate follow crop. If herbicide carryover is suspected, knowing the appropriate sampling procedures and soil tests to obtain will assist in determining and minimizing herbicide carryover.

Herbicide half-life

How long will a herbicide persist in the environment? That depends on a lot of factors, but there is a gauge by which we can predict herbicide persistence. Herbicide half-life is a measure of how long it takes for 50% of a chemical to degrade (Table 1). For example, oxadiazon has a half-life of 60 days. So 60 days after application, it will have degraded to ½ of the amount applied. After 120 days, the concentration will have decreased by 50% again so that only ¼ of the applied amount remains. Assuming a 240 day growing season with conditions optimum for herbicide breakdown, only 1/16 of the applied product will remain at the end of the season.

Herbicide half life values presented in Table are derived from many sources. These values were determined using various methods, under different environmental conditions, in different parts of the world. Comparing one herbicide to another is almost like comparing apples to oranges, but it is the best gauge we have right now. Use half-lives as a guide in selecting herbicides, not as absolute values for predicting herbicide degradation.
Rotational Restrictions (wait-periods) for herbicides applied prior to fall seeding of potential crops in West Virginia.

<table>
<thead>
<tr>
<th>Herbicide</th>
<th>Winter Wheat</th>
<th>Rye</th>
<th>Barley</th>
<th>Alfalfa</th>
</tr>
</thead>
<tbody>
<tr>
<td>2,4-D</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Atrazine</td>
<td>24</td>
<td>24</td>
<td>24</td>
<td>24</td>
</tr>
<tr>
<td>Pendimethalin</td>
<td>4</td>
<td>9</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Metolachlor</td>
<td>4.5</td>
<td>4.5</td>
<td>4.5</td>
<td>4</td>
</tr>
<tr>
<td>Dicamba</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Imazaquin</td>
<td>3</td>
<td>18</td>
<td>11</td>
<td>18</td>
</tr>
<tr>
<td>Metribuzin</td>
<td>8</td>
<td>12</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Imazathapyr</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

Factors Affecting Herbicide Carryover

Herbicide Characteristics

**Adsorption:** All herbicides bind to the soil particles and organic matter (OM) to some degree. The strength and extent of the binding will affect the persistence and carryover of the herbicide in the soil. Soil factors such as moisture, pH and OM significantly affect the adsorption of herbicides in the soil. Binding of the herbicide to soil particles increases as moisture content decreases and is particularly important for herbicides that are usually weakly adsorbed. Under dry soil conditions the herbicide is bond to the soil and unavailable for breakdown. When soil moisture is adequate the herbicide becomes available for breakdown.
**Water Solubility:** How easily the herbicide dissolves in the soil water or its water solubility, will determine how readily the herbicide is available for breakdown or leaching. The more water soluble the herbicide, the more readily it can breakdown or leach in the soil. While high solubility in water can reduce the potential for herbicide carryover, other factors such as biological activity and persistence influence the re-crop restrictions.

**Vollatility:** Some herbicides are very volatile and evaporate readily. Phenoxy herbicides such as 2,4-D are relatively volatile; other herbicides that are volatile are triallate (Avadex) and trifuralin (Treflan etc.). The more volatile the herbicide, the more quickly it dissipates from the soil. As a result of this characteristic, many of the soil-applied herbicides are incorporated into the soil soon after application to avoid losses due to volatilization.

**Herbicide Degradation**

**Microbial Decomposition:** Soil bacteria, algae and/or fungi metabolize some herbicides. Warm, moist, fertile soil favors the growth of the soil microorganisms and as a result stimulates breakdown of certain herbicides.

**Chemical Degradation:** Some herbicides may react with water or other chemicals in the soil, changing the nature of the molecules responsible for the herbicidal activity. For example, the sulfonylurea (SU) chemically reacts with water in a process called hydrolysis. Once the SU is in contact with water, the chemical breakdown is initiated, and the herbicide is no longer biologically active.

**Photodecomposition:** Some herbicide will degrade when exposed to sunlight. These products when exposed to ultra-violet light on soil or leaf surface, they breakdown relatively quickly.
Herbicides such as trifluralin, ethalfluralin and the cyclohexanones (Poast, Achieve, Select) can degrade in sunlight. Specific management strategies such as soil incorporation and the use of effective surfactants minimize the impact of photo-degradation.

**Plant Uptake:** Once plants absorb the herbicide, it is metabolized. This effectively removes residues from the soil. When plant stand densities are low, removal of herbicide residues is also low.

**Soil Factors:** Several soil factors are important in determining the persistence of a herbicide. The extensive variability of such factors as pH, OM and even texture with a field or field-to-field challenge the decision-making process regarding crop selection.

**Soil pH:** The pH of the soil solution is a critical factor in the breakdown of certain herbicides. Soil pH may cause herbicide degradation directly by affecting the stability of the herbicide or indirectly by its effect on the soil microbes. The sulfonylureas (SU) herbicides breakdown more quickly in acid soils, hence persisting longer in high pH soils > pH 7.0. The Imadazolionones breakdown more readily in alkaline soils and therefore persistence is increased in low pH < pH 7.0 soils. In addition, OM plays a key role in longevity of the residues.

**Organic Matter:** Organic matter (OM) can absorb large amounts of herbicides, so the less the OM, the more biologically active the herbicide residue. The organic matter binds the herbicide and releases them more slowly. In high organic soils herbicides persist longer. In addition, soil rich in OM support microorganism, which play a critical role in the degradation of most herbicides.

**Soil Texture:** The relative percentage of sand, silt and clay in a soil determines its texture. Clay particles provide extensive amounts of surface area that can adsorb significant amounts of herbicide. So in clay soils, herbicide residues tend to be less severe. Since water tends not to move as fast or as deep in clay soils, the potential for herbicide leaching is also diminished. In sandy soils, herbicide leaching is more significant since the amount of herbicide bond to the soil is less.

**Soil Moisture:** Soil moisture is an extremely important factor in determining the rate of breakdown of a herbicide in the soil. The higher the soil moisture levels the higher the rates of leaching, volatilization and microbial/ chemical degradation. The drier the growing season, the
greater will be the potential for herbicide carryover to the following season. The timing of available soil moisture is critical. The rainfall that occurs after the herbicide application can profoundly affect persistence. The less rainfall after spraying, the more likely there will be higher than normal carryover increasing the risk of damage to sensitive crops.

**Soil Temperature:** Herbicide residue will disappear more rapidly when the soil is warm >15 °C. Early freeze up in the fall and late spring thaw do not allow sufficient time for herbicide breakdown and can result in increased herbicide carryover. Warm, moist soils favor herbicide breakdown.

**Management Factors**

**Application Rate:** The higher the initial application rate, the longer it will take for the herbicide residue to dissipate.

**Time of Application:** The greater the amount of time between the application and the seeding of a sensitive crop, the less likely injury will occur. Fall applications generally involve less risk of rotational crop damage than do spring applications. For example 2,4-D applied in the spring results in greater damage to canola, field peas and lentils than fall applications.

**Use Patterns:** Consecutive application of the same or related herbicides can increase the risk to rotational crops. The use of residual herbicides from the same Herbicide Group can result in an additive or cumulative effect, potentially limiting crop choices the following year. Keeping good field records and avoid back-to-back use, this management strategy will assist in minimizing re-crop concerns.

**Uniformity of application/incorporation:** Residual herbicides that have been applied and incorporated at recommended rates should not be a problem the following season. Non-uniform application or incorporation can cause hot spots where higher than recommended concentrations of herbicide occur in patches. Damage usually occurs on headlands and corners or in strip throughout the field. Double spraying or not shutting off the sprayer on turns, results in these hot spots.

**Tillage System:** In direct-seeded fields where minimal disturbance is done, the herbicide residues remain in a concentrated band on the soil surface. In a conventional tillage system,
tillage mixes the herbicide residues throughout the soil profile, accelerating rates of microbial degradation and diluting the herbicide residues.

**Fertility and Plant Growth:** Plants absorb herbicides from the soil reducing the concentration in the soil. A good crop stand will absorb the herbicide and in most cases breakdown the herbicide residue (an exception is clopyralid). Plant growth and herbicide decomposition by microbes are influenced by soil fertility. If the soil is low in fertility, the growth of microorganisms and the degradation of herbicides is slower.

**Avoiding or Minimizing Herbicide Carry-over Effects**

It is important to plan a weed control strategy carefully so that herbicide carry-over can be avoided. Planning a weed control program should be based on the weed problem, herbicide options, including formulation and persistence, soil characteristics, weather conditions and crop rotation. Remember that this plan will have an effect on cropping options in the year(s) ahead.

**Always leave an untreated check area in the field for future comparison** - Good record keeping is essential to avoid crop losses caused by herbicide carry-over. A weed control plan to minimize or eliminate herbicide carry-over should include:

**Integrated weed management** – Use various management techniques such as seeding date, crop selection and fertilizer placement to promote a vigorous competitive crop that has an advantage over weeds and helps to minimize the level of carry-over the following year.

**Herbicide rotation with crop rotation** – This is important in reducing the need to apply herbicides that may carry-over in the soil in successive years. For example, it is not advisable to apply Sundance or Everest to soil treated with Odyssey the previous year.

**Selection of herbicides with minimum carry-over potential** – Choosing a herbicide with little or no carry-over given your local soil and weather conditions will eliminate future crop injury problems.

**Applying minimum rates of herbicides** – Theoretically, the rate of herbicide applied should never be more than the amount required to achieve an acceptable level of weed control (this may vary with soil type and moisture conditions). This practice will reduce the potential for carry-
over. For example, the amount of trifluralin required on a low organic matter sandy soil is much less than required on higher organic matter clay soils.

**Time of application** – Research has shown that early removal when the weeds are small reduces competition and improves crop yield. Early season application also assists in reducing the carry-over potential to succeeding crops. The longer the herbicide is exposed to breakdown factors such as, moisture and temperature the lower the risk of carry-over.

**Accurate application** – Careful and accurate application of herbicide is essential to reduce the potential for carry-over. Always read the label before using, follow all the instructions and precautions, mix the correct amount of active ingredient and ensure the sprayer is properly calibrated and applying uniformly across the boom width. Avoid overlapping on the spray run as this doubles the application rate. And, avoid sharp turns with the sprayer operating as the application rate increases several fold from the inside boom.

**Tank-mixture opportunities** – Combining a non-residual herbicide with the lowest recommended rate of a residual herbicide in a tank-mixture can reduce carry-over potential. It is important to use only registered tank-mixes and to apply according to the application instructions on the product labels.

**Grow a tolerant crop** – When herbicide residue is detected or suspected a tolerant crop should be grown. A tolerant crop has the ability to either store or degrade the residue to non-toxic compounds. For example, when carry-over Pursuit is suspected, crops such as canola (non-Clearfield) and flax should be avoided.

**Soil additives** - Adsorption of herbicide residue can be increased by the addition of adsorbent material such as activated charcoal. The use of activated charcoal on a large scale is not economic. However, on small areas as a spot treatment for chemical spills or where high value crops are produced its use might be economically justified.

**Application of fertilizer** – The addition of fertilizer enhances the growth of tolerant plants, which increases the uptake of herbicide from the soil. It also promotes the growth of microflora, increasing biological breakdown of herbicide in the soil. For example, addition of phosphate enhances the microbial breakdown of the phenoxy herbicides 2,4-D and MCPA.
Adjuvants

Adjuvant is defined as any substance either in a herbicide formulation or added to the spray tank, that modifies herbicidal activity or application characteristics. Adjuvants are added with the following objectives:

1. Improve herbicide selectivity to the non target plants,
2. Improve herbicide safety to the users,
3. To improve its shelf life, and
4. To improve toxicity on target plants (weeds).

Some herbicides adjuvants can affect selectivity, resulting in crop damage. In emulsions and suspensions of flowable formulations, water has a tendency to be repelled by the other liquid, or by the solid in the suspension. In order to overcome the tension between the two surfaces, a substance is needed which has an affinity for both water and the other material. Such a substance will have a molecule that orients itself between the two surfaces so they are bounded in a more intimate contact. Such substances are known as surfactants.

Surfactants (surface active agents) include wetting agents, vegetable and mineral oils, stickers, emulsifiers and spreading agents. One or more of these type of surfactants are included in all herbicide formulations, or added just before application.

Water is not compatible with many of the chemicals used as herbicides. By adding a surfactant, a particular herbicide can be mixed with water in order to form an emulsion which can be readily applied through a boom spray. Water is also repelled by the wax like cuticle found on plant surfaces. By adding a surfactant, in the form of a wetting agent, the effectiveness of a herbicide may be enhanced.

There are different types of adjuvants with different types of surfactants (Gupta, 1998).

Surfactants

The leaves of plants with waxy, hairy or thick cutin surfaces are not wetted properly in the absence of surfactants. The surfactants possess surface modifying properties. They aid (a) in
wetting the waxy leaf surfaces with the aqueous herbicide sprays (wetting agents) (b) in spreading the hydrophilic herbicides uniformly over the foliage (spreaders) and (c) in the penetration of the herbicide into the target leaves and stems (penetrants). Wetting and spreading are aspects of the same phenomenon and they occur simultaneously. However, certain surfactants have exhibited innate phytotoxicity and growth stimulating effects depending upon the concentration used (Foy and Smith, 1965).

Surfactants can be classified as non-ionic or ionic depending on their ionization or dissociation in water. Non-ionic surfactants have no particle charge, whereas ionic surfactants have either a positive or negative charge. Non-ionic surfactants are classed as non-electrolytes and are usually chemically inactive. Because of this, they can be mixed with most herbicides without reacting. Unless otherwise specified, non-ionic surfactants should be used in preference to others.

Examples of surfactants

**Non-ionic**

S-145 or Tween 20 = polyoxyethylene sorbitan monolaurate  
Surfactant WK = dodecyl ether of polyoxyethylene glycol

**Anionic**

Santomerse = Dodecylbenzene sodium sulphonate  
Vatsol-OT = Sodiumdioctyl sulphosuccinate  
SDS = Sodium dodecylsulphate

**Cationic**

Quatenary-0 = Alkylimidazolinium chloride  
Aliquat-4 = Lauryltrimethylammonium chloride  
CTAB = Hexadecyltrimethyl ammonium

**Wetting agents**

Surfactants or wetting agents (wetters) reduce the surface tension of water, and allow spray droplets to spread on the leaves, adhere better and achieve better penetration of the herbicide. The degree of effectiveness of a wetting agent can be measured by the reduction in the surface
tension of a liquid, or the increase in spread of a liquid over a surface area. This type of information in general is not available on product labels. Water droplets containing a wetting agent spread in a thin layer over a waxed surface. Without a wetting agent the water will still as a droplet, with a small area of contact with a waxy surface. Penetration of herbicides from these droplets into the plant is poor and the droplets are more likely to fall to the ground. The effectiveness of herbicides is generally increased by the addition of wetting agents. However, if high rates of water and high volume of water are applied, they may result in excessive run off. Increased herbicide activity through the use of wetting agents can reduce herbicide selectivity, resulting in crop damage.

**Stabilizing agents**

The stabilizing agents include emulsifiers and dispersants.

Emulsifiers (or emulsifying agents): Emulsifiers are chemicals that coat the outside of the droplets and keep an emulsion form separating. An emulsifier causes an emulsion concentrate to disperse spontaneously into small, stable droplets when added to water. It substitute for constant, physical agitation of spray liquids during the field operation. Examples: 15-S-9, Tergitol-NPX, ABS, Altox-3406 to 3408 and Solvaid.

Dispersants (or dispersing agents): Dispersants stabilize suspensions. They keep fine particles of the wettable powders in suspension in which even after initial vigorous agitation has been withdrawn from the spray tank. They act by increasing the hydration of fine particles of wettable powders laden with the herbicides. Examples – Multifilm, Tryad and Biofilm. If a fine powder is mixed with an excess of water, the particles will settle slowly at a rate proportional to their size. The particles may tend to clump together (flocculate), so increasing their size and thus the rate of setting. Dispersing agents are chemicals that surround the powder particles and reduce their tendency to clump together.

**Kind and composition of herbicide emulsions**

There are three kinds of herbicide emulsions, namely

1. oil in water (O/W) emulsion (normal emulsion): In normal emulsion (O/W) the toxicant particles are embodied in oil globules and water is the surrounding continuous phase.
(2) Water in oil (W/O) emulsion (invert emulsion): in invert emulsion (W/O), the carrier water is broken into discontinuous globules and they are surrounded by continuous phase of oil containing the toxicant.

(3) Oil in water in oil (O/W/O) emulsion (bivert or double invert emulsion): In the bivert system, each spray droplet has an oil centre, an outer layer of water and yet another layer of oil. Herbicide may be included in either phase.

**Coupling agents (Solvents and Co-solvents):** Coupling agent is a chemical that is used to solublize a herbicide in a concentrated form in so far as the resulting solution is soluble or miscible with water in all proportions. 2,4-D (acid form), for instance, is almost insoluble in water, but it can be dissolved in polyethylene glycol to make it water soluble. Other common solvents used for solublizing the otherwise water insoluble herbicides are: lanolin, carbowax, HAN, xylene, petroleum ether, carbon tetrachloride, methyl chloride, various alcohols, acetone etc.

**Humicants (Hygroscopic agents):** Humicant prevent rapid drying of herbicide sprays on the foliage, thus providing an extended opportunity of herbicide absorption. Himicants are also called hygroscopic agents. Example – glycerol.

**Adhesive or sticking agents:** These are chemicals added to herbicide concentrates to hold the toxicant to intimate contact with the plant surface. They also reduce washing off of the toxicant from the treated foliage by rain. Example- synthetic latex polymers, emulsifiable mineral oils, polymerized fatty acids and emulfiable resins.

**Spreaders:** Spreaders and wetting agents are closely related. When the wetting agent reduces the surface tension, spreading flows naturally with these chemicals, known as spreaders.

**Compatibility agents:** Some farmers, in order to save one spraying operations mix herbicides with another herbicide, insecticide, fungicide, fertilizer etc. at the time of spray (tank-mix). To achieve this objective some compatibility agents like ‘Compex’ are added to herbicide formulation in order to avoid any antagonistic effects of two chemicals which are applied in one operation of spraying.
**Activators** (synergists): Activators are chemicals with such cooperative action with herbicide that the resultant phytotoxicity is more than the effect of the two working independently. Many phytoblend oils such as arche X 796, 7N, 11E, Mobilsol-ICO etc. help in improving herbicide activity. Ammonium thiocyanate (NH4SCN) is a widely used activator of amitrole and the combination is designated as amitrole-T. Also nitrogenous fertilizers like urea, ammonium chloride and ammonium nitrate have been employed to enhance 2,4-D phytotoxicity and reduce its rate of application.

**Drift control agents:** Lower than 150 micron diameter spray droplets are considered drift susceptible. Spray droplet diameter can be increased by using (1) invert emulsion or (2) thickening agents or (3) particulating agents. Thickening agents make the spray liquid viscous. Example- sodium alginate, hydroxyethylcellulose and dacagin. Decagin resists both spray and vapour drifts. It is used as 2-8 kg/100 gal spray.

**Crop oils:** Crop oils are premium light petroleum oils. Emulsifiers are added to allow mixing in water. The use of oil is based on the premise that they improve the efficacy of herbicides by acting as surfactants/spreaders, penetrants, deposition and antidrift agents and anti-evaporants.
There are various application methods for treating weeds with herbicides. It's important to choose the right method for your particular weed problem and the types of chemicals you are using. Mowing is a commonly used control procedure for small brush because the equipment is readily available and the results are immediate. However, this method generally provides only short-term success because it leaves live stumps and root-stocks that re-sprout. Another strategy that can reduce some troublesome species is fire. However, fire can be tricky to manage and it is difficult to generate a fire with sufficient heat capacity to kill most hardwood species along fencerows, ditch banks and other sites with low plant density.

Herbicides are often the most effective and inexpensive means of controlling plants. There are several application techniques that can be used to control herbaceous weeds, trees and bushes of various sizes. Not all bush species are equally susceptible to herbicides. Therefore, results may vary for any of these application methods, relative to bush size and species.

**Foliar spray**

In foliar spraying, the herbicide is diluted with water or diesel at a specific rate, and sprayed over the foliage to point of run-off (until every leaf is wetted, but not dripping).

This method is most suited to shrubs, grasses and dense vines less than 6 m tall. Advantages include quickness and economy. Disadvantages include the potential for spray drift and off-target damage.

Foliar spraying can be done a number of ways, including:

- blanket spraying using a boom spray from a tractor
- a hose and handgun spraying solution from a herbicide tank
- a backpack spray unit
- with splatter guns (larger droplets at higher concentrations) for regrowth.
For weedy species auxin-type herbicides (such as triclopyr) are generally most effective early in the season while enzyme-inhibiting herbicides (imazapyr and others) are most effective in the late summer or fall. Glyphosate is most effective in late summer or fall—after blooming, but prior to change in leaf color.

Complete coverage of all foliage is essential for control, but over-application (that leads to spray run-off) will reduce effectiveness. It is important to control spray drift when making foliar applications. Certain desirable hardwood and crop species are highly sensitive to spray drift and can be inadvertently damaged. If plants have been mowed, it is important to allow them to regrow to a height of 3 or 4 feet before herbicide application.

**Basal barking**

This method involves mixing an oil-soluble herbicide in diesel and spraying the full circumference of the trunk or stem of the weed.

Basal bark spraying is suitable for:

- thin-barked woody weeds
- undesirable trees
- saplings, regrowth, and multi-stemmed shrubs and trees.

Basal barking will usually destroy weeds as long as the bark is not wet or too thick for the diesel to penetrate. It is important that the lower 12 to 18 inches of the stem be treated on all sides with the herbicide/oil mixture. Adequate coverage is essential, since treating only one side of the stem will result in controlling only half of the tree. Basal applications can be made any time of the year, but are most effective during the dormant season when leaves are not present.
Basal applications will not provide rapid control. Herbicide injury is often not observed for several weeks after treatment and total control may require several months. Additionally, basal treatment is not effective on older trees with thick bark. For older trees, other application techniques should be employed.

**Hack and Squirt**

The hack-and-squirt technique is ideal for control of large trees that cannot be managed with basal applications. This method requires that you use a small axe, machete, or hatchet to cut through the thick bark and into the sapwood. When hacking, it should be done in a downward motion, leaving a “cup” to hold the herbicide solution. If the cut does not hold herbicide solution, it will leak out and become ineffective. After hacking the entire circumference of the tree, 1 squirt (approximately 1 ml) should be placed in each cut (Fig). The addition of a basal oil is not required for this procedure.

This method of application is advantageous because it is highly selective and injury to surrounding species is not common. It can also be done at any time during the year, but treatment of some species in the spring can be reduced because of heavy sap flow pushing the herbicide from the cut surfaces. Rainfall soon after application washes the herbicide away and limit uptake.

**Stem injection**

The stem injection method involves drilling or cutting through bark into the sapwood tissue of woody weeds and trees to transport the chemical throughout the weed.

It is essential to apply the herbicide immediately (within 15 seconds of drilling or cutting), as stem injection relies on the active uptake and growth of the weed to move the herbicide through its tissue.
Drill and fill method

The drill and fill method is used for trees and woody weeds with stems or trunks greater than 5 cm in circumference, and is also referred to as tree injection.

This method uses a battery-powered drill to drill downward-angled holes into the sapwood approximately 5 cm apart. Using a backpack reservoir and syringe can deliver measured doses of herbicide solution.

Only trees and shrubs that can be safely left to die and rot should be treated this way. If the tree or shrub is to be felled, allow it to die completely before felling.

Axe cut method

The axe cut method can be used for trees and woody weeds with stems or trunks greater than 5 cm in circumference. It involves cutting through the bark into the sapwood tissue in the trunk, and immediately placing herbicide into the cut. The aim is to reach the tissue layer just under the bark, which will transport the herbicide throughout the weed.

Using an axe or tomahawk, horizontal cuts are made into the sapwood around the circumference of the trunk at waist height. The axe is then leaned out to make a downward angled pocket to allow herbicide to pool. It's important not to entirely ringbark the trunk, as this will decrease the uptake of herbicide.

Cut stump

The cut stump method involves cutting off the weed completely at its base (no higher than 15cm from the ground) using a chainsaw, axe, brushcutter or machete. A herbicide solution is then sprayed or painted onto the exposed surface of the cut stump, with the objective of destroying the stump and the root system.
The herbicide should be applied to the cut surface as quickly as possible after the sawdust has been removed. If applied immediately, a herbicide/water solution is sufficient. If herbicide treatment is delayed and the cut surface has begun to dry, a herbicide/basal oil mixture must be used and applied to the top and around the collar of the stump.

For stumps greater than 3 inches in diameter, thoroughly wet the outer edge while avoiding herbicide runoff (Fig). This is because the only living tissue in larger trees is around the outer edge. Covering the entire cut surface will require more herbicide, most of which will provide little effect. For smaller stems it is appropriate to cover the entire cut surface (Fig). Herbicides can be applied using a backpack sprayer, squirt bottle, or paint brush. Regardless of how the herbicide is applied, a tracer dye should be included to ensure treatment of all individual stumps.

**Cut and swab**

This method is similar to the cut stump method, but is suited to vines and multi-stemmed shrubs. Here, the weed stems are cut through completely, close to the ground. Herbicide is then applied immediately to the cut surface emerging from the ground, via spray or brush application.

**Stem scraper**

Stem scraping is used for weeds and vines with aerial tubers. A sharp knife is used to scrape a very thin layer of bark from a 10 cm section of stem. Herbicide is then immediately applied to the exposed, underlying green tissue.
**Wick applicators**

This method consists of a wick or rope soaked in herbicide from a reservoir attached to a handle, or assisted with 12-volt pump equipment. The wetted wick is used to wipe or brush herbicide over the weed.

**Soil Spots**

This procedure is particularly useful when attempting to reclaim an area with a high density of small stems. This practice can also be used to remove individual specimens, but soil spotting is not as selective as other techniques and must be done with caution if desirables are in the vicinity of the application.

In areas with high stem density, herbicides should be applied as thin streams (not broadcast) on a grid pattern. The application rate and size of the grid depend on the soil texture and species composition. For fencerows, a single band may be applied, but larger stems should be treated individually to ensure control.

Soil spotting requires that the herbicide be taken up by the roots in order to be effective. Therefore, only soil active herbicides (imazapyr, hexazinone, tebuthiuron) can be used for this type of application. Soil spotting is often a slow process that may require multiple years to fully control some species. Highly susceptible species will be removed quickly, but those with higher tolerance can often endure several defoliation cycles before complete control is realized.

**Tables: Recommended herbicides for woody species in some application procedures**

<table>
<thead>
<tr>
<th>Herbicide</th>
<th>Application Rate</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Foliar Application</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imazapyr (Arsenal)</td>
<td>1 – 3%</td>
<td>Excellent control of sweetgum and maples. Use higher rates for oaks and cherry. A non-ionic surfactant is required.</td>
</tr>
<tr>
<td>Glyphosate (Several)</td>
<td>5 - 8%</td>
<td>Cover as much of the foliage as possible and spray until wet. If the brush has been cut, delay application for approximately 1 year. Retreatment is commonly required.</td>
</tr>
<tr>
<td>triclopyr + 2,4-D (Crossbow)</td>
<td>1 - 1.5%</td>
<td>For control of various herbaceous and woody species. This product contains 2,4-D ester precautions to manage drift must be employed. Repeat applications are often required.</td>
</tr>
<tr>
<td>Herbicide</td>
<td>Application Rate</td>
<td>Comments</td>
</tr>
<tr>
<td>---------------------------</td>
<td>------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Basal Bark</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imazapyr (Stalker)</td>
<td>8–12 oz/gal</td>
<td>Best for trees less than 4 inches DBH*. Be aware that imazapyr is highly active in the soil. If desirables are near to a treated individual, it is possible for the herbicide to wash off into the soil and injure or kill the desirable.</td>
</tr>
<tr>
<td>Triclopyr (Pathfinder)</td>
<td>100%</td>
<td>Pathfinder is a &quot;Ready to use&quot; product that is formulated and dosed correctly for this type of application. Apply Pathfinder at 100% strength as directed.</td>
</tr>
<tr>
<td><strong>Hack-and-Squirt</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imazapyr (Arsenal AC)</td>
<td>6 oz/gal</td>
<td>One hack per 3 inches DBH.</td>
</tr>
<tr>
<td>Triclopyr (Garlon 3A)</td>
<td>50%</td>
<td>One hack per 3 or 4 inches DBH. Apply 0.5 ml undiluted herbicides or 1 ml of 50% solution in water.</td>
</tr>
<tr>
<td>Hexazinone (Velpar)</td>
<td>100%</td>
<td>One hack per 4 inches DBH. Use undiluted herbicide.</td>
</tr>
<tr>
<td>Glyphosate (several)</td>
<td>50%</td>
<td>1 ml per 2 or 3 inches DBH, applied below the branches. For larger trees, best results are observed from applying glyphosate in a continuous frill around the stem.</td>
</tr>
<tr>
<td><strong>Cut Stump</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imazapyr (Arsenal AC or Stalker)</td>
<td>6 oz/gal (for Arsenal AC) or 8-16 oz/gal (for Stalker)</td>
<td>Apply to the top and side of a freshly cut stump. Garlon 3A is excellent for this use. If surface of stump has began to dry prior to herbicide treatment, apply Chopper, Garlon 4, Remedy or Pasturegard in basal oil - or recut the stump and apply the freshly cut surface. Garlon 3A will not effectively mix with basal oils.</td>
</tr>
<tr>
<td>Triclopyr (Garlon 3A)</td>
<td>50 - 100% in water</td>
<td></td>
</tr>
<tr>
<td>Glyphosate (several)</td>
<td>50-100%</td>
<td>Apply to cut stumps immediately after cutting. Glyphosate is not effective on stumps that have started to dry after cutting. If immediate treatment is not possible, other herbicides should be selected since glyphosate will not mix with basal oils.</td>
</tr>
<tr>
<td><strong>Soil Spot</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hexazinone (Velpar)</td>
<td>100%</td>
<td><strong>Individual plants.</strong> Use 2-4 ml of undiluted herbicide per 1 inch DBH. Apply within 3 ft of root collar. If more than 4 ml is required, apply as evenly spaced spots around the stem.</td>
</tr>
<tr>
<td>Tebithiuron</td>
<td>2.5 to 20 lb/A</td>
<td>Control is rate- and species-specific. See label for control of</td>
</tr>
<tr>
<td>Herbicide</td>
<td>Application Rate</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------</td>
<td>------------------</td>
<td>----------</td>
</tr>
<tr>
<td>(Spike 20P)</td>
<td>(20P)</td>
<td>specific species. Tebuthiuron will move in water, so only apply in areas where runoff will not carry the herbicide to desirable species. Spike 20P is restricted to certain Florida counties, so consult product label before use.</td>
</tr>
</tbody>
</table>

**Application of herbicides to plants grown under protection**

Special care is needed where herbicides are used under glass or polythene. In this environment, day temperatures are higher, plants grow more rapidly, leaf cuticle is thinner and foliage is softer. In general, there is a greater risk of damage from doses of leaf acting herbicides that would be safe in the open. For example, Butisan S is increasing in popularity as a treatment for nursery stock when foliage is firm and not actively growing. Soft young foliage is liable to be injured and small scale testing of this or other approved herbicides is necessary before general applications are made on plants grown under protection.

In addition, there is a greater risk of damage in protected environments from the volatilisation of certain herbicides such as oxyfluorfen.

**Herbigation**

Chemigation is the process of applying an agricultural chemical (pesticides or fertilizers) to the soil or plant surface with an irrigation system by injecting the chemical into the irrigation water. Depending on the type of agricultural chemical being applied, chemigation may be referred to as herbigation, insectigation, fungigation, fertigation, etc. Application of herbicides through irrigation systems appears to have a bright future. However, farmers should proceed with caution. As with any new practice, each farmer should become familiar with the practice and know how it will perform with his conditions. The future of herbigation will depend largely on development of equipment that is accurate and simple to use.

Many herbicides have been demonstrated to selectively control weeds in crops when applied through a well designed and properly functioning sprinkler irrigation system. These include preemergence agents like alachlor, atrazine, benefin, bensulide, butylate, chloramben, cyanazine, dimethazone, diphenamid, trifluralin, and vernolate. Postemergence herbicides used successfully
via herbigation include acifluren, bromoxynil, chloramben, fluazifop, halaxyfop, lactofin, naptalam+dinoseb, tridiphane, and xylafop. Trifluralin has also been used successfully as a postemergence treatment in corn for suppression of shattercane and control of sandbur (applied prior to 4-leaf stage of corn) (3). Bentazon, glyphosate, imazaquin, MSMA, paraquat, and sethoxydim have also been used, but have shown poor or variable activity. Crop tolerance to preemergence herbigation has been good to excellent, and crop tolerance to postemergence herbigation has been good also.

Postemergence herbigation results have been encouraging but somewhat variable, most likely due to herbicide runoff from plant foliage. Currently, postemergence application of herbicides through sprinkler irrigation is not feasible in less than 0.1 inch or 2,715 gallons/acre of water, exceeding the volume of water necessary to cause foliage runoff by approximately 28 times. All water soluble or water-miscible herbicide formulations have generally shown poor activity when applied postemergence via herbigation, with the exception of sethoxydim. However, additions of nonemulsifiable vegetable or petroleum-based oil have been shown to aid in the improvement of more consistent postemergence efficacy via herbigation (tridifane activity was not positively influenced by an oil carrier).

Chemigation is a method that has been used successfully for many years in certain types of agriculture and is mostly used in conjunction with drip or sprinkler irrigation systems. It has a number of advantages over other types of spray applications, but also some disadvantages.

**Advantages include:**

- It is generally much cheaper than other forms of application
- It provides more uniform coverage
- Less labour is required
- The chemical is applied more accurately
- The chemical can be applied in conditions unfavourable for other forms of application (i.e. when the soil is wet)
- There is limited soil compaction and crop damage
- There is less human exposure to chemicals.
Disadvantages include:

- Initial cost of equipment can be high
- It requires more knowledge and management
- Extra equipment is needed for safety and storage
- It presents hazards through potential run-off and groundwater contamination
- Application times are generally longer
- There is a possibility of equipment malfunction while unattended
- There is a potential to over-water crops in periods when chemicals are needed, but not water.

The biggest hazard by far of chemigation is that it has the potential to contaminate irrigation water sources if safety measures are not implemented and maintained. Risks include the chemical flowing back into the water source when the system shuts off, the chemical continuing to be injected after the irrigation water is shut off, causing flow back or spillage, or the chemigation system shutting down while the irrigation system continues to run, causing overflow and spillage.

Safety devices that need to be installed to prevent this from happening include:

- A main pipeline check valve between the point of chemical injection and the irrigation pump, to prevent flow back into the water source
- Interlocked chemigation and irrigation power and water supplies, so that if one shuts down, the other does too
- A solenoid valve installed in the chemical injection line as an added precaution
- A check valve in the chemical injection line between the meter and the point of injection
- A shutdown switch that activates at lower pressure installed in the main pipeline.

In addition to these devices, other safety measures include using double-skinned chemical supply tanks, not locating them too close to the water source, ensuring that all components in contact with chemicals are chemical-resistant, and posting clear warning signs on the field’s perimeters during chemigation treatment.
Herbicide Resistance

Resistance to pesticides is a serious, and growing, problem. Worldwide, more than 600 species of pests have developed some level of pesticide resistance. If resistance to a particular pesticide or “family” of pesticides evolves, these products can no longer be effectively used thereby reducing the options available for pest management.

Resistance is defined as a change in the sensitivity of a pest population to a pesticide, resulting in the failure of a correct application of the pesticide to control the pest. Resistance can develop when the same pesticide or similar ones with the same mode of action are used over and over again. It often is thought that pests change or mutate to become resistant. However, it is not the individual pest (insect, weed or microorganism) that changes, but the population.

Herbicide resistance is the inherited ability of a biotype of a weed to survive an herbicide application to which the original population was susceptible. A biotype is a group of plants within a species that has biological traits (such as resistance to a particular herbicide) not common to the population as a whole. In simple terms, resistance refers to a situation where a given herbicide, applied at the recommended rate and time, once controlled a particular weed population but, after repeated use, that herbicide no longer controls that population. That population is said to be resistant (or resistance has developed in that population).

Wherever herbicides are used on a consistent basis, it is important to recognize the difference between weed population shifts, and resistance. Both weed population shifts and resistance are undesirable and make weed management difficult.

Herbicide Resistance Terms to Know

Susceptibility and Tolerance: Herbicide susceptibility is the degree to which a plant is subject to injury or death due to a particular herbicide. Herbicide tolerance is the inherited ability of a species to survive and reproduce following an herbicide treatment. There was no selection to make the plants tolerant; those plants simply possess a natural tolerance.

Weed Population Shift versus Herbicide Resistance: A weed population shift is a change over time in the relative abundance of the species comprising a weed population. With the repeated use of an herbicide certain species may become dominant due to selection for those that are
tolerant. In some cases weed shifts can also occur when a ‘low’ rate is used repeatedly and more difficult to control species may become dominant. These populations are not herbicide resistant.

**Herbicide resistance** causes changes in the composition of the population because of resistant biotypes. Originally at very low frequencies in the weed population, resistant biotypes build up when the herbicide to which those individuals are resistant is used repeatedly. **Selection and selection pressure:** Selection is the process by which control measures favor resistant biotypes over susceptible biotypes. Selection pressure refers to the intensity of the selection.

**Mode of action and target site or mechanism of action:** Mode of action (MOA) describes the plant processes affected by the herbicide, or the entire sequence of events that results in death of susceptible plants. It includes absorption, translocation, metabolism and interaction at the site of action. Target site of action or mechanism of action is the exact location of inhibition, such as interfering with the activity of an enzyme within a metabolic pathway. Herbicides are organized by families that share a common chemical structure and express similar herbicidal activity on plants. Of the hundreds of different herbicides on the market today, many of them work in exactly the same way or, in other words, have the same mechanism of action. Fewer than 30 plant-growth mechanisms are affected by current herbicides.

**Types of resistance:**

Multiple resistance is the phenomenon in which a weed is resistant to two or more herbicides having different mechanisms of action. An example would be a weed resistant to sulfonylurea herbicides (ALS inhibitors) and glycines (EPSP synthase inhibitors). Multiple resistance can happen if a herbicide is used until a weed population displays resistance and then another herbicide is used repeatedly (without proper resistance management) and the same weed population also becomes resistant to the second herbicide, and so on. Multiple resistance can also occur through the transfer of pollen (cross-pollination) between sexually compatible individuals that are carrying different resistant genes. **Cross resistance** occurs when the genetic trait that made the weed population resistant to one herbicide also makes it resistant to other herbicides with the same mechanism of action. An example would be a weed resistant to imidazolinone herbicides (ALS inhibitors) and
sulfonylurea herbicides (also ALS inhibitors). Cross resistance is more common than multiple resistance, but multiple resistance is potentially of greater concern because it reduces the number of herbicides that can be used to control the weed in question.

Is Resistance to Blame?

Most weed control failures are not due to resistance. Before assuming weeds surviving an herbicide application are resistant, eliminate other possible causes of poor control:

1. Herbicide application
   a. Inadequate rate
   b. Poor spray coverage and/or incorporation
   c. Improper timing of application of postemergence herbicides (after weeds are too large to control)
   d. Failure to use an adjuvant (if needed)
   e. Excessive dust with post-emergence applications
   f. Spray intercepted at application by wheel traffic
   g. Antagonism between two or more herbicides

2. Soil and/or climatic conditions
   a. Excessively wet or dry soil
   b. Seedbed condition (clods, etc.)
   c. Herbicide adsorption to soil particles or organic matter
   d. Stress conditions, such as hot and dry
   e. Lack of timely rainfall for activation
   f. Wash-off of postemergence herbicides

Incidence and History of Herbicide Resistance

The first reported case of herbicide resistance in the United States was reported in the 1950’s. Field bindweed resistant to 2,4-D was reported in Kansas in 1964, and common groundsel resistant to triazine herbicides was discovered in Washington in 1970. Beginning in the 1980’s, the number of reported resistant biotypes began increasing rapidly in the U.S. and worldwide. Resistance to one or more of 25 herbicide families has been observed in more than 65 weed species in the U.S.
Herbicide resistance has been a greater problem with some modes of action than with others. The greatest number of resistant biotypes worldwide has been observed with the ALS inhibitors (imidazolinones, pyrimidinylthiobenzoates, sulfonylureas, triazolopyrimidines). The second most common group with resistant biotypes is the photosystem II inhibitors (primarily triazines). Note that no resistant biotypes have been reported to some mechanisms of action, to date. Part of this is obviously due to the extent of usage of some herbicide groups. There are numerous ALS inhibitors, for example, and they have been widely used. On the other hand, there are few cellulose inhibitors and they have been used on comparatively fewer acres. However, some modes of action are more prone to resistance than others. The ALS inhibitors and ACCase inhibitors appear to be at high risk; this should be considered in management plans. Dinitroaniline and triazine herbicides appear to have a medium level of inherent risk while chloroacetamides and synthetic auxins have a low inherent risk. It is important to remember that a low inherent risk for resistance combined with extensive use can result in a higher risk potential. For example, glyphosate was generally considered to have a low inherent risk of resistance evolution. However, with the wide-spread use of Roundup Ready crops, glyphosate is being used extensively in the absence of other weed control methods, thus greatly increasing the incidence of resistance.

Mechanisms of Herbicide Resistance

What occurs within a resistant plant that allows it to survive after an herbicide application? What characteristics do the resistant plants possess that the susceptible plants lack? The four known mechanisms of resistance to herbicides are:

1. Altered target site:
   An herbicide has a specific site (target site of action) where it acts to disrupt a particular plant process or function (mode of action). If this target site is somewhat altered, the herbicide no longer binds to the site of action and is unable to exert its phytotoxic effect. This is the most common mechanism of herbicide resistance.
2. Enhanced metabolism:

Metabolism within the plant is one mechanism a plant uses to detoxify a foreign compound such as an herbicide. A weed with the ability to quickly degrade an herbicide can potentially inactivate it before it can reach its site of action within the plant.

3. Compartmentalization or sequestration:

Some plants are capable of restricting the movement of foreign compounds (herbicides) within their cells or tissues to prevent the compounds from causing harmful effects. In this case, an herbicide may be inactivated either through binding (such as to a plant sugar molecule) or removed from metabolically active regions of the cell to inactive regions, the cell wall, for example, where it exerts no effect.

4. Over-expression of the target protein:

If the target protein, on which the herbicide acts, can be produced in large quantities by the plant, then the effect of the herbicide becomes insignificant.
Proactive Herbicide Resistance Management

Early detection of resistance means management will be easier, and it increases the potential to avoid the spread of the resistant biotype. Unfortunately, because resistant plants and susceptible plants look alike, resistance often is not detected until the resistant biotype has spread to 30% or more of the field and perhaps to surrounding fields. Therefore, a proactive approach using diverse weed control tactics is the most effective way to manage herbicide resistance.

The primary objective of proactive resistance management is to reduce selection pressure by:

1) selecting and using herbicides correctly;

2) recognizing weed characteristics that promote resistance; and,

3) managing fields, farms, or sites wisely.

Doing what it takes to proactively manage herbicide resistance is a good alternative to paying the price for overcoming resistance once it occurs.

1. Select and use herbicides correctly:

   a. Rotate herbicides with different mechanisms of action, not just different label names. Avoid consecutive applications of the same herbicide unless it is used in a tank-mix or prepack containing an herbicide with a different mechanism of action or used with other weed management options such as cultivation and cultural methods. The herbicides and/or alternative methods used must be active against the target weed.

   b. Use herbicides with different mechanisms of action in the same spray tank, in a given crop year or between years. This can be accomplished most efficiently with tank-mixes and pre-packs. Tank-mixes and Pre-packs are combinations of two or more herbicides applied as a single mixture. Tank-mixing allows for adjusting of the ratio of herbicides to fit local weed and soil conditions, while premixes are formulated by the manufacturer. The combinations are designed to broaden the spectrum of weeds controlled by an individual herbicide and, if the combination is composed of herbicides with different mechanisms of action active against the same weeds, will contribute to resistance management. The different herbicides in the mixture must be active against the target
weeds so that weed biotypes resistant to one mechanism of action are controlled by the herbicide partner with a different mode of action. Theoretically, repeated use of any tank-mix or pre-pack combination may give rise to herbicide resistance, if resistance mechanisms to each herbicide in the mix arise together but the probability is very low.

c. Minimize the use of long-residual herbicides. Susceptible plants emerging later in the season following use of a long-residual herbicide (or multiple applications of a non-residual herbicide) are still exposed to the herbicide, thereby increasing selection pressure.

d. Apply herbicides evenly and accurately (link to calibration module), and use labeled application rates. Lower than label rates have been shown to increase the likelihood of metabolic resistance. Using higher than labeled rates is illegal and can also enhance selection pressure for resistance.

2. **Recognize weed characteristics that promote resistance:**

a. Some weed populations are more prone to resistance in which there is a high frequency of the resistance trait. If the initial frequency is 1 resistant plant in a population of 100,000, as compared with 1 in 10 million, resistance will develop much more rapidly. A high initial frequency of resistant biotypes in many weed families is one reason resistance to ALS inhibitors develops rapidly.

b. Annual weeds can have several reproductive generations per growing season, and those that cross pollinate tend to be more prone to develop resistance. Species that have adaptations for seed dispersal over greater distances also tend to be more prone to develop resistance, or the resistant biotype spreads more rapidly.

c. Where a strong resistance trait is present, failure will be more apparent in weed species that are initially highly susceptible to the herbicide. If a species is highly susceptible, fewer susceptible plants escape treatment and reproduce. Only resistant biotypes survive and reproduce.
3. **Manage fields, farms, or sites wisely:**

   a. Capitalize on cultural practices to reduce reliance on herbicides. When fewer non-chemical control methods (such as cultivation) are used, resistance is more likely to develop, or will develop more rapidly. Use correct row spacing, proper fertility, optimum planting dates, and management of other pests to maximize the crop's ability to compete with weeds. Where erosion and/or limited soil moisture are not a concern supplement chemicals with timely cultivation. For annual cropping systems, start with a clean field and control weeds early by using a burn down treatment or tillage in combination with a pre-emergence residual herbicide, as appropriate. Do not let weeds go to seed.

   b. Rotate crops to allow greater flexibility in choosing different tillage practices and/or the use of herbicides with different mechanisms of action. Changing tillage practices and herbicides makes it more likely that you avoid consistently favoring the same weed species.

   c. Scout fields regularly and keep good records of herbicide use to aid in planning for future years. Note the weed species that have been present in a given field, which herbicides were applied and where, and what level of control (%) was achieved.

   d. Be aware that resistant weeds can spread from highways, railroads, utility rights-of-way and other areas near your farm or treated site.

**How to Manage Herbicide Resistance**

If herbicide resistance is confirmed or highly suspected, diverse approaches to managing herbicide resistance need to be incorporated into weed management strategies immediately for the species in question.

1. It is best to stop using the herbicide in question and other herbicides with the same mechanism of action. However, in many cases the herbicide continues to work on a large number of weeds and is still the best choice for overall weed control. If the decision is made to continue using the herbicide, there are several options:

   a. Use proactive weed control (preplant or preemergence) with an herbicide tank mixture or prepack having at least one mechanism of action that is known to control the resistant weed.

   b. Use postemergence herbicides only in tank mixtures or prepacks with at least one mechanism of action that is known to control the resistant weed.
c. Do both a. and b.

* Any of these options provides at least one additional MOA that will help to prevent further spread of the resistant weed. In addition, other weed control tools should be used to complement the MOA that is still active on the resistant weed so that undue selection pressure is not placed on the additional MOA.

2. If the resistant weed is confined to relative small areas, take steps to prevent seed production. If the weed is still small enough to control with other herbicides, treat the affected spots. Alternatively, the weed could be removed by hand, or the crop in infested patches could be sacrificed and the weed controlled by destructive tillage or with the use of a non-selective spot herbicide application. Do not let resistant weeds go to seed.

3. Avoid moving seed or vegetative propagules to other fields and farms. Use a power washer or compressed air to help remove seed and plant parts from any equipment used in the field. If any fields have a history of herbicide resistant weeds, use farm equipment in those fields last.

4. Seek advice from University Extension weed specialist to assist in the long term planning of weed control in subsequent crops.

Adhering to the resistance management principles outlined above will help delay or prevent resistance from recurring and prove beneficial in managing resistance the long term.

**Spraying by the Numbers**

Herbicides are grouped according to family (e.g., triazines) and target site of action or mechanism of action (e.g., acetolactate synthase, or ALS, inhibitors). Herbicides within a family have similar chemical structures and typically the same site or mechanism of action (MOA). Knowing the chemical family and MOA group to which an herbicide belongs and knowing which other herbicides have the same MOA are critical for creating a plan to prevent or delay development of herbicide resistance.
Herbicide Antidotes

Antagonists (also known as safeners, antidotes or crop protectants) – are substances used along with herbicides to protect crop plants from possible damage by the herbicide. Adsorbents physically shield the crop seed from contact with the herbicide that otherwise would cause injury. This approach included the use of activated carbon, lignin by products, ion exchange resins, and various clays. The requirements of an application technique different from that used for herbicide application, the expense, and inadequate control of weeds have forced the search for a better alternative. The observation of an antagonistic interaction between the herbicide 2,4-D and the herbicide 2,4,5-T (Hoffman, 1953) led to development of chemicals that may be applied with the herbicide to protect the crop plant against herbicide injury. Research by Hoffman led to the introduction of naphthalic anhydride as the first commercial safener in 1971 against thiocarbamate herbicides in corn.

The research in this field has proceeded and a large number of chemicals have been screened as potential safeners. As related to their terminology, the word safener is used interchangeably with antidotes, antagonists and protectants. Among several chloroacetamide chemicals tested, dichlormid (2,2-dichloro-N,N-diallylacetamide) was shown to be the most active chemical to protect cereal crops against thiocarbamate herbicides (Chang et al., 1973; Pallos et al., 1978). Dichlormid was introduced as a commercial safener in 1972.

The safening activity of oxime ether compounds has been successfully examined and three of them were introduced as commercial safeners (e.g., cyometrinil, oxabetrinil, and CGA-15281) against chloroacetanilide herbicides in sorghum (Ellis et al., 1980; Chang and Merkle, 1982). Later, the thiazolecarboxylic acid (flurazol) was introduced against alachlor in sorghum (Sacher et al., 1983). Then, the safener fenclorim was introduced against the herbicide pretilachlor in rice (Hatzios, 1996). Other chemicals such as pyrimidine triazole derivatives (Strelkov et al., 1998), or of undisclosed identity have shown different types of safening activity.

Some success has been achieved in the protection of broadleaved crops such as soybean (Glycine max L.) against metribuzin (Varvina, 1987; Phatak and Varvina, 1989), against chloramben in cucumber (Kneer and Hoppen, 1989), and against sulfonyleurea herbicides (Devlin and Zebiec, 1990). Microorganisms that degrade EPTC were applied by inoculation and used as safeners in corn (Nagy et al., 1991). A list of names of common safeners is listed in Table.
Soil-applied safeners –
These safeners are applied with the seed prior to planting or applied to the soil or crop together with the herbicides. They protect large-seeded grass crops (corn, sorghum, rice) against pre-plant incorporated or pre-emergence applications of thiocarbamate and chloroacetanilide herbicides. There has been limited success in protecting broadleaved crops. Most of the evidence suggests that these safeners protect the crop from injury by inducing enzymes that metabolize the herbicide, rather than by affecting herbicide absorption and translocation or its site of action. For example, these safeners increase the level of glutathione conjugation of chloracetanilide and thiocarbamate herbicides by inducing glutathione-S-transferases. As a result they may also increase the level of glutathione in the plant tissue.

Foliar-applied safeners –
These safeners enhance the activity of cytochrome P<sub>450</sub> monooxygenases and glucosyltransferases to protect grass crops from aryloxyphenypropionate, sulfonylurea or imidazolinone herbicide injury. An example is the safener, fenchlorazole-ethyl used to protect grass crops (i.e. wheat, rye, barley) from fenoxprop-ethyl injury. Normally, this class of herbicides controls grasses, but not broadleaf plants. These crop species possess higher levels of glutathione than grassy weeds and the safener enhances these levels further in the crop species, protecting them from herbicide damage.

Table: Chemical nomenclature of herbicide safeners

<table>
<thead>
<tr>
<th>Compound</th>
<th>Chemical name</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,8-Naphthalic anhydride</td>
<td>Naphthalene-1,8-dicarboxylic anhydride</td>
</tr>
<tr>
<td>Dichlormid</td>
<td>N,N-diallyl-2,2-dichloracetamide</td>
</tr>
<tr>
<td>Cyometrinil</td>
<td>(Z)-cyanomethoxyimino(phenyl)acetonitrile</td>
</tr>
<tr>
<td>Oxabetrinil</td>
<td>(Z)-1,3-dioxolan-2-ylmethoxyimino-(phenyl)acetonitrile</td>
</tr>
<tr>
<td>Flurazolone</td>
<td>Benzyl-2-chloro-4-trifluoromethyl-1,3-thiazole-5-carboxylate</td>
</tr>
<tr>
<td>Fenclorim</td>
<td>4,6-Dichloro-2-phenylpyrimidine</td>
</tr>
<tr>
<td>Benoxacor</td>
<td>(RS)-4-dichloroacetyl-3,4-dihydro-3-methyl-2H-1,4-benzoxazine</td>
</tr>
<tr>
<td>Fluxofenim</td>
<td>4-Chloro-2,2,2-trifluoroacetophenone O-1,3-dioxolan-2-ylmethyloxime</td>
</tr>
</tbody>
</table>
Uses
Safeners selectively protect crop plants from herbicide injury without protecting weeds. Many safeners are structurally similar to the herbicides that they antagonize. Naphthalic anhydride is the most versatile safener. It is less specific botanically and chemically than others. It protects various crops against a wide range of herbicides. Naphthalic anhydride has been tested successfully to protect corn against thiocarbamates and chloroacetanilides. It was useful in the protection of rice, grain sorghum, and oats, and was capable of providing safening activity against post-emergence application of selected herbicides. Naphthalic anhydride enhanced the tolerance of wheat, oats and barley to Barban. Also its safening activity has been extended to include broadleaved crops such as bean against EPTC. Codde (1988) detected its safening activity against the herbicide diclofop-methyl in oats. It was active against Metsulfuron in corn. Use of NA also protects crops from damage by imazaquin. NA protects rice against alachlor. Similarly, cyometrinil protects sorghum against alachlor.

Dichlormid was effective among other chloroacetamides (when added in small amount) in preventing the onset of EPTC injury to plants. The safening activity of dichlormid was effective against chloroacetanilide herbicides, sethoxydim, and against chlorsulfuron. Naphthalic anhydride (NA) or dichlormid can protect maize, sorghum and rice injury by chlorsulfuron.

Oxime ethers exhibited good chemical and botanical specificity in being highly selective safeners against chloroacetanilide herbicides in grain sorghum.
Furthermore, the safener flurazole has been introduced commercially against the herbicide alachlor in grain sorghum. Flurazole has high degree of chemical specificity failing to protect corn against thiocarbamate herbicides. Later on, the safener fenclorim was introduced to protect rice against the herbicide pretilachlor.

Methods of application
Several safeners can be formulated as seed dressings. Chemical and botanical specificity of the safener determines the most appropriate method of application. Naphthalic anhydride has limited specificity and offers protection to weeds when applied to soil. Thus it is mainly used as seed coating at a rate of 0.5% (w/w) of seed. Dichlormid has a good availability to plants from soil. Due to simplicity of this method, dichlormid is sold as a pre-packed formulation with the herbicide. Due to their marginal selectivity and their protection of weeds when applied to soil,
oxime ether safeners are used as seed coating of grain sorghum. Furthermore, the safener flurazole was effective when applied to soil as seed dressing. It seems that the commonly used safeners are having limitations either in offering protection for certain crops or against specific herbicides. The challenge is to introduce safeners that have wide range of activity in the field.

**Structure–activity relationship**

Among the studies examined structure–activity relationships of safeners, there was disagreement on significance of specific functional group for safenening activity. The presence of a dicarboxylic anhydride group and at least one aromatic ring attached directly to the anhydride was essential for the safening activity of naphthalic anhydride and its analogues (Zama and Hatzios, 1986).

On the other hand, the similarity of structures between chloroacetamide safeners and chloracetanilide herbicides has been studied as a base for their activity. In a soil-free system, the most effective safener was the one most similar in structure to the herbicide. Mono and trichloroacetamides were less effective as safeners compared to dichloro compounds. Those chemicals with two substitutes on the nitrogen were more active than those with one group. However, dichloroaceto group was essential for activity of chloroacetamide safeners (Dutka and Komives, 1987).

Examination of structure–activity relationship of oxime ethers revealed that presence of oxime and pyridine groups or two oxime groups and an aryl group attached to oxygen in the molecule was essential for the safening activity of oxime ethers.

**Transformation**

Safeners undergo transformation in soil, plant and in mammals. Rate of transformation and transformation products depends upon the safener, the medium where it resides, and environmental factors. In soil, decarboxylation of naphthalic anhydride represented a major pathway of its degradation.

Dichlormid underwent dechlorination, dealkylation, oxidation and hydrolysis following application to soil. Naphthalic acid has been identified as a metabolite of naphthalic anhydride in plants. Dichlormid transformed through oxidation, dealkylation, dechlorination, hydrolysis and conjugation with plant constituents. On the other hand, conjugation of flurazole with glutathione (GSH) has been demonstrated.
In mammals, metabolism of dichlormid was similar to that demonstrated in plant and soil. Glycolamide, glyoxamidoxamic acid and dichloroacetic acid were identified as final metabolites of dichlormid.

**Limitations and adverse effects**

In the field, performance of safeners is influenced by environmental factors such as temperature, soil moisture, soil structure, and the rate of application of the safener. As an example, application of naphthalic anhydride at high rate that that used for the safening activity caused an injury of crops (Blair, 1979). At a commercial rate, injury symptoms such as stunning and chlorosis have been detected following application of naphthalic anhydride in corn and sorghum. Further, performance of naphthalic anhydride against EPTC was marginal in sandy or silt soil compared to clay loam soil.

Soil incorporation, seed placement, cultivator type and herbicide behavior (e.g. leaching, breakdown) have affected performance of dichlormid. The difference in the degree of solubility between EPTC and dichlormid caused leaching of the two compounds at different rate under heavy rainfall or irrigation conditions, causing the loss of the two chemicals from the treated zone, then causing plant injury. Published reports showed significant reduction in the germination rate of sorghum following the use of oxabetrinil and cyometrinil.

**Mechanisms of action**

Early investigations suggested that safeners might act through a single mechanism that was assumed to be common to all crop-herbicide safener combinations. Ezra et al. (1983) hypothesized that action of safeners may be the result of a series of multiple interactions between safener and herbicide. The fact that the currently available safeners exhibit botanical specificity for graminaceos crops with moderate tolerance to herbicides and chemical selectivity toward soil applied and shoot absorbed thiocarbamates and chloroacetanilides led to a suggestion that the action of safeners relates to physiological, biochemical and molecular function(s). The explanation is that these functions may are unique in these crops or are highly efficient in the graminaceos crops that affected by thiocarbamates and chloroacetanilides and altered by safeners. But it seems that these systems are either not present or not affected to the same extent by herbicides and safeners in other crops (Hatzios, 1997).

Similarity between the site(s) of uptake and action of both safeners and herbicides in the coleoptiles region of the plant shoot led to a suggestion that safeners might counteract herbicides
at the site of uptake or action. The fact that the available safener should be applied simultaneously or before application of the herbicide indicates they prevent but not reverse the injury caused by the herbicide. Details of proposed mechanisms of actions of the available safeners are outlined below.

**Absorption and/or translocation**
Safeners effect on absorption/translocation of the herbicide could be through chemical interaction, biochemical disruption of some processes that may cause an alteration in pattern of uptake and distribution of the herbicide, or by competition at the site(s) of entry with the herbicide. Previous studies showed that safeners reduced uptake and/or translocation of herbicides, simulated the uptake, or resulted of no effect.

**Metabolism**
Tolerance of plant to the herbicide correlates with rate of detoxification. Safeners might alter herbicide metabolism directly by acting as chemicals activators of particular functional group(s) in the herbicide, or by affecting biological system(s) (e.g. enzymes) involved in herbicide metabolism. Alteration of herbicide metabolism has been proposed as the most likely mechanism of action of safeners.

Metabolism of thiocarbamates and chloroacetanilides is believed to be catalyzes by mixed function oxidase enzymes. Saferens could induce activity of the mixed function oxidase enzymes. This induction may enhance oxidation and hydroxylation of the herbicides. However, there is no conclusive evidence correlate induction of these enzymes by the action of the available safeners.

Conjugation of herbicides with GSH is a common pathway of metabolism. Rate of conjugation is related to tolerance of plant to a herbicide. Published studies showed that safeners increased GSH content in plants, and/or induced activity of glutathione-S-transferase (GST) enzyme. The suggestion is that an increase of GSH content of in the activity of the enzyme that catalyze conjugation if the herbicide may increase rate of metabolism of the herbicide into non-toxic metabolites. In this aspect, elucidation a conclusive mechanisms of action of safeners will remain a challenge that requires further research.

**Competition at site(s) of action**
One of the proposed hypotheses of safeners mechanisms of action is that safeners may counteract action of the herbicide at the target site(s) buy affecting physiological and biochemical process
involved in the action of the herbicide. Some of these effects are listed in Table. Mechanism of action of thiocarbamate and chloroacetanilide herbicides has not been fully elucidated. Their effects on plants show that more than one site of action is involved in their mechanism of action.

Symptoms of injury caused by thiocarbamates included stunting and dark-green leathery leaves, inhibition of shoot growth, failure of young leaves to unroll; twisting of shoots, and distorted, brittle and hard leaves. Safeners could compete with herbicides at the target site either by preventing the herbicide from reaching or acting on the target site, or through acting at a different site producing an effect that alter physiological or biochemical process which ultimately lead to counter action of the herbicide. Previous studies examined several site of actions of herbicides and the effect of safeners of these sites.

Table Effects of safeners on physiological or biochemical systems

<table>
<thead>
<tr>
<th>Compound</th>
<th>Effect</th>
<th>Crop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dichlormid</td>
<td>Lipid biosynthesis</td>
<td>Corn</td>
</tr>
<tr>
<td>1,8-Naphthalic anhydride and dichlormid</td>
<td>Fatty acid synthesis</td>
<td>Corn</td>
</tr>
<tr>
<td>Fenclorim</td>
<td>Acetate incorporation into lipid</td>
<td>Rice</td>
</tr>
<tr>
<td>Dichlormid</td>
<td>Cuticle formation</td>
<td>Corn</td>
</tr>
<tr>
<td>Dichlormid</td>
<td>Protein synthesis</td>
<td>Corn</td>
</tr>
<tr>
<td>1,8-Naphthalic anhydride and dichlormid</td>
<td>Membrane permeability</td>
<td>Onion roots</td>
</tr>
<tr>
<td>Fenclorim</td>
<td>Long chain fatty acids</td>
<td>Rice</td>
</tr>
</tbody>
</table>

**Lipid synthesis**

Lipid biosynthesis has been detected as a site of action of thiocarbamates and chloroacetanilides. Dichlormid reversed inhibition of lipid biosynthesis that caused by EPTC in corn. Naphthalic anhydride and dichlormid countered thiocarbamates effects on fatty acid synthesis in corn. Application of fenclorim with pretilachlor reversed inhibition of acetate incorporation into total lipid that caused by pretilachlo). Wu et al. (2000) suggested that the herbicides pretilachlor and metolachlor may inhibit the synthesis of very long chain fatty acids or of the incorporation of unsaturated fatty acids into non-lipids of rice. Such inhibition might be counteracted by the safeners fenclorim, benoxacor and fluxofenim.
Epicuticular wax Degradation of epicuticular wax layer increases plant susceptibility to environmental stresses and increase rate of transpiration resulting in an increase of uptake of herbicides from soil. Herbicides and safeners interfered with cuticle formation in an opposite ways in corn.

Protein synthesis Inhibition of protein synthesis by thiocarbamates and chloroacetanilides has been reported. Sagral (1978) reported dichlormid reversed inhibition of protein synthesis caused by EPTC in corn. Han and Hatzios (1991) showed that safeners reduced inhibition of valine incorporation into protein in rice that caused by pretilachlor.

Other sites of action
Inhibition of the biosynthesis of gibberellins by thiocarbamates and chloroacetanilides was reversed by dichlormid in corn. Alteration of membrane permeability by EPTC and metolachlor was prevented by dichlormid and naphthalic anhydride in onion roots. Sagral (1978) reported that dichlormid countered inhibition of photosynthesis as a result of CO2 fixation caused by EPTC in corn.

Pro-safeners
These are chemicals that exert safening activity after transformation(s). An example of pro-safener is the compound L-oxothiazolidine-4-carboxylate (OTC) that transform into cysteine that is incorporated into GSH, and GSH in turn conjugates with herbicides.

Microbial safeners
Genes encoding for enzymes that degrade herbicides may be incorporated into appropriate plastids. Then introduced into bacteria colonizing roots or seeds of susceptible plants. This method has shown to be successfully to provide protection to corn against thiocarbamates injury.

In summary there is no conclusive mechanism explaining action of the available safeners. The suggestion that safeners act through a single mechanism unrealistic and the interaction of a series of steps leading to safening is likely. It seems that interference of safeners with GSH and related systems is the most common hypothesis trying to explain the mechanisms of action of safeners. Selected areas that need to be investigated further include:

(1) The mechanisms of safener action should be extended to the molecular level. Recent report examining structure–activity relationship between safeners and herbicides using comparative molecular field analysis is promising (Bordas et al., 2000).
(2) Effect of safeners on gene expression in plants and weather theses regulatory effects of safeners are related to the biochemical and physiological effects exerted by the antagonized herbicides is an area of research (Veylder et al., 1997).

(3) The physiological, biochemical and molecular interactions between herbicides and safeners on plants should establish which effects of safeners and herbicides are of primary or secondary importance and if the secondary effects are totally unrelated to safening activity.

(4) Do safeners conjugate with GSH. Is this conjugation catalyzed by specific GST enzymes or by the same GST enzymes that catalyze the conjugation of the herbicides? Dixon et al. (1998) reported that the safener dichlormid and the herbicide metolachlor enhanced expression of GST II subunit in corn.

(5) What is the role of mixed-function oxidases in the metabolism of herbicides whose effects are counteracted by safeners? Are they induced by safeners? Ohkawa et al. (1999) reported the involvement of cytochrome $P_{450}$ monooxygenases in the mode of action of safeners. Are other oxidative enzymes such as peroxynasenes, peroxidases, lipoxygenases, etc., involved in the metabolism or action of selected safeners.

(6) What is the impact of safeners on herbicide environmental fate. Their effect on herbicides metabolism by soil microorganisms, especially in soils where high herbicide residue may exist? What is the effect of that on the amount of the herbicide available to the plant, as well as the influence on the carry over problems, and their effects on herbicide enhanced degradation and to environmental consideration? Recent report ruled out effect of the safener dichlormid on photodegradation of the herbicide EPTC (Abu-Qare and Duncan, 2002).
Herbicide Interactions

Simultaneous or sequential application of herbicides, insecticides, fungicides, antidotes, fertilizers etc., is followed in a single cropping season. These chemicals may undergo a change in physical and chemical characters, which could lead to enhancement or reduction in the efficacy of one or more compounds. The interaction effects were seen much later in the growing season or in the next season due to build up of persistent chemicals or their residues in the soil. Knowledge on the interactions of various chemicals can be helpful in the formulation and adoption of a sound and effective plant protection programme. It can also help to exploit the synergistic and antagonistic interactions between various pesticides for an effective eradication of weed and other pest problems.

When two or more chemicals accumulate in the plant, they may interact and bring out responses. These responses are classified as additive, synergistic, antagonistic, independent and enhancement effects.

i. **Additive effect:** It is the total effect of a combination, which is equal to the sum of the effects of the components taken independently.

ii. **Synergistic effect:** The total effect of a combination is greater or more prolonged than the sum of the effects of the two taken independently. The mixture of 2,4-D and chlorpropham is synergistic on monocot species generally resistant to 2,4-D. Similarly, low rates of 2,4-D and picloram have synergistic response on *Convolvulus arvensis*. Atrazine and alachlor combination, which shows synergism is widely used for an effective control in corn. Other examples are 2,4-D+Dicamba; 2,4-D+Atrazine, and Amitrole+Ammonium thiocynate.

iii. **Antagonistic effect:** The total effect of a combination is smaller than the effect of the most active component applied alone. Eg. Combination of EPTC with 2,4-D, 2,4,5-T or dicamba have antagonistic responses in sorghum and giant foxtail. Similarly, chlorpropham and 2,4-D have antagonism. When simazine or atrazine is added to glyphosate solution and sprayed the glyphosate activity is reduced. This is due to the physical binding within the spray solution rather than from biological interactions within
the plant. EPTC with 2,4-D or Dicamba in sorghum; Simazine or Atrazine with glyphosate reduces the activity of glyphosate.

iv. **Independent effect:** The total effect of a combination is equal to the effect of the most active component applied alone.

v. **Enhancement effect:** The effect of a herbicide and non-toxic adjuvant applied in combination on a plant is said to have an enhancement effect if the response is greater than that obtained when the herbicide is used at the same rates without the adjuvant. Eg. Mixing Ammonium sulphate with glyphosate.

A considerable amount of research has been conducted to define interactions among herbicides. Herbicides applied in combination either preplant incorporated or preemergence generally increase the spectrum of weed control or the length of residual weed control. For example, pendimethalin is often applied in combination with alachlor, dimethenamid-\textit{P}, metolachlor, or \textit{S}-metolachlor to improve early season weed control. Alachlor, dimethenamid-\textit{P}, metolachlor, or \textit{S}-metolachlor can be applied with diclosulam, flumioxazin, or imazethapyr preemergence to enhance weed control with a single application. Combinations of preplant incorporated or preemergence herbicides currently registered for use in peanut have not been shown to increase peanut injury over either herbicide component applied alone. However, several herbicides that are no longer registered for peanut increased peanut injury when co-applied as compared to the herbicides applied alone (Wilcut et al., 1995).

In reduced tillage systems, herbicides are needed to control winter weeds and summer annual weeds that have emerged prior to planting peanut. These herbicide applications include glyphosate, paraquat, or 2,4-D alone or in combinations with other herbicides. Combinations of glyphosate and 2,4-D broaden the spectrum of weed control compared with each herbicide applied alone (Flint and Barrett, 1989a). However, in some instances, 2,4-D can negatively affect efficacy of glyphosate, but this interaction is typically noted only on grass weeds (Flint and Barrett, 1989b). Efficacy of paraquat is generally not negatively affected by 2,4-DB (Wehtje et al., 1992a). Glyphosate and paraquat can also be applied with herbicides that provide residual weed control. This approach is designed to control emerged weeds and provide residual weed control prior to and following planting (Wilcut et al., 1995).
Paraquat is often applied at peanut emergence or up to 28 days after peanut emergence. Other non-residual herbicides such as bentazon or acifluorfen plus bentazon as well as residual herbicides such as alachlor, iclosulam, dimethenamid-P, imazethapyr, metolachlor, or $S$-metolachlor are applied postemergence to broaden the spectrum of control. Injury associated with paraquat can be reduced by coapplication with bentazon. However, the chloroacetamide herbicides alachlor, dimethenamid-P, metolachlor, or $S$-metolachlor applied with paraquat can increase peanut injury. Diclosulam and imazethapyr did not affect injury potential from paraquat. Weed control with these herbicide combinations generally increases depending on the weed species and size of the weed. For example, bentazon and imazethapyr co-applied can increase control of emerged common cocklebur and yellow nutsedge, while control of annual grasses by paraquat can be reduced when paraquat is co-applied with bentazon. Residual control by chloroacetamide herbicides, diclosulam, and imazethapyr was not affected by paraquat applied alone or with bentazon.

Co-application of postemergence herbicides with efficacy against dicotyledonous weeds and sedges generally increases control of weeds or broadens the spectrum of control compared with components of the mixture applied alone. In contrast, efficacy of clethodim and sethoxydim, often referred to as graminicides, can be reduced when applied in mixture with herbicides that control dicotyledonous weeds and sedges. The interaction of bentazon and sethoxydim is one of the most notable examples of reduced graminicide efficacy caused by a herbicide that controls dicotyledonous plants and sedges. Annual and perennial grass control by sethoxydim is reduced by bentazon through reduced absorption of sethoxydim into grasses. The mechanism of reduced control is associated with physical interactions of the herbicides in the spray solution prior to reaching the target weed. Acifluorfen and imazethapyr also can reduce efficacy of clethodim and sethoxydim. In contrast to reduced grass control when these herbicides are co-applied, control of dicotyledonous plants and sedges is not reduced by clethodim and sethoxydim. Efficacy of clethodim can also be reduced by acifluorfen, acifluorfen plus bentazon, bentazon, imazethapyr, imazapic, lactofen, and 2,4-DB. The magnitude of reduced efficacy can be minimized or eliminated by applying the herbicides sequentially, increasing the graminicide rate, or applying more efficacious adjuvants. Grass species, plant size, and plant stress also can affect the magnitude of negative interactions. York and Wilcut (1995) reported that bentazon reduced control of yellow and purple nutsedge by imazethapyr.
Chloroacetamide herbicides can be applied postemergence without injuring peanut. While these herbicides provide residual control of grasses and some dicotyledonous and sedge weeds, they do not control weeds that have emerged. These herbicides can be applied with herbicides that have efficacy against emerged weeds. Dimethenamid-P and S-metolachlor did not reduce grass control by the graminicides clethodim or sethoxydim or the dicotyledonous and sedge herbicides acifluorfen, acifluorfen plus bentazon, or imazapic. However, visible injury caused by acifluorfen increased when acifluorfen was applied with chloroacetamide herbicides. Johnson et al. (1993) reported that injury from postemergence application of paraquat was not increased when following several chloroacetamide herbicides applied at planting, in contrast with injury observed when the herbicides were co-applied.

Compatibility of herbicides with other pesticides

Herbicide-Antidote Interactions:
The herbicide antidote interactions are antagonistic in nature. The antidotes like NA, R-25788 and CDAA reduce the toxicity of Herbicides like alachlor, EPTC and Butylate to certain plants (Crops).

Metribuzin activity on ivy leaf morning glory (Ipomoea hederacea) can be increased when the synergist PABA (picolinic acid t-butyl amide or MZH 2091) is included in the spray solution. Normally, ivy leaf morning glory is able to metabolize metribuzin via deamination followed by conjugation. However, in the presence of PABA, deamination is slowed and thus, ivy leaf morning glory is more susceptible to metribuzin.

Herbicide - plant growth regulator interactions
Prohexadione calcium is the primary plant growth regulator available for use in peanut. Efficacy of the herbicides acifluorfen, acifluorfen plus bentazon, bentazon, imazethapyr, imazapic, lactofen, and 2,4-DB was not affected by prohexadione calcium.

Herbicide- Insecticide Interactions:
Herbicide and Insecticides are often applied simultaneously or serially to crops within a short period. These chemicals are usually not harmful when used as per recommended practices. The tolerance of plants to a herbicide may be altered in the presence of insecticide and vice versa. The Phyto-toxicity of monuron and diuron is increased on cotton when applied with phorate. Similar effects were also observed on oats. Combination of Organo-phosphate insecticide and
Atrazine on phyto-toxicity appeared to involve an effect of the insecticides on herbicides absorption and translocation.

**Corn root-worm insecticides and sulfonylureas:**
The root-worm insecticide, terbufos (an organophosphate insecticide), is applied as an in-furrow treatment with corn seeds. This treatment enhances the activity of primisulfuron and thus, corn injury. Crop safety is lost because terbufos binds to cytochrome P450 enzymes in corn, so the crop is less able to detoxify the herbicide. To avoid this problem, growers were advised not to use an in-furrow type treatment or to use a new formulation of Counter called Counter CR (Controlled Release). Now growers can also use BT-resistant crops.

**Insecticides and propanil:**
Propanil activity on rice weeds can be increased with the addition of malathion (an organophosphate insecticide). Organophosphate insecticides can inhibit acyl arylamidase, the enzyme responsible for propanil metabolism. This approach also increases the likelihood of crop injury if too much enzyme inhibition occurs in the crop as well.

Timing of application of herbicides and insecticides overlap during much of the growth cycle of peanut. As with other crops, potential interactions between herbicides and insecticides applied in the seed furrow to control thrips and suppress plant parasitic nematodes can occur. Acephate and aldicarb applied in the seed furrow at planting did not affect injury potential of peanut following postemergence application of acifluorfen plus bentazon or bentazon; however, the insecticide phorate applied in the seed furrow enhanced visible injury associated with bentazon, although this injury was generally transient. Although interactions of nicosulfuron and pyrithiobac-sodium increased injury in corn and cotton, respectively. However, chlorpyrifos applied at planting did not affect peanut response to diclosulam, S-metolachlor, or flumioxazin applied preemergence or acifluorfen, acifluorfen plus bentazon, imazapic, or paraquat plus bentazon applied postemergence. Efficacy of graminicides can be affected by insecticides applied to peanut. Carbaryl and dimethoate applied postemergence in combination with sethoxydim reduced annual grass control; no adverse effect was noted when acephate was mixed with sethoxydim. Pyrethroid insecticides did not affect efficacy of postemergence herbicides.
Herbicide Interaction with Pathogens and Fungicides:

Herbicides interact with fungicides as the disease causing organisms. Dinoseb was known to reduce the severity of stem rot (White mould) in groundnut. Diuron and Atrazine which inhibit photosynthesis may make crops susceptible to tobacco mosaic virus. Where as diuron may decrease the incidence of root rot in wheat. Atrazine was found to have antagonistic interaction with the fungicide Dexon on many crops.

Similar to herbicides and insecticides, timing of application of postemergence herbicides and fungicides to control foliar and soil-borne diseases overlap considerably during the peanut growing season. Fungicides are applied beginning approximately 45 days after peanut emergence and can be applied until a few weeks prior to digging and vine inversion.

Efficacy of clethodim and sethoxydim can be reduced by co-application with copper containing fungicides or azoxystrobin, chlorothalonil, and pyraclostrobin. Fluazinam and tebuconazole did not reduce grass control compared with graminicides applied alone. Efficacy of herbicides that control dicotyledonous and sedge weeds is not generally affected by fungicides. As was noted for interactions of herbicides, weed species and size and plant stress can affect the magnitude of interactions between herbicides and fungicides.

Although not used in peanut, efficacy of glyphosate was not affected by azoxystrobin, pyraclostrobin, or tebuconazol. Weed control by metribuzin, rimsulfuron, and thifensulfuron-methyl applied to tomato was not affected by azoxystrobin or pyraclostrobin. However, pyraclostrobin increased tomato injury from thifensulfuron-methyl when co-applied. Chlorothalonil increased persistence of metolachlor in soil although cyproconazole, flutriafol, and tebuconazole did not affect dissipation of metolachlor.

Tillage and Cover Crop Effects on Herbicide Degradation

Management systems that include reduced tillage and cover crops are gaining popularity. These practices typically increase plant residues at the soil surface and organic matter in the surface soil. In turn, microbial activity is increased, and the soil develops a greater capacity to adsorb and retain many types of farm chemicals, including herbicides. Accordingly, tillage and cover crops variously affect the degradation of herbicides and their movement with surface water runoff and internal drainage.
Tillage

Herbicide degradation in soil is controlled by the interaction of several factors. These include non-biological properties (such as texture, organic matter and pH) and conditions (moisture and temperature) and biological conditions (numbers, types and activities of microorganisms). The main non-biological effect of tillage on herbicide degradation is probably increased herbicide adsorption onto soil solids because of increased soil organic matter with reduced tillage. This has been shown for several different cotton herbicides including Bladex (active ingredient, cyanazine), Cotoran (fluometuron), Prowl (pendimethalin) and Zorial (norflurazon), among others.

In some cases, increased adsorption of the herbicide in reduced tillage soils has led to its slower degradation and longer persistence, despite typically higher microbial populations and activities. But when the effect of adsorption on reducing the concentration of herbicide susceptible to microbial degradation is factored out, it appears that degradation would actually be faster in the reduced tillage soils. These calculations suggest that reduced tillage usually increases the rate of herbicide degradation. But from the practical standpoint of how much herbicide actually remains over time, there is no clear, general conclusion on how tillage affects herbicide degradation rates.

Cover Crops

As with reduced tillage, planted cover crops increase soil organic matter and microbial numbers and activities. Comparison of herbicide adsorption in conventionally tilled soil with and without cover crops has shown that buildup of soil organic matter because of the cover crop increases adsorption. But the differences in herbicide adsorption in reduced tillage soil because of the cover crop tend to be smaller since levels of organic matter are higher.

Given the ambiguous results for effect of tillage on herbicide degradation, it is not surprising that there is no clear trend for effect of cover crop. This is particularly true for reduced tillage soil with cover crops. In some cases, increased herbicide adsorption caused by increased soil organic matter has led to slower degradation in soil planted with cover crops than soil without a cover crop. Whether this occurs may depend on the type of cover crop and the biological and chemical properties of its residue. For example, while there was no difference in the degradation rate of
Prowl in no-till Gigger silt loam soil with volunteer native annuals or a wheat cover crop, degradation of Prowl was noticeably slower with a hairy vetch cover crop. Besides effect of cover crop on herbicide degradation in soil, interception of spray-applied herbicide by crop and cover crop residue in reduced tillage systems may affect herbicide degradation and weed control. For example, Cotoran is relatively easily washed off cover crop residue compared to Prowl. Consequently, interception of Cotoran by a dense layer of vetch residue may have a negligible effect on herbicidal efficacy. On the other hand, since Prowl tends to be retained by residue, little Prowl that is intercepted by plant residue may ever be transferred to the soil by rainfall. Instead, it is degraded by biological and chemical processes within the residue.

**Herbicide-Fertilizer Interactions:**
Application of fertilizer with herbicides is becoming increasingly popular in developed countries. No detrimental properties were observed when herbicide were combined with suspension of fertilizers. Application of complete fertilizer (Containing N,P and K) reduce the atrazine absorption by plants, thus reducing phytoxicity. Atrazine was more toxic in the presence of PK than in the presence of NP and NK. The addition of Urea or ammonium sulphate in 2,4-D and glyphosate increased the efficiency of herbicides. Boron and manganese are the primary micronutrients applied to peanut. Occasionally, these can affect herbicide performance. For example, efficacy of clethodim and imazethapyr was reduced by micronutrients for some, but not all, weeds evaluated (Jordan *et al.*, 2006; Lancaster *et al.*, 2005b).

**Herbicide and Soil Moisture or Irrigation Interaction:**
Adequate soil moisture in surface soil is necessary for germination of large population of weeds and their effective killing by herbicides. The adequate soil moisture increase the adsorption and translocation of soil applied as well as foliage applied herbicides which increase efficiency of herbicides in killing weeds. The excess moisture as well as soil moisture stress affect the absorption and efficiency of herbicides and heavy rains after application of herbicides may cause washing away of foliar applied herbicides.
Herbicide resistance is the major trait that has been engineered into crops and herbicide-resistant crops (HRCs) occupy the largest area under transgenic crops in the world. The relevance of HRCs in Indian agriculture, however, has been debated due to socio-economic and technical reasons. The problem to weed resistance to herbicides has provided an invaluable opportunity for visionary scientists to develop crops resistant to previously non-selective herbicides. Researchers explored ways to develop crops resistance to herbicides with the goals of improving herbicide selectivity, expanding weed control spectrum, as well as minimizing crop injury. HRCs can be classified as non-transgenic (traditional genetic methods of selection of resistance traits) and transgenic (genetically engineered). Non-transgenic HRCs were developed using conventional breeding techniques. The first such example is triazine resistant canola that was developed through a breeding programme in 1984. Thereafter, various methods such as microspore selection, seed mutagenesis, pollen mutagenesis, tissue culture, cell selection, and transfer from a weedy relative have been used for generating non-transgenic HRCs. Agronomic performance of non-transgenic HRCs, often, did not meet the expectations of growers and commodity groups. Scientists therefore began to look at alternative ways to develop HRCs as weed management tools to manage broad-spectrum of weeds.

Transgenic herbicide resistant crops

The initial efforts to develop transgenic HRCs using genetic engineering techniques resulted in the release of bromoxynil-resistant cotton in 1995 and canola in 2000 (Table 1). Bromoxynil is not a broad-spectrum herbicide, therefore, bromoxynil resistance cotton and canola were discontinued. The real turning point occurred in 1996-97 with the commercial release of glyphosate resistant (GR) canola, soybean and cotton. These crops allowed the application of glyphosate multiple times in the growing season without risk of crop injury. Additional GR crops, alfalfa, corn and sugarbeet were released to growers between 1998 and 2007. Glufosinate resistant canola, released as early as in 1995, did not catch on as well as GR canola. Glufosinate resistant cotton was commercially made available in 2004, but was inferior in yield as compared to GR cotton. A new generation of GR cotton was developed in 2006 that has enhanced tolerance to glyphosate while at the same time allowing in-season glyphosate applications during the reproductive phase of the crop. New GR and glufosinate resistant corn was commercialized for the first time in 1997 and was combined with GR corn varieties as a
‘double stacked trait’ in the mid 2000s. This allowed control of a broader spectrum of weeds with the two unique modes of herbicide action. Invariably, most transgenic cotton and corn HRCs on the market also carry insect-resistance traits (Bt trait).

Table 1. Current transgenic resistant crops and associated trait genes

<table>
<thead>
<tr>
<th>Crop</th>
<th>Resistant trait</th>
<th>Trait gene</th>
<th>First sale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alfalfa</td>
<td>Glyphosate</td>
<td>cp4 epsps</td>
<td>2005</td>
</tr>
<tr>
<td>Canola</td>
<td>Glyphosate</td>
<td>cp4 epsps and goxv 247</td>
<td>1996</td>
</tr>
<tr>
<td></td>
<td>Glufosinate</td>
<td>Pat</td>
<td>1995</td>
</tr>
<tr>
<td></td>
<td>Bromoxynil</td>
<td>Bxn</td>
<td>2000</td>
</tr>
<tr>
<td>Cotton</td>
<td>Bromoxynil</td>
<td>Bxn</td>
<td>1995</td>
</tr>
<tr>
<td></td>
<td>Glyphosate</td>
<td>cp4 epsps</td>
<td>1996</td>
</tr>
<tr>
<td></td>
<td>Two cp4 epsps</td>
<td>2006</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Zm-2mepsps</td>
<td>2009</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Glufosinate</td>
<td>Bar</td>
<td>2004</td>
</tr>
<tr>
<td>Corn/maize</td>
<td>Glyphosate</td>
<td>Three modified cp4 epsps</td>
<td>1998</td>
</tr>
<tr>
<td></td>
<td>Two cp4 epsps</td>
<td>2001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Glufosinate</td>
<td>Pat</td>
<td>1997</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + Glufosinate</td>
<td>Double stack</td>
<td>Not clear</td>
</tr>
<tr>
<td>Soybean</td>
<td>Glyphosate</td>
<td>cp4 epsps</td>
<td>1996</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cp4 epsps</td>
<td>2009</td>
</tr>
<tr>
<td></td>
<td>Glufosinate</td>
<td>Pat</td>
<td>2009</td>
</tr>
<tr>
<td>Sugarbeet</td>
<td>Glyphosate</td>
<td>cp4 epsps</td>
<td>2007</td>
</tr>
</tbody>
</table>

Genetic engineering of herbicide tolerance

Before the emergence of plant genetic engineering, options for selective crop protection against herbicides were limited. Specific herbicides could be used in the crops that were naturally resistant to the herbicide. In rare cases, resistance could be induced in crop varieties through mutations. For example, monocots are naturally resistant to triazine and hence triazine could be used as selective herbicide in monocot crops to control dicot weeds. Developments in plant genetic engineering and knowledge of biochemical action of herbicides on plants spurred innovative approaches to engineer crops to withstand herbicides. These strategies usually involve isolation and introduction of a gene from another organisms, mostly bacteria, which is able to overcome the herbicide-induced metabolic blockage. For example, tolerance to the herbicide glufosinate (Basta) is conferred by the bacterial gene bar, which metabolizes the herbicide into a
non-toxic compound. Glyphosate (another most popular herbicide) resistance is achieved by the introduction of either *Agrobacterium* gene *CP4* that codes for a glyphosate-insensitive version of the plant enzyme, EPSP-synthase, or *gox* gene from *Achromobacter*, which codes for glyphosate oxidoreductase in the breakdown of glyphosate. A number of other genes have been identified that can alleviate the herbicide action through various ways (such as detoxification, sequestration, etc.) and thus confer resistance to the plants carrying them. Thus genetic engineering technology has made it possible to tailor crop varieties to resist specific herbicides by introducing relevant genes. Consequently, the range of selective herbicides has now greatly expanded, wherein specific genotypes and varieties can be conferred resistance rather than generic crops displaying resistance to specific herbicides. These developments have provided the herbicide companies new opportunities to promote their herbicides through development and marketing of genetically engineered HRCs.

**Potential impacts (risks and benefits) of HRCs and the herbicides that are used with them**

Risks and benefits are very geography and time dependent. In the context of replaced herbicides and agronomic practices, the health and environmental benefits of the herbicides/HRC combinations that have been used are significant.

The only herbicides currently being used with HRCs, glyphosate and glufosinate, are more environmentally and toxicological benign than many of the herbicides that they replace. The effects on soil, aoir and water contamination and on non-target organisms are relatively small.

Soil erosion causes long-term environmental damage. Being broad spectrum, foliarly applied herbicides, with little or no activity in soil, glyphosate or glufosinate are highly compatible with reduced or no-tillage agriculture and have contributed to the adoption of these practices in the western hemisphere. This contribution to environmental quality by HRCs is perhaps the most significant one.

Transgenic foods are tested and evaluated for safety to a much greater extent than traditional foods that are usually derived by conventional breeding methods involving the transfer of many genes. New crops and new crop varieties of conventional crops are usually introduced without any testing for safety or nutritional problems related to genetics. In contrast, a rigorous safety or
nutritional problems related to genetics. In contrast, a rigorous safety testing paradigm has been developed for transgenic crops which utilize a systematic, stepwise and holistic approach. Foods and feeds derived from genetically modified crops are as safe and nutritious as those derived from traditional crops. The lack of any adverse effects resulting from the consumption of transgenic crops grown on hundreds of millions of cumulative hectares over the last 15 years supports this conclusion.

High levels of adoption of HRCs have dramatically increased the use of herbicides, with a following increase in resistant weeds and weed species shifts. Management of resistance weeds requires alternative strategies that should not rely solely on different herbicide mechanism of action. However, exciting new technologies such as new generation of HRCs are development or approaching commercialization in the next few years, which will help manage resistant weeds and reduce their spread.

All the potential environmental risks are reversible (even that of soil erosion) and are in most cases not exclusive to transgenic crops, except for those associated with flow of transgenes to other plants (the same species or other species). Little or no impact or risk is expected from the HRC transgene if it introgresses into wild populations, as it is fitness neutral. However, when the HRC transgene is linked with genes that could provide a fitness advantage in a natural habitat, introgression could be aided by elimination of competing plants of the hybrid by the herbicide. Over the long term, this could be the greatest risk of HRCs. Several methods could be used to prevent introgression, but more research should be done to discover and/or develop technology to prevent it.

**Future herbicide resistant crop technologies**

HRCs and other transgenic crops are here to stay. While the benefit of these crops are plentiful, there are certain inherent consequences, some obvious and a few not quite apparent. The most important issue requiring attention following commercialization of HRCs has been the evolution of resistance to herbicides in weed populations. The agrochemical industry, seed companies and related entities have invested most of their resources in development of the next generation of HRCs (and other transgenic crops) with the aim of diversifying the growers crop portfolio as
well as combating weed resistance by providing cropping technologies that allow application of more than one mode of action herbicides.

New HRC technologies currently under development are outlined in Table 2.

Table 2. Future transgenic herbicide resistant crops with earliest expected commercialization

<table>
<thead>
<tr>
<th>Crop</th>
<th>Stacked multiple herbicide resistant traits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cotton</td>
<td>Glyphosate + glufosinate</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + glufosinate + dicamba</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + glufosinate + 2,4-D</td>
</tr>
<tr>
<td>Corn/maize</td>
<td>Glyphosate + glufosinate + 2,4-D</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + dicamba</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + HPPD (4-hydroxyphenylpyruvate dioxygenase)</td>
</tr>
<tr>
<td>Soybean</td>
<td>Glyphosate + glufosinate</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + glufosinate + dicamba</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + ALS inhibitors</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + glufosinate + 2,4-D</td>
</tr>
<tr>
<td></td>
<td>Glyphosate + HPPD</td>
</tr>
</tbody>
</table>

**Worldwide use of transgenic crops**

Transgenic crops were first introduced in the 1990s. According to a 2010 database maintained by a non-profit environmental risk assessment institution, 60% (87 of 144) of all transgenic/biotechnological events reported involved herbicide resistance traits (CERA, 2010). All herbicide resistance traits that had regulatory approval did not result in commercialization and sales. In 2003, 67.7 million were planted to transgenic crops (both herbicide and insect resistance) in the world (Dill, 2005) and by 2010, the area increased to 148 million ha (James, 2010).

By 2012, 84.6% of all genetically modified (GM) crops worldwide carried herbicide resistance traits (144 mil. ha). Herbicide-resistant (HR) crops occupy about 59% of the 170.3 million hectares under GM cultivation globally, with GM crops with stacked traits (basically herbicide and insect resistance) covering 25.6%.

The cumulative area planted to transgenic crops from 1996 to 2010 exceeded 1 billion ha. An unprecedented 87 – fold increase in transgenic crop hectarage, from 1.7 million ha in 1996 to
148 million ha in 2010, makes transgenic crop technologies the most widely accepted in crop husbandry. Since 1996, the only year to year double digit (10%) growth in transgenic crop area was for 2009 to 2010. While the number of countries that planted transgenic crops increased to 29 in 2010 from 25 in 2009, the top ten countries each grew more than 1 million ha for the first time. Of the 29 countries growing transgenic crops 19 were developing and 10 were developed.

Among the HRCs, soybean was the most dominant transgenic crop in 2010, occupying 73.3 million ha or 50% of global area planted to transgenic crops. Among the traits, herbicide resistance remained the most planted trait. In 2010, herbicide resistance crops: soybean, maize, canola, cotton, sugarbeet and alfalfa accounted for 61% (or 89.3 million ha) of the global transgenic area (148 million ha). Stacked traits are increasingly becoming important for weed control and economic reasons. In 2010, eight of 11 countries planted stacked trait crops were developing nations.

While 29 countries planted commercialized transgenic crops in 2010, an additional 30 countries, totaling 59 have granted regulatory approvals for transgenic crops import for food and feed use and for release into the environment since 1996. The global value of the transgenic seed market alone was valued at $11.2 billion in 2010 with commercial biotech maize, soybean grain, and cotton valued at an estimated $150 billion for 2010 (James, 2010).

**Herbicide Development**

The agrochemical industry has been very successful in developing new herbicides. New chemicals with improved properties, especially providing significantly reduced use rates, and often new modes-of-action have been discovered, developed and launched for many different crops. This success has positively influenced agriculture as a whole. However, these days the introduction of new herbicides with either a new mode-of-action or novel chemical classes has slowed. Since 1991, when sulcotrione, an HPPD herbicide, was introduced in the marketplace, no new mode-of-action has been commercialized in Europe, while there were 10 new modes-of-action commercialized between 1970 and 1985 and five new ones between 1986 and 1991 (Schulte, 2004).
The high adoption of chemical weed control and the broad range of solutions already available to manage most weed problems are significant hurdles to the development and launch of new herbicides. Business potentials are influenced by the high technical and biological standards provided by existing herbicides, as well as the intense competition in the market place. Other factors adding complexity are agronomic, structural and technological changes, including the introduction of herbicide-tolerant crops, and the high costs of development for new active ingredients, mainly due to increasing regulatory requirements. In the light of increasing weed resistance to widely used herbicides, securing diversity in agronomy as well as weed management is a key to efficient crop production in future. In order to support this objective, new herbicides, preferably with new modes-of-action, will need to be discovered and developed.

However, the potential success of innovations also depends much on product price and cost. Operating a farm successfully is a complex task (Zoschke & Quadranti 2002). Farmers are under pressure, commodity prices for most agricultural produce are low and subsidies are under scrutiny. The change in real agricultural income per worker in EU-25 has overall declined by 6.6% in 2005. Farmers suffered most in Hungary, Slovakia, Spain, Portugal, Slovenia, France and Italy where agricultural income per worker declined by 19%, 15%, 12%, 11%, and 10% respectively. In order to reduce costs, farmers might think of cutting product rates, applying less than recommended on the label. However, this not only puts the success of a weed control programme at risk, but potentially favours the build-up of resistance of herbicides to weeds. Likewise, the intensive use of a single herbicide is likely to promote herbicide resistance development, but at an accelerated pace, and especially so, if used in various crops grown in the same rotation, as currently experienced with glyphosate in herbicide-tolerant crops in the United States (Powles & Preston 2006). Applying full use rates, as recommended, is a critical success factor to keep herbicides effective for as long as possible.

For environmental reasons, short residual, low water solubility and highly soil-adsorptive compounds are preferred. On the other hand, herbicides with a certain persistence and good soil mobility would make good agronomical sense for many crops. The chemical industry has adapted screening methods that allow the elimination of environmentally critical compounds early in the screening cascade. But the optimization towards both acceptable weed control and
acceptable leaching potential continues to be a tricky decision-making process in the early screening stages.

**Impact on the herbicide portfolio**

With the invention and introduction of selective herbicides in the late 40’s, a constant flow of new active ingredients provided crop producers with highly effective weed control tools. As a result, there are almost 400 world-wide herbicides covering more than 17 different modes of action. The average global government regulatory approval rate for novel herbicides has been 10-12 active ingredients per year over the last three decades. This figure has been slowing in recent years as a consequence of increasing demands in product performance as well as higher regulatory hurdles. HT crops have also contributed to this trend. Many companies have reacted by shifting more of their research resources into biotechnology activities. Monsanto doesn’t screen for new agrochemicals anymore and DuPont’s research efforts are primarily breeding and trait driven.

**The Innovation Deficit**

Today, herbicides involving only 6 different modes of action occupy approximately 75% of the total herbicide market, and most of them have been around for more than 10 years. The discovery rate of active ingredients has slowed significantly. The most recent discovery of a commercially relevant mode of action dates back to the early 80’s (sulfonylureas). There has been a steep decline in the number of patent applications and publications for new active ingredients. In 1990, more than 250 herbicide active ingredient patent applications were filed. This number dropped to less than 60 in 2006, and does not look as if it will rebound. This decrease is a reflection of not only the reduced number of companies with activity in herbicide discovery, but also as a result of the ever decreasing success rate of those companies still willing to invest. Whereas 20 years ago it was sufficient to screen 10,000 new chemistries in the greenhouses of an ag-chem company in order to discover one novel herbicide, this number has increased today to several hundred thousands. As a result of regulatory demands and due to the high standards in the market, costs for the development of new molecules have soared.
Objectives of agrochemical companies in the development of new herbicide technologies

Companies developing new plant protection molecules using conventional chemistry search for compounds with certain characteristics.

1. Broad spectrum efficacy against grass and dicot weeds (to reduce the complexity and number of compounds required per unit area)
2. Flexibility for pre- and post-emergence use (to increase the utility and opportunity for individual molecules to participate in more than one market segment)
3. Outstanding selectivity (demanded by customers)
4. Modes of action suitable to complement the existing herbicide rotation regimes (for the management of weed resistance)
5. Low application rate (small environmental footprint and better cost position)

Existing herbicides which do not fulfill the requirements of modern agriculture will be replaced, and new ones falling short will simply not be developed.

New Product Development: from the Laboratory to the End User

The mission of product discovery and development is to find new pesticide products that can be used safely and effectively, with true profit potential for the manufacturer.

It costs an average of $180 million—plus the cost of a new manufacturing facility—to introduce an active ingredient to the marketplace; so marketing professionals continually seek products that can be used successfully on multiple crops in various geographic situations. Multiple formulations of a pesticide widen its use capabilities and increase the manufacturer’s profit.

Methods used to discover new pesticides and related chemistries differ among manufacturers, and sometimes even within a company. It is important to realize that new compounds come from three types of research: empirical, in which researchers look for new chemical entities; the ‘me-too’ route, in which researchers look for loopholes in patents or methods so that products similar to those already on the market can be launched; and the mimic, where the researcher studies the way natural products work (eg how enzymes fit into active sites) and then tries to make molecules that act similarly. Computer modelling plays an increasingly large role in these development routes.
The process by which a molecule is discovered, developed, and formulated into a pesticide product is tenuous and risky. From the examples presented, it is easy to understand the importance of seeking input from a diverse group of specialists, scientists, and analysts throughout the process. Even so, fewer than one in 140,000 molecules tested for pesticidal properties ever becomes a registered pesticide product.

It takes an average of one to two years to determine if a molecule has useful pesticidal properties. A three-year predevelopment phase follows for compounds that show sufficient promise, then a three-year development phase and a commercial launch phase. New pesticidal chemistry is identified by altering existing compounds or discovering new ones.

**The team of experts**

Making a new compound is an important job for a chemist, but before a new compound can be brought to the market there is much work to be done. Marketing a product is time consuming and it is important that this aspect is linked to the actual production of a new herbicide.

**Research chemists**

The company has well equipped laboratories with a good analytical chemistry section (with instrumentation such as mass spectrometers, and infrared, ultraviolet and nuclear magnetic resonance spectrometers) and a good science library. The chemists have had experience in suggesting possible chemical compounds and have developed skills to produce and isolate them. The chemists have also worked with biochemists to suggest possible compounds based on shape and stereochemistry to fit appropriate receptor sites by basing their products on more theoretical considerations. At a more practical level, they are also used to making a number of new compounds based on a particular structure, and then screening them all to see if any of them have the required properties.

**Economists**

As a commercial company any product that is developed must be economically viable. The economist knows about the economics of producing of crop protection products and their modes of application.
Toxicologists
If the toxicity of any potential herbicide exceeds acceptable levels towards animals it is abandoned. Approximately 50% of biologically active compounds may be abandoned for this reason. The specialist is well versed in acceptable toxic levels and risk factors.

Residue environmental metabolism (REM) Specialists
Concern for the environment is a serious and important issue. REM specialists are experienced in checking for any residual compound in the soil, run off water, and in the plant itself. These specialists are knowledgeable about procedures to assess the rate of biodegradability and to determine how long the compounds remain in the plant or the soil. They know what types of products are formed when the residue is broken down in both the plant and the soil: these are likely to be different because enzymes will cause decomposition in the plant, while microorganisms break the compound down in the soil.

Biologists/botanists
Any potential herbicide will need to be thoroughly tested, first under laboratory conditions by using samples of the weed, and then the crop to be protected. The biologists, botanists or botanist-cum-biochemists are experienced in such screening methods. Generally, both plant pathologists and entomologists (who study insects) are required during trialling to ensure that the effectiveness of the herbicide is tested against all pests.

Patent officers
It is important to protect any new product so that competitors cannot ‘steal’ the compound or idea before the company has had ample opportunity to cover the costs of research, development and marketing; and provide sufficient rewards for its investors and shareholders. A patent is likely to be taken out about three to four years after the first synthesis and, because the patent has a finite lifespan (20 years), it is important to determine the best time to take out the patent and describe the area of activity to give good protection whilst allowing for further development. Patents usually take one – two years to be awarded after an application has been submitted in their country of origin. International patents take about two years to be awarded, but in both cases the award is from the date of submission rather than the date of award.

Agronomists (agriculturalists)
Field testing under conditions that approximate those experienced by the farmer is also an important part of the development of any new crop protection product. It is possible that a
potential product tested with plants under laboratory conditions (where they are perhaps not as hardy as in the open) gives different results when used on plants in the fields. Field experiment officers therefore need to collaborate with biologists/botanists and environmental officers.

**Process research chemists**

The process research chemists consider the most efficient and cost effective production routes for the manufacture of the potential product. They are aware of production methods that are protected by patents and the need to explore alternatives, and that the new routes can be protected by patents.

**Registration specialists**

Before any new compound can be put on the market it has to meet national requirements for safety. The registration specialist prepares careful records of testing programmes which are presented to the government registration office for critical evaluation.

**Formulation chemists**

Applying the herbicide effectively is a difficult problem. The problem has been likened to spreading 500 g of lard over the turf in Wembley stadium so that every blade of grass is covered. How should the active herbicide be applied eg as a solution in oil or in water? The formulation chemist behaves like a pharmacist making up a prescription where support ingredients, method of dispensing and the best method for releasing of the active ingredient need to be considered. Hence the inclusion of surfactants, wetting agents, stabilisers, and possibly buffering agents, must be considered as well as their relative quantities. Micro-encapsulation, whereby the active ingredient is put into a capsule that slowly disintegrates, is another technique that might be used by the formulation chemist.

**Marketing specialist**

Next we need to consider the marketing of the product. Farmers are unlikely to use a new product unless they are convinced that it is effective in terms of: controlling the pest (weed); cost; safety for the crop, animals and workers on the farm; and that it is environmentally acceptable.

**Manufacturing specialist**

Once the decision has been taken to support production of a new product, the production chemists will advise about the best synthesis route, but an expert on manufacturing is also required. He or she will have to decide where and how to build a manufacturing plant, taking
into account all the factors that will affect the smooth and efficient operation of the plant once it is finished. In many cases the best formulation is as an emulsion. Decisions then have to be taken on marketing the product. How should it be presented to the consumer? When is it best to launch the product, particularly if the market is seasonal? How and where should the product be advertised?

**Innovation of Existing Chemistry**

The goal of companies that choose to develop new products from existing compounds is to enhance certain critical characteristics—spectrum, residual qualities, selectivity—that will lure customers. Scientists modify compounds to improve their efficacy, spectrum of control, and chemical profile. The pyrethroid insecticides are good examples.

The first synthetic pyrethroid was based on the structure of pyrethrum derived from chrysanthemums. Useful innovations of that original molecular structure have been created to increase soil persistence and decrease toxicity to nontarget organisms.

**New Discoveries**

The pesticide industry must continually introduce new products to maintain long-term profitability; and these breakthrough products bring premium prices. Products developed from new chemistries (versus those adapted from existing pesticides) generally are easier to patent, provide longer-lasting value for the manufacturer, and increase customer satisfaction. Customers do not choose one product over another solely because of its novelty, but the manufacturer usually profits by providing the customer an alternative he has sought.

**Compound Synthesis and Acquisition: Where Do the Molecules Come From?**

Promising new chemistries can be found various ways. Manufacturers may purchase, sell, or trade molecules from other commercial laboratories, pharmaceutical companies, and universities.

Other molecules may be selected by computer on the basis of characteristics determined internally. These may be targeted materials about which a scientist has read in a journal or learned at a meeting; or they may be natural molecules accessed through agreements with universities or companies that specialize in unusual natural products.
Microbes are collected from the bottom of the sea, from coral reefs, and from soil profiles, plant extracts, and numerous other locations worldwide. Pharmaceutical companies have collected, grown, and cataloged microbial populations for more than a hundred years. Penicillin is one of the best-known products originating from microbial organisms. Pesticide chemists are interested in testing chemicals that microbes release into their environment, as they grow, which alter their surroundings (e.g., chemicals used to eliminate their competition). The pesticidal properties of chemicals released by microbes are variable, depending on type of growth media, temperature, humidity, and light regimes. Pesticides that originate from microbial organisms are important sources of new and unique products.

**Designer Molecules: Modeling of Target Sites/Building Structures in the Laboratory**

The scientific knowledge necessary to build molecules with pesticidal properties can originate from proprietary research conducted by a company. This knowledge provides a basis for biochemists, chemists, and computer modelers to design molecules that have the desired efficacy, safety profiles, and physical and environmental properties.

Scientists generally understand which metabolic sites in weeds, insects, and diseases are most vulnerable; that is, where to target control. Modelers create a three-dimensional visualization of the structure of an enzyme or receptor to determine how bioactive molecules interact and inhibit the target enzyme. This visualization can be used to propose new molecules for better efficacy. Modelers sometimes can design a new molecule based entirely on a computer model of how it will interact at specific target sites.

Conversely, by predicting which molecular structures may interact with target sites known for becoming resistance mechanisms, modelers can help companies avoid the development of molecules prone to rapid development of pest resistance.

Especially significant among other properties of candidate molecules that may be altered with computer techniques are those that can improve environmental or human safety. Environmental chemists can move functional groups within the molecule in an effort to fine-tune its environmental characteristics. For example, a chemist may be able to change molecular properties so that a pesticide which previously leached from the soil becomes more tightly bound in the soil.
Genomics promises to speed the process by which new modes of action are discovered. By unraveling the genetic code of plants, insects, and diseases, chemists may discover specific sites sensitive to interruption by novel pesticides.

**Computer Screening: Does the Molecule Meet the Company’s Selection Criteria?**

The process examines hundreds of thousands, perhaps millions, of molecules in the search for a dozen or so promising candidates. Because the discovery process is so costly, scientists must eliminate questionable molecules as early as possible and focus their attention and resources on marketable compounds. Very few ever become viable products.

Computers are effective, efficient tools for sorting, selecting, and discarding millions of molecules offered for sale by vendors. Companies can purchase electronic lists of molecules (virtual compounds) and their structures. The computer programs generate information on molecular weights, functional groups, and other chemical attributes for each molecule, based on its structure.

The molecules on the electronic list are evaluated against a series of selection criteria (established by the company) based on product goals, technical attributes, and the company’s experience. This virtual screening is quicker and less expensive than biological screening.

The selection criteria are known as filters or screens; they eliminate molecules that do not meet desired specifications. For instance, one screen might reject molecules that contain heavy metals; another filter might select for physical properties required for mobility in plants. In this manner, molecules are screened for specific structures, functional groups, and physical properties; molecules that do not meet the criteria are rejected.

The computer sorts through thousands of molecules and assigns a selection score to each, based on the filters it passes. Ultimately, the computer classifies a few into a small subset that is reviewed by synthetic chemists, biochemists, modelers, and market analysts; this is the final quality control procedure before selecting molecules to order from the vendor.

Evaluation of the chemical library is a very generic sweep of potential pesticides. Many of the molecules eliminated from consideration have pesticidal properties, yet it would be too costly and time consuming to test each one for
Advances in Weed Management

biological activity. Pesticide manufacturers are not interested in finding just another pesticide; they are searching for the pesticide that meets or exceeds the needs of their customers.

**Detection of Biological Activity: Does the Molecule Show Any Activity?**

Biological activity is determined for molecules that pass the screens. Most molecules do not demonstrate biological activity at reasonable doses and are eliminated quickly. Methods and criteria used to screen these molecules vary, but most companies use “high throughput screens.”

High throughput screens are highly automated tests that use typical analytical methods to measure activity. The activity may take the form of chemical reactions such as color changes and new products.

Other methods to test the activity of a compound involve the use of an insect, weed, or disease organism. The procedure might be as simple as placing an insect larva in a well (small container) with food containing the molecule. A technician monitors and records the larva’s responses to the insecticide; scientists may examine the test for several days to see if the larva is affected.

High throughput screens often are yes-no filters that provide qualitative results. Assuming thousands of molecules are entered into a high throughput screening, fewer than 100 show activity suggesting they are worthy of further research.

**Detection of Biological Activity: Does the Molecule Have Activity on Whole Organisms?**

Molecules that pass the computer and high throughput screens have desirable chemical characteristics and a level of biological activity warranting further investigation. Since weak molecules sometimes can be modified to become more active, chemists test them against “indicator” insects, weeds, diseases, or other organisms known to be sensitive to low levels of biologically active pesticides. The common theme among indicator organisms is that they must be cheap to produce uniformly in large numbers, and they must be easy to rear in the laboratory.

Fewer than 10 out of 100 molecules advance for further evaluation.

**Screening for Herbicides**

Lemna is an aquatic plant used by many companies for assessing bioactivity; it is very sensitive to exogenous chemicals. Another plant used in genetic research is *Arabidopsis thalania*, a well-
known relative of mustard and oilseed rape. Other plants used as sentinel species include common weeds such as foxtail, velvetleaf, lambsquarters, and waterhemp.

**Developing Analogs: Can the Molecule Be Improved?**

The manufacturer is interested in the commercial potential of the few molecules that pass the early phases of testing. A team of scientists reviews each compound to determine if it is unique enough to warrant continued investigation. Molecules that pass this review are tested further; chemists look for ways to alter each molecular structure to improve efficacy or reduce environmental impact.

If the biologically active molecule has a mode of action for which the chemists can design a three-dimensional structure, the better the modifications would fit the target site. Chemists can use three-dimensional structures of the target site to screen new targets on the computer to make sure the desired synthetic changes will not decrease binding to the target site.

For example, a biologist might determine that a molecule affects only a certain mildew species in the fungal screens, but a broader spectrum of activity is needed. The team then might ask questions such as, We have limited spectrum, so what is limiting its activity on other fungi? It could be that the molecule is not mobile; that is, it does not move to untreated portions of the plant. Another possibility is that the pest metabolizes the molecule rapidly after application. Key questions seek to determine how the molecule might be modified to make it more effective.

Molecules may demonstrate unfavorable environmental impact, be phytotoxic to the plant, and/or have toxic characteristics. Chemists may be able to develop analogs (different forms of the same molecule) with decreased toxicity to humans, the host plant, wildlife, and the environment; or they may ask the vendor if there are specific analogs available for purchase. A dozen chemists may be assigned to develop as many as 500 analogs of a single molecule.

Chemists begin by looking for structures similar to the successful candidate compounds. They can alter the chemical characteristics of a compound by making slight changes in the structure through modification of its constituent elements. Generally, the more adaptable the molecular structure, the more opportunities there are to find the right combination. The discovery team eventually focuses on the most promising analog, bringing along three to six secondary analogs.
as backups. The secondary analogs sometimes are substituted for the lead analog once factors such as cost, performance, and product registration criteria are considered.

**Initial Greenhouse Screening: Will the Molecule Control Key Market Pests?**

The screening process moves to the greenhouse for detailed testing of a few molecules in a simulated real-world situation. Generally, only one to five molecules remain of the thousands initially evaluated; but sometimes greenhouse screening may involve higher numbers, especially for analog comparison.

A single chemist may spend weeks or months synthesizing enough of the compound in the laboratory for testing in the greenhouse. The discovery team then begins to test the molecule against organisms that correspond to some of the pests identified in the product marketing goals. If the goal is to control weeds in wheat, perhaps a dozen key weeds normally found in wheat are used as indicators of real market value.

Many molecules fail to advance beyond this point, for a wide variety of reasons:

- Activity observed during initial screening is not present in greenhouse studies.
- Control is limited to a very narrow range of pests or a poor pest spectrum.
- Phytotoxicity occurs in the crop species.
- Control rates are too high; the product would not be economically viable.
- Low water solubility or other undesirable chemical characteristics make the product difficult for applicators to use.

Usually, only one or two molecules justify more detailed greenhouse studies.

**Exploratory Tests Describe the Molecule’s Nontarget Toxicity**

During this segment of the evaluation process, the molecule is examined for its acute toxicity to laboratory animals, its potential to leach or run off into surface water, and its ecotoxicological profile for wildlife. Manufacturers use these probe studies to provide a conservative estimate of the benefit/risk profile of a molecule or a series of chemistries.
These exploratory tests are conducted only to identify and describe, in general terms, the toxicological parameters and environmental characteristics of the molecule. Candidate compounds are administered to test animals (e.g., rats and mice), and the dosage is calculated to determine the range of toxicity. Environmental chemists also might conduct environmental fate studies to estimate the persistence of the molecule in soil and water, its fate in the treated plant, and its soil adsorption properties.

These exploratory tests are early indicators that the molecules advanced to the greenhouse will or will not need to be altered to reduce potential adverse effects to humans, wildlife, and water.

**Advanced Greenhouse Screening: How Does the Molecule Handle the Real World?**

The desired outcome of more advanced greenhouse studies is to describe product performance in a simulated real-world setting. These studies identify effective rates and the spectrum of pests controlled at those rates. The rate that provides control of target weeds in early greenhouse tests often becomes a benchmark by which the discovery team calibrates three or more rates at higher and lower levels. An intermediate rate may be 1/10 of the benchmark dose, while the lowest dose may be 1/100 of the benchmark. Variations in the treatment rate allow researchers to see how pests respond to changing rates at different life stages and under different environmental conditions.

Application methods also change as the molecule moves to the more advanced greenhouse studies. Instead of applying the molecules through a syringe or hand sprayer, a track sprayer is used to apply them in “field normal” dilute solution. Greenhouse researchers might test the activity of potential insecticides against different insect growth stages, or they might determine how well potential herbicide compounds control weeds at different growth stages. These studies help researchers identify effective application rates, timing, and the most sensitive pest and crop stages.

**Initial Field Testing: Does the Product Respond Under Field Conditions?**

Ultimately, only one or two molecules of the hundreds of thousands initially screened for pesticidal properties warrant field testing. The true test of these few surviving molecules, now called product candidates, is how well they perform under field conditions. The volume of active ingredient necessary to conduct field trials and additional toxicological and ecotoxicological studies at this point in testing increases from milligrams (ounces) to kilograms (pounds). Since
there is no manufacturing plant for these new compounds, the production of quantities to support field trials usually is accomplished on a pilot scale by the company. It can be very time consuming and expensive.

Acute and long-term environmental and toxicological studies, including application and user exposure studies, are conducted at this time. Field evaluation of a molecule is similar to the tiered testing approach used to screen compounds in the laboratory and greenhouse. Products proceed through initial, first-year field testing on limited weed, insect, and disease pests to more complex studies involving more pest species, different soil types, and wider geographic distribution.

Companies use various approaches and protocols to conduct field tests. They may have numerous field research facilities, strategically located around the world, where they test the compounds under different soil and weather conditions and on pests native to the geographic area. Companies also use academic institutions and private research facilities for this screening.

**Small Plots Artificially Infested with the Pests**

The protocols for the first field tests and the final greenhouse tests are nearly identical. The principal question addressed by field trials is whether the results are similar to greenhouse results. Researchers are specifically interested in whether environmental stress interaction with pests, crops, and molecules will produce undesirable effects such as increased crop injury or reduced pesticide efficacy or persistence.

Due to limited quantities of the sample, initial field trials are conducted on relatively small plots (e.g., 5 x 25 feet) and replicated two to four times. Field scientists are reluctant to rely on naturally occurring pests to infest the plots, due to this limitation, and often artificially infest the plots with insects, weeds, and diseases produced off-site. Irrigation and the use of untreated buffer rows and trap crops improve the probability of successful trials.

Indicator crops often are planted in plots known to be pest-free to prevent interference from nontarget pests. For example, it would do little good to start a trial for control of late-season aphids on corn if the crop were likely to be destroyed or seriously damaged by cutworms, first. After the plot area has been infested artificially with the desired pest and the pest has been given time to become established, backpack sprayers are used to apply the experimental compound at
predetermined times and rates. For a disease, the crop may be inoculated with the pathogen before or after pesticide application, depending on how the product is expected to be used (preventive or curative). For weed evaluation, the soil may be sterilized to kill seeds in the soil. Field researchers then plant rows of specific weeds (e.g., foxtails) and crops in one direction, and the test compound is sprayed perpendicular to the plantings.

Intensive data collection begins after the treatments have been applied, with focus on spectrum of control, rates necessary for effective control, and crop injury. Depending on the targeted pests, field researchers can evaluate

- the percentage of pests controlled.
- which pests are controlled.
- the length of the residual effect (if any).
- how quickly the pests are controlled.
- crop injury from the treatment.
- how the molecule compares to existing company and competitive products.
- the percentage of crop plant damage from insect feeding and/or disease formation.
- how performance is influenced by soil characteristics and environmental conditions.

These data may be collected visually or by counting and measuring. Plots are observed on a regular basis over a period of days, weeks, or months, depending on the type of pesticide being evaluated and the object of the trial.

In some cases, treated plant parts are removed from the field and infested in the lab. These field/lab bioassays often are used to test early stage insecticides. Generally, these tests are conducted on a crop destruction basis; yields can be taken but not used or sold.

**Data Analysis: What Will the Product Do and Not Do?**

The research team reviews the strengths and weaknesses of the product following the field test cycle, taking considerable time to statistically analyze the tremendous amount of data collected. At conclusion, the development team will have determined
• which crops and pests the pesticide will be used on, worldwide.

• whether crop safety and selectivity are issues.

• how pests respond to various product use rates.

• whether product performance is reliable.

• how field performance of the molecule compares to that of competitive products and whether it satisfies the company’s internal standards.

**Predevelopment Stage: Making the Commitment to Move Forward**

Discovery phase research, which typically takes four years, is followed by the predevelopment stage. At this point, the company must decide whether to continue research on the few viable experimental compounds that remain. It requires millions of dollars to commit a compound to multi-year toxicological and environmental studies essential to registration by EPA and foreign governments.

The product efficacy in fields is fairly well determined by this point in the testing process. What remains to be determined is whether the molecule shows enough potential to generate profits for the company.

Experts from numerous departments within the company are asked to work collectively to build a business case. Financial predictions for the molecule are based on the following questions:

• What will the customer pay for the product?

• What kind of short- and long-term market share can we expect from the product?

• How much sales revenue will the product generate?

• What will be our expenses, including promotion, advertisement, and demonstrations?

• What will be the dose rates by country, crop, and pest?

• What quantity of active ingredient will be needed?

• Are there unique manufacturing problems associated with the product?

• What will be the cost per unit to manufacture the product?
• How much will the manufacturing plant cost to build and maintain?

• What will it cost to develop the molecule globally?

Biological data from the laboratory, greenhouse, and field are summarized by technical managers as a set of technical assumptions that will drive market predictions. A number of people representing discovery, marketing, manufacturing, regulation, and safety participate.

Technical assumptions are given for each crop on which a herbicide is expected to be used. For example, technical assumptions for a herbicide candidate might be as follows:

• The compound will control weeds a, b, and c in rice when applied at 2 ounces per acre with a level of control nearing 95 percent.

• The compound results in five percent injury to rice, with no expectations of crop rotation restrictions.
  • Probability for weed resistance to the molecule appears low.
  • Environmental and toxicological profiles appear to conservatively meet expectations of regulators.

Marketing professionals turn technical assumptions into sales projections. For example, controlling weeds a, b, and c should result in the product capturing 10 percent of the market for rice herbicides. Marketers and financial planners use technical attributes developed specifically for each crop to analyze and predict how much of the revenue the crop will contribute to the total projected worth of the molecule over its expected market life.

Making the Case to Management: Benefits/Risks of Advancing a Molecule

The case for moving a molecule forward is presented to upper-level company managers. Presenters of the data make recommendations regarding their estimated success potential of the compound and its importance to the company. They present the unique characteristics of the product: what it does; the markets in which it will be positioned; its expected financial returns; and the assumptions on which they based all calculations, estimates, and predictions.
Management expects complete details, backed by documentation, with particular interest in whether field or greenhouse studies raised any red flags; that is, whether there were any inconsistent results that might preclude the product from success in the competitive market.

Management now has a difficult decision to make. Not only does the cost of research, discovery, and development rest on their decision, but the costs of further development and marketing present added risks. They can choose to halt development and shift their investment dollars to more promising pipeline products; or they can advance the compound to registration and gamble that it will generate enough revenue to cover its own costs and those of numerous discontinued projects as well. Another option would be to put the project on hold until additional tests are completed and questions raised by data insufficiencies are answered satisfactorily.

**Management's Decision to Move Forward: Completing the Field Evaluations**

During the final years of field tests, researchers simulate grower practices in terms of application and crop culture. Experiments are expanded geographically. Application rates and timing are more defined. Field research at this point examines tank mixture with other pesticides, tolerance of different crop varieties, new formulations, control of secondary pests, crop rotation issues, and secondary market uses. Comparison to competitive products becomes more intense.

Field testing in the late stages of product development lasts three or four years and usually is conducted in 10-foot by 30- to 40-foot plots; three or four replications are performed at all locations to allow thorough statistical evaluation of the data. Field researchers might use a trap crop to attract insects or establish diseases in the area; it normally would be placed in the middle of the crops being evaluated. The use of indigenous pest pressures where multiple flushes of weeds or insects may occur during the crop cycle allows more realistic testing of the product. Additional field studies are used to refine existing information on the molecule.

**Refining the Rates**

Refined product rates used in these tests are based on the population density and growth stages of pests present. For example, the dose range applied in initial field studies may have been 0.1 to 1 pound per acre, as indicated by greenhouse trial results and limited early field tests. Additional field tests may refine the effective range to between 0.2 and 0.5 pound per acre. During follow-up field studies, project coordinators evaluate application rates to determine the optimal use rate
of the product, often with a variety of optimized formulations. The lowest effective rates may reduce application costs, but these results must be balanced against product reliability and company reputation.

Testing on Other Pests

Certain information in earlier field studies may have been collected on unplanned, incidental, or secondary pests. As field-testing moves forward, work may be expanded to develop detailed information on such secondary pests. Broadening the pest spectrum facilitates comprehensive labeling, which adds value for the customer and the company.

Evaluating Timing of Application

The timing of pesticide application is critical. Various experiments are conducted to evaluate pest response to the candidate product based on application timing. Application timing evaluations for weeds include preemergence, early postemergence (weeds 3–4 inches tall), and later postemergence (weeds 6–8 inches tall) trials. The timing of application for insect control is evaluated against both small and large larvae. Evaluation for diseases requires application before and after infestation to determine whether the molecule has curative and/or preventive properties. All of this information is critical to label wording that will provide customers the latitude to use the product to its fullest advantage.

Identifying Soil Type Responses

The performance of some molecules is affected by soil type. Some bind tightly to soil particles while others hardly bind at all. Variation in binding characteristics involves organic matter, clay content, pH, etc., and determines whether application rates will have to be adjusted. Soil type also may influence the persistence of the compound in soil. These factors are critical to pesticide performance and environmental safety.

Determining How to Position the Product

Company business managers and agricultural scientists meet to discuss the experimental product once field testing is complete. They offer insights on continually evolving customer needs, competitive products, and steps the company can undertake to make the product more attractive.
to customers. Their input is used to build a business model and plan for the experimental product.

**The Make-or-Buy Decision**

The company that decides to market the product commercially must decide whether to make the compound or buy it. If the manufacturer owns facilities with the necessary capacity, equipment, and personnel—or if it is feasible to build a new plant—the decision may be to make the product. If not, production may be outsourced to another company.

**Writing a Product Label**

A label can be written and submitted to EPA before a marketing decision is made. Assembly of use directions for the product begins during the predevelopment phase. For instance, information from field tests may show that a herbicide label should allow application only as a postemergent treatment for controlling grasses up to 4 inches tall. About 80 percent of the label information is prepared during field testing; the remainder is written according to EPA-required human toxicological, ecotoxicological, and environmental fate testing data.

**Writing a Marketing Plan**

Preparing the marketing plan is the next step. It can include the following sections:

- Market assumptions
- Competitive product strengths and vulnerabilities
- Internal assumptions and estimates on development and manufacturing costs, registration timetables, and pricing
- Distribution channels
- The “Five Ps of Marketing”: product, placement, price, promotion, and positioning
- Technology transfer strategy to train company representatives and customers on proper use and handling
Development Stage: Getting the Product Registered

The principal focus during the development stage is to collect and summarize data and write official reports to provide EPA the registration information required by the Federal Insecticide, Fungicide, and Rodenticide Act as amended by the Food Quality Protection Act. Registration submission and approval normally begins six to eight years into the development process and ends in years eight to ten.

Commercial Stage: Preparation for Product Launch

While the product is undergoing review for registration, work on producing, marketing, and positioning the product continues. Details are added to the marketing plan as information from additional studies becomes available.

During this phase, experimental products are placed in university trials and on-farm demonstration plots under code numbers or agreements of confidentiality. Distributors, sellers, buyers, and consultants can tour the plots and learn about the product and how it performs. These additional trials build a strong efficacy history for the product and indicate how it will fit into an integrated crop production system.

Developing a brand name for the new product is a critical step in the marketing process. Trademark laws and industry groupings make this process highly complex and challenging. Pesticide products are in the same category as veterinary and pharmaceutical products, so it can be difficult to find a brand name that does not conflict with others currently in use. Each company is looking for something easy to remember that reflects the desired positioning.

Finalization of all product manufacturing details also must be addressed. If an outside vendor is used, the manufacturer’s process engineers begin working with the contractor to make sure it has the plant capacity, equipment, and processes in place to meet label specifications; stringent environmental health and safety requirements must be satisfied as well.

Introducing the Product to the Customers

The Food Quality Protection Act and the Federal Insecticide, Fungicide, and Rodenticide Act prohibit the manufacturer from advertising or promoting any product before an EPA registration number is assigned. This includes distributing promotional caps or other forms of advertisement.
bearing the product logo. However, the manufacturer is allowed to distribute educational bulletins to dealers who will sell the new product.

Once the product has an official EPA registration number, the company activates a multimedia advertising campaign focused toward potential customers, primarily, but also distributors and dealers.

The manufacturer’s research and development staff work closely with the sales force—usually a year before the company expects to launch the product—to make sure they thoroughly understand the product’s strengths and weaknesses.

The sales staff will listen to classroom presentations and visit demonstration plots, learning details about the product’s

- performance expectations,
- potential niche in the market,
- projected competitiveness against competitors’ products,
- advertising,
- availability (which markets will get it first), and
- promotional programs used for advancement into channels of trade.

**Extending the Product to Other Markets**

As previously discussed, most products are developed and marketed initially for large-scale agriculture since its financial base is large enough to underwrite the necessary research. The active ingredient is provided to scientists in noncrop markets—urban pest control, greenhouse, aquatic, ornamental, turf, right-of-way, etc.—during the second year of field testing. Customers outside traditional agricultural markets have needs driven by some of the following factors:

- Resistance or loss of performance of current products
- Loss of EPA registrations for some uses of existing products
• Customers looking for newer solutions perceived as safer for human health and the environment

• Customers seeking better integrated pest management solutions

Needs in noncrop markets and smaller agricultural segments tend to differ from traditional agricultural uses:

• Formulations designed to fit the agricultural market do not always align with smaller-use customer needs; they may be highly concentrated, requiring dilution, and packaged in large quantities. Over-the-counter products for small uses tend to come ready to use or in less concentrated form—and in smaller packages—because this is what small-use customers prefer.

• Packaging often depends on the concentration and recommended dose, but agricultural pesticides are packaged in large quantities for large scale applicators, and they are very expensive. Purchasers of over-the-counter products are advised to purchase only the amount of pesticide they can use within a reasonable amount of time, especially since most do not have access to proper methods of disposal for outdated products.

• Regulations stipulate highly specialized use precautions for some products, and properly trained professionals must apply them. Since labels for agricultural crops do not address these issues, a small-use customer would not have label instructions for his intended use.

• Risk is a primary consideration. Even though a need may exist for the new product in some of the smaller market segments, the proposed uses may be too risky for the company to pursue. The segment may be of so little value that the company would never realize a return on its development costs. Perhaps the use would allow pest resistance to develop very quickly; or there might be environmental and/or human health risks that make these uses unattractive.

In an effort to address pesticide needs in a smaller potential market, manufacturers may begin development for that market following the decision to develop the compound for agriculture. There are several options the manufacturer can pursue if it lacks the expertise required:

• Develop internal capabilities to enter these market segments.
• Partner with or acquire a company that has the expertise.

• Out-license the compound for a third party to develop these segments.

• Decide that the compound will not be developed and used in that segment.

The active molecule is likely to be developed and marketed if the opportunity is attractive to the basic manufacturer and the risk is manageable.

**Registration of herbicides in India**

Herbicides are registered under Insecticide Act, 1968 (An Act to regulate the import, manufacture, sale, transport, distribution and use of insecticides/herbicides/fungicides with a view to prevent risk to human beings or animals, and for matters connected therewith). The brief description of the registration procedure is given here as below

1. Any person desiring to import or manufacture any insecticide may apply to the Registration Committee for the registration of such insecticide and there shall be separate application for each such insecticide.

2. Every application under sub-section (1) shall be made in such form and contain such particulars as may be prescribed.

3. On receipt of any such application the registration of an insecticide, the Committee may, after such inquiry as it deems fit and after satisfying itself that the insecticide to which the application relates conforms to the claims made by the importer or by the manufacturer, as the case may be, as regards [on such conditions as may be specified by it] and on payment of such fee as may be prescribed, the insecticide, allot a registration number thereto and issue a certificate of registration in token thereof within a period of twelve months from the date of receipt of the application.

3a. In the case of applications received by it prior to the 31st March, 1975 notwithstanding the expiry of the period specified in sub-section (3) for the disposal of such applications, it shall be lawful and shall be deemed always to have been lawful for the Registration Committee to dispose of such applications
at any time after such expiry but within a period of one year from the commencement of the Insecticides (Amendment) Act, 1977 (24 of 1977):

3b. Where the Registration Committee is of opinion that the Insecticide is being introduced for the first time in India, it may, pending any inquiry, register it provisionally for a period of two years on such conditions as may be specified by it.

3c. The registration Committee may, having regard to the efficacy of the insecticide and its safety to human beings and animals, vary the conditions subject to which a certificate of registration has been granted and may for that purpose require the certificate-holder by notice in writing to deliver up the certificate to it within such time as may be specified in the notice.

4. Notwithstanding anything containing in the section, where an insecticide has been registered on the application of any person, any other person desiring to import or manufacture the insecticide or engaged in the business of, import or manufacture thereof, shall on application and on payment of prescribed fee be allotted a registration number and granted a certificate of registration in respect thereof on the same conditions on which the insecticide was originally registered.

**Registration of Insecticides Under Insecticides Rules, 1971**

6. **Manner of Registration**

1a. An application for registration of an insecticide under the Act shall be made in Form I and the said Form including the verification portion, shall be signed in case of an individual by the individual himself or a person duly authorised by him; in case of Hindu Undivided Family, by the Karta or any person duly authorised by him; in case of partnership firm by the managing partner; in case of a company, by any person duly authorised in that behalf by the Board of Directors; and in any other case by the person in-charge or responsible for the conduct of the business. Any change in members of Hindu Undivided Family or partners or the Board of
Directors or the person in charge, as the case may be shall be forthwith intimated to the secretary, Central Insecticides Board and Registration Committee and the Licensing Officer.

1b. The Registration Committee may, if necessary, direct inspection of the ‘testing facility’ for establishing the authenticity of the data.

2. An application form duly filled together with a bank draft of Rs. One hundred only, drawn in favour of the Accounts Officer, Directorate of Plant Protection, Quarantine & Storage, payable at Faridabad towards registration fee shall be sent to the Secretary, Registration Committee, Directorate of Plant Protection, Quarantine & Storage, NH-IV, Faridabad-121001, Haryana. One Self addressed stamped envelope and one stamped envelope must be enclosed along with the application.

3. The registration fee payable shall be paid by a demand draft drawn on the State Bank of India, Faridabad, in favour of the Accounts Officer, Directorate of Plant Protection, Quarantine and Storage, Faridabad, Haryana.

4. The certificate of registration shall be in Form II or Form II-A, as the case may be and shall be subject to such conditions as specified therein.

**6A. Issue of duplicate certificate of registration**

A fee of rupees five only shall be paid for a duplicate copy of the Certificate of Registration if the original is defaced, damaged or lost.

7. Appeal

   1. An appeal against any decision of the Registration Committee under section 9 shall be preferred in writing [in Form II-B in duplicater] to the Central Government in the Department of Agriculture.

   2. The appeal shall be in writing and shall set out concisely and under distinct heads the grounds on which the appeal is preferred.

   3. Every appeal shall be accompanied by a treasury challan evidencing the payment of fee of rupees ten and a copy of the decision appealed against.
4. The fee payable for preferring an appeal shall be deposited under the head "XXI" by a demand draft drawn on the State Bank of Indai, New Delhi in favour of the Pay and Accounts Officer, Department of Agriculture & Cooperation, New Delhi.

Conclusions

The decision of a company to invest in the discovery, development, and launch of a new pesticide carries a high level of risk. It begins with an expectation or assumption of future needs and gets translated into goals which, if innovative discoveries are made, transform into actionable projects.

Much of that risk is inherent to two major factors: the time between goal development and eventual product launch is long; biological systems and the markets around them fluctuate unpredictably. These two factors may create market space for a new product. Another risk factor that differentiates pesticides from typical consumer products is the highly regulated environment in which they participate. This requires the company to invest in a wide range of technical disciplines and predict regulatory outcomes and time lines; i.e., they have to predict how global agencies like EPA will review the testing data and whether those agencies will levy additional testing requirements.

Companies seek collaborative relationships with universities and independent researchers—and sometimes other companies—to assist in the development process. This enhances the company’s understanding of business opportuni-ties and effects a greater understanding of pesticide risks and the mitigation measures that can be used to reduce risks.

 Undertaking pesticide discovery, development, and launch is very high-risk, and successful recovery of investment is never guaranteed. No manufacturer commits without diligently assessing all the risks, and only a few large enterprises sustain a presence in the pesticide market. Exceeding customers’ needs and expectations is mandatory for the success of any new technology. Successful companies continually introduce innovative, lower risk technologies to minimize the impact of pests, provide for our safety, and promote a healthy environment for all of us to enjoy.
Bioherbicides

In irrigated agriculture, weed control through chemical herbicides, creates spray drift hazards and adversely affects the environment. Besides, pesticide residues (herbicides) in food commodities, directly or indirectly affect human health. These lead to the search for an alternate method of weed management, which is eco-friendly. In this regard the biological approach (a deliberate use of natural enemies to suppress the growth or reduce the population of the weed species) is gaining momentum. This approach involves two strategies: the classical or inoculative strategy, and the inundative or bioherbicide strategy. In the inoculative approach, an exotic biocontrol agent is introduced in an infested area. This method is slow and is dependent on favourable ecological conditions, which limits its success in intensive agriculture. Whereas in the inundative approach, bioherbicides are employed to control indigenous weed species with native pathogen, applying them in massive doses in the area infested with target weed flora.

A **bioherbicide** is a biologically based control agent for weeds. Bioherbicides are made up of microorganisms (e.g. bacteria, viruses, fungi) and certain insects (e.g. parasitic wasps, painted lady butterfly) that can target very specific weeds. The microbes possess invasive genes that can attack the defense genes of the weeds, thereby killing it. Bioherbicides may be compounds derived from microbes such as fungi, bacteria or protozoa; or phytotoxic plant residues, extracts or single compounds derived from other plant species. A bioherbicide based on a fungus is called a mycoherbicide. In the industry, bioherbicides and other biopesticides are often referred to as "naturals".

A bioherbicide is a preparation of living inoculum of a plant pathogen, formulated, and applied in a manner analogous to that of a chemical herbicide in an effort to control or suppress the growth of weed species. The use of bioherbicides is based on the fundamental epidemiological principles of plant pathology. Plant disease is the result of the interaction among the host plant, the pathogen and the environment, commonly referred to as the disease triangle. Although serious, devastating disease epidemics of crop plants occur, they are the exception rather than the rule and many factors can limit disease development. Pathogen factors such as low inoculum levels, weakly virulent pathogens, and poor spore dispersal mechanisms; environmental factors such as unfavorable moisture and/or temperature conditions; and plant factors such as low
susceptibility of the host, and widely dispersed host populations often limit disease. The bioherbicide approach is an attempt to bypass many of these restraints on disease development by periodically dispersing an abundant supply of virulent inoculum uniformly onto a susceptible weed population. The application is timed to take advantage of favorable environmental conditions and/or the most susceptible stage of plant growth. Similarly the bioherbicide is formulated to avoid unfavorable environmental conditions and to facilitate application. As a consequence, the development of an effective bioherbicide requires a comprehensive understanding of the pathogen(s) involved, the biology and population dynamics of the target weed(s), the optimum requirements for disease initiation and development, and the complex interactions within the hostpathogen system.

Bioherbicides offer many advantages. They include a high degree of specificity of target weed; no effect on non-target and beneficial plants or man; absence of weed resistance development, and absence of residue build-up in the environment.

Commercial bioherbicides first appeared in the market in USA in early 1980s with the release of the products Devine\(^1\), Collego\(^2\) and Biomal\(^3\). Success stories of these products and the expectation of obtaining perfect analogues of chemical herbicides have opened a new vista for weed management. Plant pathologists and weed scientists have identified over 100 microorganisms that are candidates for development as commercial bioherbicides. Some of these are described here.

Devine, developed by Abbott Laboratories, USA, the first mycoherbicide derived from fungi (*Phytophthora palmivora* Butl.), is a facultative parasite that produces lethal root and collar rot of its host plant *Morrenia odorata* (stangler wine) and persists in soil saprophytically for extended periods of residual control. It was the first product to be fully registered as a mycoherbicide. It infects and kills strangler wine (control 95 to 100%), a problematic weed in citrus plantation of Florida. Commercially Collego, a formulation of endemic anthracnose fungus *Collectotrichum gleosporioides f. sp. Aeschynemone* (cga) was developed to control northern joint vetch (*Aeschynemone virginica*) in rice and soybean field. Dry powder formulation containing 15% spores (condia) of cga as an active ingredient was registered in 1982 under the trade name Collego, having a shelf-life of 18 months. It is the first commercially available mycoherbicide for use in annual weed in annual crops with more than 90% control efficiency.
The successful development of Collego led to the discovery of another Collectotrichum-based mycoherbicide, ‘Biomal’ by Philom Bios Inc., Canada. It contains spores of C. gleosporiodes (Penz.) Sacc. f. sp. Malavae. It is used to control Malva pusilla (round-leaved mallow) in Canada and USA. The most effective period of application is at an early stage, although it can be effective at any stage of weed growth. Further, the rust fungus Puccinia canalicuta (Schw) legrah is commercialized under the name Dr. Biosedge for control of Cypruss esculantus L. (yellow nut sedge). Recently the potential of many microorganisms, especially fungus to control weeds in several crops has been reported. Some of them are listed here. Alternaria cassiae (Casst), Cercospora rodmani (ABG-5003), Cercospora coccodes (Velgo), Collectotrichum orbicular, Fusarium aonifliral, Deleterious rhizobacteria (DRB), Pseudomonas spp., Agrobectarium, Xanthomonas spp., Ervinia herbicola, Pseudomonas syringae pv. Tagetis (Pst), Xanthomononas campaestris pv. Poannua, (Xcp), S. hygroscoplus (Bialaphos).

**BASIS OF THE BIOHERBICIDE APPROACH**

In the development of any new pest control strategy, safety and efficacy are the two primary concerns (Watson & Wymore, 1989b). As a consequence, safety (in relation to crop plants, the environment, and human health) and efficacy (in relation to environmental tolerance, level of damage to the weed, and ability to be integrated within the crop production system) are the major criteria in the selection of suitable plant pathogens.

The better understanding of the genes of both microorganisms and plants has allowed scientists to isolate microbes (pathogens) whose genes match particular weeds and are effective in causing a fatal disease in those weeds. Bioherbicides deliver more of these pathogens to the fields. They are sent when the weeds are most susceptible to illness. The genes of disease-causing pathogens are very specific. The microbe’s genes give it particular techniques to overcome the unique defenses of one type of plant. They instruct the microbe to attack only the one plant species it can successfully infect. The invasion genes of the pathogen have to match the defense genes of the plant. Then the microbe knows it can successfully begin its attack on this one particular type of plant. The matching gene requirement means that a pathogen is harmless to all plants except the one weed identified by the microbe’s genetic code. This selective response makes
bioherbicides very useful because they kill only certain weed plants that interfere with crop productivity without damaging the crop itself. Bioherbicides can target one weed and leave the rest of the environment unharmed.

The preferred characteristics of a potential bioherbicide pathogen include: 1) growth and sporulation on artificial media, 2) highly virulent, 3) genetic stability, 4) restricted host range, 5) broad tolerance range, 6) prolific propagule production, 7) capacity to damage its host plant, and 8) innocuous in ecological effects (Templeton et al., 1979).

In determining the suitability of a particular weed species as a target for bioherbicide development, native or naturalized weed species should have a larger complement of indigenous pathogens to select from as compared to fewer pathogens associated with recently introduced weeds. Templeton et al. (1986) suggest that bioherbicides have greatest potential for control of: a) weeds infesting small specialized areas where chemical herbicide development would be too costly, b) weeds that have been intransigent to chemical control, c) crop mimics, and d) parasitic weeds. Since potential return on investment is critical to industrial involvement in bioherbicide development, major weeds, presently not controlled by available technology, in major crops are perhaps the ideal targets for the bioherbicide approach.

Intuitively, annual weed species may be considered preferred targets when compared to perennial weed species. However, the growth habit, growth rate and other biological parameters which determine susceptibility and subsequent disease development are more critical than whether the weed is an annual or a perennial. For example, annual weeds such as velvetleaf (*Abutilon theophrasti*) with their erect habit of growth and rapid rate of stem elongation may be less susceptible to disease development of foliar pathogens when compared to vigorous perennials such as field bindweed (*Convolvulus arvensis*) and dandelion (*Taraxacum officinale*) with their prostrate habits of growth.

STEPS IN BIOHERBICIDE DEVELOPMENT

The development of a biological herbicide involves three major phases or stages: 1) discovery, 2) development, and 3) deployment (Templeton, 1982). The discovery phase involves the collection of diseased plant material, isolation of the causal organism, demonstration of Koch’s postulates,
identification of the pathogen, culture of the pathogen on artificial media, and maintenance of the pathogen cultures in short-term and long-term storage. The development phase involves the determination of optimum conditions for spore production, determination of optimum conditions for infection and disease development, determination of host range and elucidation of mechanism of action of the pathogen. The final phase, deployment, often involves close collaboration between nonindustrial and industrial sectors through the formulation, scale-up, field evaluation, and marketing stages of commercialization process of a new bioherbicide product.

The proposed close collaboration between industrial and non-industrial sectors is not always easy, especially when the objectives of the two groups are often not completely compatible. Both Baker (1986) and Scher and Castagno (1986) point out that despite intensive research and numerous apparently successful biological control agents, very few have reached the marketplace. Baker (1986) suggests the need for more research related to understanding the basic mechanisms of biological control, whereas Scher and Castagno (1986) suggest the reason for the paucity of marketable biocontrols is because most biocontrol agents have been developed from a scientific point of view only, without an industrial perspective.

**Limitations in use of Bioherbicides**

Besides many advantages of bioherbicides, certain factors have been reported to limit the development of bioherbicides into commercial products. These include biological constraints (host variability, host range resistance mechanisms and interaction with other microorganisms that affect efficacy), environment constraints (epidemiology of bioherbicides dependent on optimum environmental conditions), technical constraints (mass production and formulations development of reliable and efficacious bioherbicide), and commercial limitations (market size, patent protection, secrecy and regulations).

The bioherbicides approach is gaining momentum. New bioherbicides will find place in irrigated lands, wastelands as well as in mimic parasite weeds or resistant weed control. Research on synergy test of pathogens and pesticides for inclusion in IPM, developmental technology, fungal toxins, and application of biotechnology, especially genetic engineering is required. However, bioherbicides should not be viewed as a total replacement to chemicals, but rather as complementary in integrated weed management systems.
Allelochemicals

Allelopathy is a biological phenomenon by which an organism produces one or more biochemicals that influence the growth, survival, and reproduction of other organisms. These biochemicals are known as allelochemicals and can have beneficial (positive allelopathy) or detrimental (negative allelopathy) effects on the target organisms. Allelochemicals are a subset of secondary metabolites, which are not required for metabolism (i.e. growth, development and reproduction) of the allelopathic organism. The term allelopathy or Teletoxy was introduced by Molisch (1937). *Parthenium* daughter plants exhibiting teletoxy to its parent plants is known as autotoxy. The word allelopathy is derived from Greek – allelo, meaning each other and patho, an expression of sufferance of disease.

Allelopathy is characteristic of certain plants, algae, bacteria, coral, and fungi. Allelopathic interactions are an important factor in determining species distribution and abundance within plant communities, and are also thought to be important in the success of many invasive plants. Allelochemicals are found to be released to environment in appreciable quantities via root exudates, leaf leachates, roots and other degrading plant residues, which include a wide range of phenolic acids such as benzoic (1) and cinnamic acids (2), alkaloids (3), terpenoids (4) and others. These compounds are known to modify growth, development of plants, including germination and early seedling growth.

Allelochemicals appear to alter a variety of physiological processes and it is difficult to separate the primary from secondary effects. There are increasing evidences that allelochemicals have significant effects on cell division, cell differentiation, ion and water uptake, water status, phytohormone metabolism, respiration, photosynthesis, enzyme function, signal transduction as well as gene expression. It is quite possible that allelochemicals may produce more than one
effect on the cellular processes responsible for reduced plant growth. However, the details of the biochemical mechanism through which a particular compound exerts a toxic effect on the growth of plants are not well known.

Allelochemicals are released in the form of:

**Vapour** (released from plants as vapour): Some weeds release volatile compounds from their leaves. Plants belonging to labiateae, compositeae yield volatile substances.

**Leachates** (from the foliage): From *Eucalyptus* allelo chemicals are leached out as water toxins from the above ground parts by the action of rain, dew or fog.

**Exudates from roots**: Metabolites are released from *Cirsium arvense* roots in surrounding rhizosphere.

**Decomposition products** of dead plant tissues and warn out tissues

The production of allelo chemicals is influenced by the intensity, quality and duration of light. Greater quantity produced under ultra violet light and long days. Under cropped situation low allelo chemicals. Greater quantities are produced under conditions of mineral deficiency, drought stress and cool temperature more optimal growing conditions.

**Commercial herbicides based on natural products**

Herbicides and agrochemicals based on natural products are attractive for a variety of reasons. Most biologically active natural products are at least partially water-soluble and, as a result of natural selection, more likely to exhibit some bioactivity at low concentrations. Natural products are frequently considered to be environmentally benign, but many plant and microbial compounds are potent mammalian toxins. Many allelochemicals exert their influence through mechanisms not possessed by commercial herbicides, making them ideal lead compounds for new herbicide discovery. Unfortunately, the complex structures of most secondary metabolites, usually containing several stereocenters, complicate structural characterization and make the feasibility of economical, large-scale synthesis of the compound questionable. Structural simplification of the lead compound often results in significantly lower biological activity. These issues, of course, are the same ones encountered in the pharmaceutical industry, but the pesticide industry has shown only modest interest in the natural product-based discovery approach to herbicides. Most of the effort that has been expended concerns natural products obtained from microbial sources rather than higher plants.
1. Organophosphorous compounds

Two herbicides based on natural products isolated from bacteria have been commercialized to date: bialaphos (5) and phosphonothricin (6). The ammonium salt of synthetic racemic 6 is glufosinate, marketed under a variety of trade names. Bialaphos (also known as phosphonothricylalanyl alanine) (5) was originally isolated from different Streptomyces strains by two independent groups and is currently marketed in Japan under the name Herbiace. Although not a natural product, the widely used herbicide glyphosate (7) bears a striking structural resemblance to 5 and 6. Another phosphonate natural product, phosphonothrixin (8), was recently isolated from Saccharothrix sp. ST-888 and exhibits phytotoxic activity against a variety of plants.

![Structures of bialaphos (5), phosphonothricin (6), glyphosate (7), and phosphonothrixin (8)]

2. Triketones

Leptospermone (9) is a major component in the essential oil of the plant Leptospermum scoparium found in Australia and New Zealand. The triketone herbicides, including sulcotrione (10) and mesotrione (11), are post-emergent broadleaf herbicides based on the leptospermone structure template that inhibit p-hydroxyphenylpyruvate dioxygenase (HPPD). The herbicidal activity of these compounds correlates well with their acidity, accounting for the electron withdrawing substituents on the benzoyl moiety of these compounds.

![Structures of leptospermone (9), sulcotrione (10), and mesotrione (11)]
3. Cinmethylin

The monoterpane ether 1,8-cineole (12) is a major component of the essential oils of a number of plant species, and was one of the first compounds implicated as an allelochemical. Compound 12 and its isomer 1,4-cineole (13) are potent phytotoxins, but their high volatility presents a problem for herbicide applications. Cinmethylin (14), a benzyloxy derivative of 13, was developed as a herbicide to control annual grasses. This strategy has prompted further research on benzyl ether derivatives of monoterpenes. Cinmethylin (14) interrupts mitosis in treated plants, and recent experiments involving asparagine synthetase demonstrated that 13 is in fact a proherbicide that requires cleavage of the benzyl group producing the active agent 15.

4. Selected allelochemicals

Compounds from numerous structural classes have been implicated in allelopathic interactions. The remainder will concentrate on allelochemicals of selected structural classes, their activity, and in some cases their synthesis. The major focus is on compounds isolated in the last 15 years. Allelochemicals isolated from sunflower (Helianthus sp.) are given special attention due to the extensive research and large number of publications in this area during the period of interest.

i) Benzoquinones

Sorgoleone (16) and its hydroquinone form 19 are allelochemicals exuded from the roots of sorghum. Compound 19 was the first natural host germination stimulant for the parasitic weed *Striga asiatica* (witchweed) to be isolated and characterized and has been the subject of a total synthesis. Sorgoleone is highly phytotoxic and inhibits chlorophyll formation and photosynthetic oxygen evolution.
More recent studies have shown that sorgoleone (16) inhibits electron transport in photosystem II (PS II) and can displace the commercial herbicide atrazine from the Q₁ binding site on the D₁ protein of PSII.

ii) Coumarins and flavonoids

Coumarins and flavonoids are ubiquitous in plants, and several have been implicated in allelopathic interactions. Coumarin (19) and its derivatives such as scopoletin (20) are known inhibitors of seed germination and growth of various plants, and 19 blocked mitosis in Allium cepa (onion). Coumarin (19) and the furanocoumarin psoralen (21) are components of Ruta graveolens (Rue), a medicinal plant with allelopathic properties. Psoralen (21) can inhibit lettuce seed germination at a concentration of 1 ppb.

Recent studies of the South American shrub Pilocarpus goudotianus yielded a number of furano- and pyranocoumarins (22–29). These compounds were tested along with selected synthetic coumarins in a lettuce germination bioassay. Significant activity was not observed below 10⁻⁴ M, thus making compounds 22, 23, and 25 most likely responsible for the allelopathic activity of P. goudotianus due to their higher natural concentrations in the plant.

The allelopathic activity of compounds isolated from Melilotus messanensis (sweet clover) has been the subject of several investigations. Several flavonoids and a coumarin isolated from sweet clover were recently subjected to bioassay, but their observed activities were low, suggesting that triterpenes and saponins are responsible for the activity of sweet clover.
iii) Terpenoids

Tens thousands of isoprenoid compounds are known and hundreds more are reported in the literature each year. Therefore, it is not surprising that these secondary metabolites have been examined for their allelopathic potential. This topic has been the subject of past reviews.

Messagenic acids A–I (30–38) are a family of nine lupane triterpenes isolated from sweet clover along with the oleanane triterpenes melilotigenins B–D (40–42). Messagenic acids D, F, and G (33, 35, 36) were prepared via semi-synthesis from the more plentiful betulinic acid (39) in order to supplement the minute amounts of the natural material available. These compounds were assayed for seed germination and growth activity using both mono- and dicotyledons. The compounds tested showed insignificant effects on lettuce but inhibited the seed germination of *Hordeum vulgare* (barley) and stimulated seed germination of *Allium cepa* (onion).

Breviones A–E (43–47) are novel diterpenoid derivatives with a polyketide moiety attached through a spiro ring junction. The breviones were isolated from *Penicillium Brevicompactum* Dierckx, and their structures were elucidated through a combination of spectroscopic and chemical methods. A mechanism for their biogenesis has been proposed. Breviones C and E (44 and 46) were the most active compounds in a etiolated wheat coleoptile assay, inhibiting growth of the wheat shoots 80 and 100%, respectively, at $10^{-4}$ M. Breviones A and B (43 and 44) were less active (40% growth inhibition at $10^{-3}$ M), but brevione D (45) was not assayed due to lack of material.
Germacranolides and guaianolides have drawn particular attention among the many classes of sesquiterpenoid lactones that have been studied for allelopathic properties. The role of the α-methylene lactone moiety and the effect of molecular conformations for a number of natural and synthetic sesquiterpene lactones of this type has been examined. More recently Macías continued these investigations, testing the natural germacranolides costunolide (48) and parthenolide (49) and synthetic derivatives 50–55 for phytotoxicity on a number of mono- and dicotyledons. Costunolide (48) and derivatives 50, 52, and 54 also stimulate germination of parasitic witchweed species.

Dehydroazulanin C (56) has been isolated from a number of plant species, and is one of the many guaianolides studied for allelopathic potential. Dehydroazulanin C (56) is unique, however, in having a dual mechanism. The α-methylene lactone moiety is responsible for germination and growth inhibition similar to other guaianolides, but the second Michael acceptor, the cyclopentenone group, is responsible for rapid leakage of the plasma membrane. Dehydroazulanin C (56) was prepared via semi-synthesis from dehydrocostuslactone (57) via *iso*azulanin (58). Other guaianolide derivatives have been prepared by manipulation of 57 and studied with Macías’ STS assay to construct a SAR. This SAR study showed that the lactone group was necessary for activity, but did not have to be unsaturated. Furthermore, a second α,β-unsaturated carbonyl enhanced activity if the molecular conformation rendered it sterically accessible. It was also concluded that the presence of additional hydroxyl groups lowered activity when polarity reached a level sufficient to inhibit membrane transport.
Nagalactone F (59), LL-Z1271a (60) and a variety of related podolactones were recently synthesized and evaluated in Macías’ STS bioassay. Compound 60 had a better activity profile than the internal standard in these studies, the commercial herbicide Logran®, at $10^{-4}$ and $10^{-5}$ M.

Ailanthone (61) is a quassinoid lactone from *Ailanthus altissima* (Tree-of-Heaven) and was recently shown to be the major phytoxin of this allelopathic plant. Ailanthone is a post-emergence herbicide, but is rapidly degraded in the field, losing its effect after several days.

Artemisinin (62) is known to most organic chemists as a promising anti-malaria agent, but this endoperoxide-containing lactone is also a selective phytotoxin. There is no correlation between herbicidal activity and activity against *Plasmodium* parasites, however. A series of synthetic artemisinin analogues was evaluated for herbicidal activity. The artemisinin analogues increased oxygen uptake and decreased chlorophyll content in treated plants. No membrane disruption was found, however, and complementation experiments suggested that the phytotoxic effect of 62 and the synthetic analogues was not due to inhibition of porphyrin biosynthesis, amino acid synthesis, or nucleic acid synthesis. Thus the mode of action of 62 is still not clear.

Trichocaranes A–D (63–66) are carotane sesquiterpenes recently isolated from the fungus *Trichoderma virens*. Trichocarane D (66) was inactive in the etiolated wheat coleoptile assay, but trichocaranes A and B (63 and 64) inhibited growth 40% at $10^{-4}$ M and trichocarane C (65) inhibited growth 86% at $10^{-3}$ M.

### iv) Strigolactones

Parasitic weeds of the *Striga* (witchweeds), *Orobanche*, and *Alectra* families affect a number of important cereal and legume crops, causing dramatic reduction in yield and in severe cases complete destruction of the crop. The problem is especially severe in Africa and is becoming
more prevalent. The seeds of these parasites only germinate in the presence of chemical stimulants exuded from other plants. Typically the host plant is the source of seed germination stimulant, but the first such germination stimulant to be characterized, strigol (67) was in fact isolated from a non-host plant. The first seed germination stimulant isolated from a *Striga* host (*S. bicolor*) was the hydroquinone form of sorgoleone (19). Structurally related witchweed seed germination stimulants sorgolactone and alectrol were isolated from sorghum and *Vigna unguiculata* (cowpea) respectively, and assigned structures 68 and 69. Both of these compounds were obtained in minute quantities and the structures were assigned based on spectral data correlated with data from strigol (67), whose structure had been confirmed by X-ray analysis. Ororbanchol, a germination stimulant for *Orobanche minor* (clover broomrape) was isolated from its host, *Trifolium pratense* (red clover), and assigned structure 70. These compounds are commonly referred to as strigolactones and are active at concentrations below $10^{-11}$ M. Recently a series of semi-synthetic lactones of various sesquiterpenoid families were tested as *Orobanche cumana* germination stimulants.

![Chemical structures of strigolactones](image)

The strigolactones have also stimulated the interest of synthetic organic chemists with intriguing results. Strigol (67) has been synthesized in racemic and optically pure form. Zwanenburg concluded that the stereoisomer is identical to natural strigolactone after synthesizing all eight stereoisomers of 68. Mori’s group disputes this conclusion, however. Mori’s group has disproven the originally proposed structures of alectrol, and orobanchol. Based on extensive synthetic effort, Mori’s group has concluded that orobanchol is actually 71. Illustrated by the case of the strigolactones, one can clearly see the importance of the synthetic chemist to allelopathic research.
Allelochemicals from sunflower

Few plants have been studied as much in recent years for their allelopathic potential as the sunflower, *Helianthus annuus*. Sunflowers are an important crop in many parts of the world, and dozens of hybrid varieties are known, 26 in the Andalusia region of Spain, for example. Leather conducted some of the first studies on this species, and showed that sunflower extracts inhibited germination of growth of a variety of weed species. *H. annuus* has activity against such troublesome weeds as morning glory, velvetleaf, pigweed, jimson weed, wild mustard, and others. Subsequent research has included examination of the effects sunflower growth stage has on the allelopathic effects, and examination of other sunflower species.

i) Heliannuols

The heliannuols, are a promising group of phenolic allelochemicals isolated from *H. annuus*. The phenolic functional group has long been associated with allelopathic activity. Heliannuol A was isolated from an aqueous extract of *H. annuus* L. var. SH-222 and has a novel sesquiterpenoid skeleton consisting of an eight-membered cyclic ether fused to a benzene ring. Heliannuols B, C and D were isolated shortly thereafter, all containing a seven-membered benzofused cyclic ether. Heliannuol E has a vinyl substituent, and is the only heliannuol that contains a six-membered benzofused ether. Heliannuols F–K have more highly oxidized benzofused ether rings, but are present in minute quantities—extraction of 6 kg of fresh sunflower leaves yielded only 1–2 mg.

ii) Sunflower terpenoids

Sesquiterpene lactones are common constituents of *Helianthus* species. Annuolides A–G are a family of guaianolides isolated from sunflower cultivars that exhibit allelopathic activity. Bioassay data indicated that the α-methylene lactone was not strictly required for inhibiting lettuce seed germination but the compounds with the α-methylene moiety were active at lower concentrations. Derivatization of the hydroxyl group closest to the methylene lactone as in annuolides F and G reduced activity.

*Helianthus* cultivars have also yielded some interesting bisnorsesquiterpenes such as annuionones A–D and helinorbisabone.

ii) Sunflower flavonoids

The sunflower has also yielded chalcones and flavonoids in the search for allelochemicals. Chalcones kulkukanin B and heliannone A were isolated from both *H. annuus* cultivars VYP
and cv. Peredovich. Bioassays indicated Chalcones, kulkukanin B and heliannone A mainly affect shoot growth of *Lycopersicon esculentum* (tomato) and *H. vulgare* (barley) seedlings.

**Allelopathic control of certain weeds using Botanicals**

For instance Dry dodder powder has been found to inhibit the growth of water hyacinth and eventually kill the weed. Likewise carrot gross powder found to detrimental to other aquatic weeds. The presence of marigold (*Tagetes erecta*) plants exerted adverse allelopathic effect on *Parthenium* spp. growth. The weed coffeesena (*Cassia* sp) show suppressive effect on *Parthenium*. The eucalyptus tree leaf leachates have been shown to suppress the growth of nut sedge and bermuda grass.

Allelo chemicals are produced by plants as end products, by-products and metabolites liberalized from the plants

1) **Allelopathic effects of weeds on crop plants.**

   - Root exudates of Canada thistle (*Cirsium* sp.) injured oat plants in the field.
   - Root exudates of *Euphorbia* injured flax. But these compounds are identified as parahydroxy benzoic acid.

**Maize**

   - Leaves & inflorescence of *Parthenium* sp. affect the germination and seedling growth
   - Tubers of *Cyperus esculentus* affect the dry matter production
   - Quack grass produced toxins through root, leaves and seeds interfered with uptake of nutrients by corn.

**Sorghum**

   - Stem of *Solanum* affects germination and seedling growth
   - Leaves and inflorescence of *Parthenium* affect germination and seedling growth

**Wheat**

   - Seeds of wild oat affect germination and early seedling growth
   - Leaves of *Parthenium* affects general growth
   - Tubers of *C. rotundus* affect dry matter production
   - Green and dried leaves of *Argemone mexicana* affect germination & seedling growth

**Sunflower**

   - Seeds of *Datura* affect germination & growth

2) **Effect of weed on another weed**

   - **Thatch grass** (*Imperata cylindrica*) inhibited the emergence and growth of an annual broad leaf weed (*Borreria hispida*).
• Extract of leaf leachate of decaying leaves of *Polygonum* contains flavonoids which are toxic to germination, root and hypocotyls growth of weeds like *Amaranthus spinosus*  
• Inhibitor secreted by decaying rhizomes of *Sorghum halepense* affect the growth of *Digitaria sanguinalis* and *Amaranthus* sp.  
• In case of *Parthenium*, daughter plants have allelopathic effect on parent plant. This is called AUTOTOXY

3) **Effect of crop on weed**

• Root exudates of wheat, oats and peas suppressed *Chenopodium album*. It increased catalase and peroxidase activity of weeds and inhibited their growth.  
• Cold water extract of wheat straw reduces growth of *Ipomea* & *Abutilon*.

4) **Stimulatory effect**

• Root exudates of corn promoted the germination of *Orcchanca minor*; and *Striga hermonthica*. Kinetin exuded by roots sorghum stimulated the germination of seeds of *stirga asisatica*.  
• Strigol – stimulant for witch weed was identified in root exudates from cotton.

---

**Herbicide Bioassays**

Bioassays are experiments that use living things to test the toxicity of chemicals. Bioassays are used to conduct a wide range of experiments relating to toxicity of known chemical solutions or unknown mixtures such as samples of water, sediment, or soil from the environment. In spite of rapid developments in analytical methods, bioassay remains a major tool for qualitative and quantitative determination of herbicides. A major advantage of the bioassay is the assurance that the phototoxic activity of the herbicide molecule is being measured. A secondary advantage is that it is not generally necessary to extract the herbicides from the substrate. Bioassay procedures are usually more economical, less difficult to perform and do not require as much expensive equipments as in chemical analytical methods.

There are various procedures for conducting the bioassay studies. The choice of a particular procedure will depends upon the herbicide and its phytotoxicity to bioassay species. At the same time the bioassay must also satisfy certain requirements. In an effective bioassay,, the indicator species should be sufficiently sensitive to detect even small amounts of herbicides and should
express the response with increasing herbicide concentrations. The rest should be rapid and the
time required for the conduct should be minimal.

2. Indicator Species:
Various organisms can serve as an indicator for a given herbicide and conversely, many
herbicides though belong to different chemical groups, can be tested by the same organism.
Microorganisms also have been used in some bioassays (Hess, 1980). The indicator species
should be sensitive to minute amounts of the chemical and should respond by clear, easily
observable and measurable symptoms. Herbicide bioassays are usually conducted with sensitive
plant species referred as indicator / test species. Cucumber, sorghum, mustard, soybean, oat and
minor millets are some commonly used indicator plants in herbicide bioassays.

Table 1. Test species used for various group of herbicides

<table>
<thead>
<tr>
<th>Herbicide group</th>
<th>Bioassay species</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aliphatic halogenated acids (Dalapon)</td>
<td>Oats, millets, cucumber, barley, wheat, rice</td>
</tr>
<tr>
<td>Acetamides (diphenamide)</td>
<td>Crabgrass, oats, barley, ryegrass, pigweed</td>
</tr>
<tr>
<td>Acetanilide (alachlor, metolachlor)</td>
<td>Cucumber, ryegrass, crabgrass</td>
</tr>
<tr>
<td>Benzoic acid</td>
<td>Cucumber, oats, foxtail, pigweed</td>
</tr>
<tr>
<td>Dicamba</td>
<td>Beans, sorghum, cucumber</td>
</tr>
<tr>
<td>Chlorpropham</td>
<td>Cucumber, oats, ryegrass</td>
</tr>
<tr>
<td>Phenoxy derivatives (2,4-D)</td>
<td>Cotton, pigweed, tomatoes, mustard</td>
</tr>
<tr>
<td>Substituted ureas (diuron, isoproturon)</td>
<td>Cucumber, ryegrass, oats, barley, millet, sorghum</td>
</tr>
<tr>
<td>Thiocarbamates (EPTC)</td>
<td>Ryegrass, oats</td>
</tr>
<tr>
<td>Dinitroanilines (trifluralin, fluchloralin)</td>
<td>Oats, sorghum, rice, cucumber</td>
</tr>
<tr>
<td>Triazines (atrazine, simazine)</td>
<td>Oats, cucumber, sugarbeets</td>
</tr>
</tbody>
</table>

3. Assessment Parameter:
The response of indicator plants to herbicides can be evaluated in various ways.
A) Germination Tests:

Many herbicides strongly inhibit the germination of sensitive species but few tests have been based simply on germination numbers. Sublethal concentrations frequently produce an inhibition of radical or shoot elongation which is dose related in a range sufficiently large to allow valid measurements. Typically, the root or shoot elongation is observed after a period of 24 – 96 hours. Cucumber, sorghum and oat are the main species used in germination tests but they are not sensitive to photosynthetic inhibitors (Kratky and Warren 1971).

B) Assessment of Plants:

Determination of dry weight is most common assessment used in bioassay. Generally, the tops only are weighed since the separation of roots from soil is laborious. Observation based on fresh weight and dry weights generally give good estimations.

For many photosynthesis inhibitors shoot growth is reduced before the appearance of the injury symptoms. Measurement of plant height or leaf length provides an assessment of herbicidal activity which may replace weighing. While leaf length is easily measured on monocots, other measurement may be considered on the other species. For instance, the petiole of the first trifoliate leaf of legumes gives dependable estimation.

Observation of plant height generally gives similar results. However, with herbicides inducing deformation in the shoot, the plant height values should be used with care. Koren et al. (1968) found that with thiocarbamates the height reduction was greater than weight reduction. Recording both the parameters is useful in experiments including different type of herbicides (Horowitz and Blumenfeld, 1973). Visual estimation of relative development or of injury intensity is often used (Scifres et al. 1972).

C) Physiological and Morphological Effects:

The physiological, bio-chemical and structural modifications of plants induced by herbicides can be used for bioassay assessment. Reduction of photosynthetic activity by photosynthesis inhibitors has been measured or leaf discus (De Silva et al. 1976). Chlorosis may be evaluated by
determining chlorophyll content through spectrophotometer (Horowitz 1970). Duffy (1972) estimation the decrease in viability of root tissue with a modified tetrazoltum test.

D) Symptoms:

Symptoms which are typical of a certain group of herbicides or of a given compound can be used for qualitative assay and if the intensity of symptoms are dose related, it can also be used for quantitative determinations. Ex. Epinasty of cotton have been used to measure the effect of 2, 4 – D.

4. Application of Bioassay:

Bioassay procedures have been used to investigate many practical aspects of herbicide behaviors in the environment. The different methods and approaches in herbicide research based on bioassay are outlined below:

A) Soil Effects:

The effect of soil factors on herbicidal activity may be determined by bioassay and correlate phytotoxicity with relevant soil physical and chemical characteristics.

B) Dissipation from Soil Surface:

Bioassays have been used in experiments dealing with the process of dissipation from soil surface especially volatilization and photodecomposition.

In volatility studies, a source at a sink for herbicide vapors may be set up in a closed container or treated soil may be exposed to an air flow. The bioassay, measures the concentration of herbicide remaining in the soil and from this the amount of herbicide volatilized is computed (Talbert et al. 1971).

Photodecomposition can be induced experimentally by irradiation herbicides in soil solution or on artificial surfaces or on soil with UV lamps or natural sunlight. The herbicidal activity is measured by bioassay after certain period of exposure and compared with non ex. Posed treatments (Parochetti and Hein. 1973).
C) Movement in Soil:

Various bioassay methods have been developed for studying the movement of herbicides in soil. In field experiments soil samples may be taken from different depths and assayed. In laboratory leaching studies using soil columns, various methods have been used to assess the movement of compounds. Nishimoto and Warren (1971) also developed bioassay techniques to examine upward and lateral movement of herbicides.

D) Degradation and Persistence:

Soil samples at different depths may be collected from the herbicide applied plots and assayed in the laboratory. A similar procedure has been used to follow dissipation of herbicides in water (Hiltribran, 1962).

E) Residual Effect:

The residual effect of herbicides on succeeding crop can be assessed using bioassays.

5. Bioassay Procedure:

The procedure developed by Crafts (1935) is followed by many researchers with little modifications. Soils treated with the herbicide at different concentrations are sown with an indicator plant and after measuring the germination and response (plant height and dry weight) of the indicator plants standard curves are drawn. By comparing these parameters obtained from the field sample where the said herbicide has been applied with that of standard, the residue is quantified. Jayakumar (1987) standardized the bioassay technique for a number of herbicides different soil types and brought out quadratic models for easy assessment.

The procedure outlined here was developed for atrazine (AAtrex), a widely used herbicide. The bioassay procedure also can be used to determine problematic residue levels of other herbicides, such as metribuzin (Sencor/Lexone®), simazine (Princep®), linuron (Lorox), pendimethalin (Prowl®), treflan (Treflan™), isoxaflutole (Balance®), imazaquin (Scepter®), nicosulfuron (Accent®), and others. Injury symptoms and susceptible crops will differ depending on the herbicide in question.
Obtaining a Representative Soil Sample

Collect a representative sample of soil from the areas of the field suspected of having herbicide residue. The assay is only as reliable or representative as your sample. A total of 4 kg of moist soil is required for each sample assayed. Assays should be carried out on moist soil samples within two days after they are obtained from the field. Samples that cannot be assayed immediately should be stored in a freezer. The amount of herbicide residue may decrease due to biological and non-biological breakdown if samples are stored under warm conditions.

Preparing Soils for the Bioassay

Wet soil should be spread out and allowed to partially dry to a workable condition. If the soil is cloddy, crush so the clods are no larger than wheat seed, but do not pulverize. Blend the moist soil together to obtain a uniform sample. It is necessary to prepare a herbicide-free soil, one where there is no herbicide residue or where the residue has been inactivated. One option is to collect a clean soil sample from a field where herbicides have not been applied or use a commercial potting mix. The second option is to treat a portion of the soil collected in the field with activated charcoal.

After blending the soil cores together, divide the soil into two equal portions (each about 2 kg). Add 1 level teaspoon of activated charcoal to half of the soil. Thoroughly mix the charcoal into the soil. Most of the herbicide will be bound to the carbon in the charcoal and will be inactivated, providing a herbicide-free soil for comparison. For best results use powdered charcoal.

Planting and Growing Bioassay Species

Flower pots may be used for the bioassay. Use the same kind and size of container for both treatments. Punch holes in the bottoms for drainage. Fill one container using charcoal-treated or herbicide-free soil and another with soil as it came from the field. Properly label each container.

Herbicide injury symptoms on seedlings should become apparent within three weeks after planting seed. Less time is required if high herbicide concentrations are present. It will take longer if the temperature is below 70°F.
To test for atrazine carry-over, plant six beans (soybeans, field beans or garden beans) or 10 oat seeds in each container. Different species may be required to test for carry-over from other herbicides. Press or punch the seeds no deeper than 1/4 inch into the soil. Plant so there is uniform spacing between the seeds. Thoroughly water the soil. Germinate beans at 72°F. Lower temperatures are satisfactory for oats.

Cover the containers with plastic food wrap until plants begin to emerge from the soil to provide favorable moisture conditions during germination. At emergence, remove the plastic. Water as needed. Do not let the soil dry out.

Keep the containers in a warm place (70 to 75°F) where they will get as much sunlight as possible. Sunlight is essential for the development of injury symptoms from atrazine and many other herbicides.

Soon after emergence, thin the beans to three plants per container by clipping them off at the soil surface. Maintain uniform spacing between the remaining plants. Thinning should be done as soon as possible after emergence to reduce the amount of herbicide removed by discarded plants. It is not necessary to thin the oats.

With too many plants per container, low concentrations of herbicides may not be detected. When large number of plants are present, each plant extracts a very small amount of herbicide from the soil. The amount may be small enough that it is not toxic to the plant. When fewer plants are grown in the same amount of soil each plant may extract more herbicide, and will show injury or die if enough herbicide residue is present. Three bean plants or 10 or fewer oat plants are all that should be used in 2 kg of soil because these numbers more nearly approach field conditions, and provide a more accurate indication of low herbicide concentrations.

**Typical Herbicide Injury Symptoms**

Herbicide injury symptoms will only appear in contaminated soils when moisture, temperature, and light conditions are favorable for the growth of the bioassay species. Specific symptoms depend on the bioassay species and the herbicide in question. In *Table I* we describe types of injury symptoms one might expect for the major classes of herbicides.
Plants growing in herbicide-contaminated soil may have one or more of the symptoms described in Table. In contrast, plants growing in herbicide-free soil (either charcoal treated or uncontaminated) should appear normal. If test plants die in both containers, disease could be the cause.

**If you have herbicide carry-over**

When the bioassay results in herbicide injury to the sensitive species, crop rotation options are limited to crops that are not sensitive to the herbicide residues. For example, if atrazine symptoms occur, plant an atrazine-tolerant crop such as corn or sorghum. This bioassay is an inexpensive and effective way to avoid experiencing crop damage from herbicide carry-over by knowing residues are in your soil that might delay crop development. When you discover herbicide carry-over, you also can plan to avoid the problem by reducing the rate of the offending herbicide in areas where it is less sensitive to degradation.

**Table. Common injury symptoms on sensitive species**

<table>
<thead>
<tr>
<th>Herbicide mode of action</th>
<th>Representative products</th>
<th>Typical injury symptoms on sensitive crop species</th>
</tr>
</thead>
</table>
| Growth regulators        | 2,4-D, dicamba (Banvel®, Clarity®, Status), clopyralid (Stinger™), fluroxypyr (Starane™) | **Broadleaf species:** leaf cupping or strapping, stem twisting or epinasty, callus tissue on stems, brittle stems, necrosis of meristematic tissue  
**Grass species:** onion leafing, leaf rolling, brace-root fusion, brittle stems, stalk bending |
| ALS inhibitors           | imazaquin (Scepter®), imazethapyr (Pursuit®), chlorimuron (Classic®), thifensulfuron (Harmony®), nicosulfuron (Accent®), flumetsulam (Python®) | **Broadleaf species:** chlorosis, stunted plants or shortened internodes, red or black veins on underside of leaf  
**Grass species:** stunted plants, interveinal chlorosis, purpling of |
<table>
<thead>
<tr>
<th>Herbicide mode of action</th>
<th>Representative products</th>
<th>Typical injury symptoms on sensitive crop species</th>
</tr>
</thead>
</table>
| **Seedling growth**     | trifluralin (Treflan), pendimethalin (Prowl®), metolachlor (Dual®), acetochlor (Harness®), dimethenamid (Outlook®) | **Broadleaf species:** swollen hypocotyls, callus on stem tissue at base of stems, root growth inhibited, inhibitors heart-shaped leaves, rough, crinkled leaves  
**Grass species:** stunted, thickened, red or purple in color, may appear drought stressed, “buggy whipping,” onion leafing or improper leaf unfurling |
| **Photosynthesis inhibitors** | Atrazine (A.Atrex®), metribuzin (Sencor®), linuron (Lorox) | **Broadleaf species:** chlorosis and necrosis at leaf tips and margins; interveinal chlorosis; brown speckling near leaf edges  
**Grass species:** chlorosis and necrosis at leaf tips and margin |
| **Cell membrane disruptors** | fomesafen (Reflex®/Flexstar®), flumioxazin (Valor®), sulfentrazone (Spartan®) | **Broadleaf species:** stunting; chlorosis and necrosis; leaf crinkling  
**Grass species:** stunting; veinal chlorosis; necrosis |
| **Pigment inhibitors** | clomazone (Command®), isoxaflutole (Balance®), mesotrione(Callisto™), topramezone (Impact), tembotrione (Laudis®) | **Broadleaf species:** white, chlorotic, then necrotic tissue  
**Grass species:** translucent, white, chlorotic, then necrotic tissue |
Appendix-I

Structures of Herbicides

Growth Regulators

Phenoxyacetic Acids/ (Aryloxy) Alkanoic Acids

Aromatic carboxylic herbicides are composed chemically of (1) an aromatic (benzene) ring structure, (2) one or more carboxyl (-COOH) group, and (3) various substitutions (e.g. –Cl, -CH₃, NH₂, NO₂ etc.) replacing hydrogen atom on the ring or aliphatic side chain or both.

![Benzene ring with positions](image)

2 position – Ortho
3 position – meta
4 position – para

Various substitutions are

<table>
<thead>
<tr>
<th>Substitution</th>
<th>Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>-OH</td>
<td>Hydroxy</td>
</tr>
<tr>
<td>-CH₃</td>
<td>Methyl</td>
</tr>
<tr>
<td>-NH₂</td>
<td>Amino</td>
</tr>
<tr>
<td>NO₂</td>
<td>Nitro</td>
</tr>
<tr>
<td>-O-CH₃</td>
<td>Methoxy</td>
</tr>
<tr>
<td>-S-CH₃</td>
<td>Methyl thio</td>
</tr>
<tr>
<td>-COOH</td>
<td>Carboxyl group</td>
</tr>
</tbody>
</table>

This group was developed in 1940s simultaneously in Britain and the USA following the discovery of MCPA and 2,4-D. It includes both 2-(aryloxy) alkanoic acid (phenoxy and pyridyloxy-) and also precursors of phenoxyacetic acid that are converted to the corresponding acids in vivo. The propionic acid derivatives contain a chiral centre and only the (R)(+) isomer are herbicidally active.

Important herbicides belonging to the group are:

- **2,4-D**: (2,4-dichlorophenoxy) acetic acid; **MCPA**: (4-chloro-2-methylphenoxy)acetic acid; **2,4,5-T**: (2,4,5-trichlorophenoxy)acetic acid
- **Dichlorprop**: 2-(2,4-dichlorophenoxy)propanoic acid; **Mecoprop**: 2-(4-chloro-2-methylphenoxy)propanoic acid
- **MCPB**: 4-(4-chloro-2-methylphenoxy)butanoic acid; **2,4-DB**: 4-(2,4-dichlorophenoxy)butanoic acid; **2,4,5-TB**: 4-(2,4,5-trichlorophenoxy)butanoic acid
Arylcarboxylic acids (Benzoic acids, picolinic acid, terephthalic acid)

There are three groups of arylcarboxylic acids based on benzoic acids, picolinic acid and terephthalic acid.

**Dicamba**: 3,6-dichloro-2-methoxybenzoic acid; **Chloramben**: 3-amino-2,5-dichlorobenzoic acid; **2,3,6-TBA**: 2,3,6-trichlorobenzoic acid; **Chlorthal-dimethyl**: 2,3,5,6-tetrachloro-diethylterephthalanolate
Chlorthal-dimethyl

Pyridines

Clopyralid: 3,6-dichloro-2-pyridinecarboxylic acid; Picloram: 4-amino-3,5,6-trichloro-2-pyridinecarboxylic acid; Triclopyr: [(3,5,6-trichloro-2-pyridinyl)oxy]acetic acid

Pyrimidinyloxybenzoic acids

Bispyribac: 2,6-bis[(4,6-dimethoxy-2-pyrimidinyl)oxy]benzoic acid; Pyriminobac: 2-[(4,6-dimethoxy-2-pyrimidinyl)oxy]-6-methoxyimino)ethyl] benzoic acid
Amino Acid Synthesis Inhibitors

**Imidazolinones**

**Imazamethabenz**: (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-4(3:2); 5-methylbenzoic acid (3:2); **Imazaquin**: 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic acid; **Imazethapyr**: 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid

![Imazamethabenz (generic), Imazamethabenz (meta), Imazamethabenz (para)](image)

**Imazaquin**

![Imazaquin](image)

**Imazethapyr**

![Imazethapyr](image)

**Triazolopyrimidine sulfonanilide or sulfonamide**

**Flumetsulam** (N-(2,6-difluorophenyl)-5-methyl[1,2,4]triazolo[1,5-a]pyrimidine-2-sulfonamide)

**Sulfonylureas**

Chlorimuron  Primisulfuron  Thifensulfuron

Triasulfuron  Nicosulfuron  Metsulfuron

Tribenuron  Rimsulfuron

Pyrazosulfuron

Amino Acid Derivatives

Glyphosate (N-(phosphonomethyl)glycine)

Lipid Inhibitors

Cyclohexanediones

Sethoxydim: 2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one; Clethodim: (E,E)-(6)-2-[1-[[3-chloro-2-propenyl]oxy]iminopropyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one; Alloxydim: methyl 2,2-dimethyl-4,6-dioxo-5-[1-[(2-propenyl)amino]butylidene]cyclohexanecarboxylate;
Sethoxydim

\[
\text{Sethoxydim} = \text{Alloxydim}
\]

Aryloxyphenoxypropionates

Seedling Growth Inhibitors

A. Root Inhibitors

1. Dinitroanilines

**Ethalfuralin**: N-ethyl-N-(2-methyl-2-propenyl)-2,6-dinitro-4-(trifluoromethyl)benzamine; **Pendimethalin**: N-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzamine; **Trifluralin**: 2,6-dinitro-N,N-dipropyl-4-(trifluoromethyl)benzamine; **Nitralin**: 4-(methylsulfonyl)-2,6-dinitro-N,N-dipropylbenzamine; **Fluchloralin**: N-(2-chloroethyl)-2,6-dinitro-N-propyl-4-(trifluoromethyl)benzamine;

- Ethalfuralin: ![Ethalfuralin](image1)
- Pendimethalin: ![Pendimethalin](image2)
- Trifluralin: ![Trifluralin](image3)
- Nitralin: ![Nitralin](image4)
- Fluchloralin: ![Fluchloralin](image5)

B. Shoot Inhibitors

1. Acetanilides

**Alachlor**: 2-chloro-N-(2,6-diethylphenyl)-N-(methoxymethyl)acetamide; **Acetochlor**: 2-chloro-N-(ethoxymethyl)-N-(2-ethyl-6-methylphenyl)acetamide; **Metolachlor**: 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl)acetamide; **Propachlor**: 2-chloro-N-(1-methylethyl)-N-phenylacetamide; **Butachlor**: N-(butoxymethyl)-2-chloro-N(2,6-diethylphenyl)acetamide; **Di-methenamid**: (RS) 2-chloro-N-(2,4-dimethyl-3-thienyl)-N-(2-methoxy-1-methylethyl)acetamide; **Pretilachlor**: 2-chloro-N-(2,6-diethylphenyl)-N-(2-propoxyethyl)acetamide;

- Alachlor: ![Alachlor](image6)
- Acetochlor: ![Acetochlor](image7)
- Metolachlor: ![Metolachlor](image8)
2. Thiocarbamates

**EPTC:** S-ethyl dipropyl carbamothioate; **Butylate:** S-ethyl bis(2-methylpropyl)carbamothioate; **Diallate:** S-(2,3-dichloro-2-propenyl)bis(1-methylthyl)carbamothioate; **Triallate:** S-(2,3,3-trichloro-2-propenyl)bis(1-methylthyl)carbamothioate; 
**Cycloate:** S-ethyl cyclohexylethylcarbamothioate; **Thiobencarb:** S-[(4-chlorophenyl)methyl]diethylcarbamothioate;

Photosynthesis Inhibitors

1. Triazines

**Ametryn**: N-ethyl-N'-(1-methylethyl)-6-(methylthio)-1,3,5-triazine-2,4-diamine; **Atrazine**: 6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-triazine-2,4-diamine; **Cyanazine**: 2-[[4-chloro-6-(ethylamino)-1,3,5-triazin-2-yl]amino]-2-methylpropanenitrile; 
**Simazine**: 6-chloro-N,N'-diethyl-1,3,5-triazine-2,4-diamine; **Metribuzin**: 4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one; **Hexazinone**: 3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4(1H,3H)-dione; 
**Aziprotryn**: 2-azido-4-isopropyl amino-6-methylthio-1,3,5-triazine; **Desmetryn**: N-methyl-N9-(1-methylethyl)-6-(methylthio)-1,3,5-triazine-2,4-diamine; **Prometryn**: N,N'-bis(1-methylethyl)-6-(methylthio)-1,3,5-triazine-2,4-diamine; **Terbutryn**: N-(1,1-dimethylethyl)-N'-ethyl-6-(methylthio)-1,3,5-triazine-2,4-diamine;
2. Phenylureas

**Diuron:** \( N'-(3,4\text{-dichlorophenyl})-N,N\text{-dimethylurea}; \)
**Fenuron:** \( N,N\text{-dimethyl-N9-phenylurea}; \)
**Chlorotoluron:** \( N'-(3\text{-chloro-4-methylphenyl})-N,N\text{-dimethylurea}; \)
**Fluometuron:** \( N,N\text{-dimethyl-N9-[3-(trifluoromethyl)phenyl]urea}; \)
**Isoproturon:** \( N,N\text{-dimethyl-N'-[4-(1-methylethyl)phenyl]urea}; \)
**Linuron:** \( N'-(3,4\text{-dichlorophenyl})-N\text{-methoxy-N-methyleurea}; \)
**Methabenzthiazuron:** \( N-(2\text{-benzothiazolyl-N,N'}\text{-dimethylurea}; \)
**Metoxuron:** \( N'-(3\text{-chloro-4-methoxyphenyl})-N,N\text{-dimethyl urea}; \)
**Monolinuron:** \( N'-(4\text{-chlorophenyl})-N\text{-methoxy-N-methylurea}; \)
**Monuron:** \( N'-(4\text{-chlorophenyl})-N,N\text{-dimethylurea}; \)
**Tebuthiuron:** \( N-[5-(1,1\text{-dimethylethyl})-1,3,4\text{-thiadiazol-2-yl}]N,N'\text{-dimethylurea}; \)
3. **Uracils**

**Bromacil:** 5-bromo-6-methyl-3-(1-methylpropyl)-2,4(1H, 3H)pyrimidinedione; **Lenacil:** 3-cyclohexyl-6,7-dihydro-1H-cyclopentapyrimidine-2,4(3H,5H)-dione; **Terbacil:** 5-chloro-3-(1,1-dimethylethyl)-6-methyl-2,4(1H,3H)-pyrimidinedione;

4. **Benzothiadiazoles**

**Bentazon:** 3-(1-methylethyl)-(1H)-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide;

5. **Nitriles**

**Dichlobenil:** 2,6-dichlorobenzonitrile; **Bromoxynil:** 3,5-dibromo-4-hydroxybenzonitrile; **Ioxynil:** 4-hydroxy-3,5-diodobenzonitrile;
6. Carbamate

Chlorpropham: 1-methylethyl 3-chlorophenylcarbamate; Propham: 1-methylethyl phenylcarbamate; Asulam: methyl[(4-aminophenyl)sulfonyl]carbamate; Desmedipham: ethyl[3-[(phenylamino)carbonyl]oxy]phenylcarbamate; Phenmedipham: 3-[(methoxycarbonylamino)phenyl] (3-methylphenyl)carbamate

7. Dicarboxylic Acid

Endothall: 7-oxabicyclo[2.2.1]heptane-2,3-dicarboxylic acid;

Cell Membrane Disrupters

1. Bipyridyliums
Paraquat: 1,1'-dimethyl-4,49-bipyridinium ion; Diquat: 6,7-dihydrodipyrido[1,2-a:2',1'-c]pyrazinedium ion
Difenzoquat: 1,2-dimethyl-3,5-diphenyl-1H-pyrazolium

2. Diphenylethers

Acifluorfen: Fomesafen: 5-[2-chloro-4-(trifluoromethyl)phenoxy]-N-(methylsulfonyl)-2-nitrobenzamide; Lactofen:
(±)-2-ethoxy-1-methyl-2-oxoethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate

Pigment Inhibitors

1. Isoxazolidinones

Clomazone: 2-[(2-chlorophenyl)methyl]-4,4-dimethyl-3-isoxazolidinone;
Objective

To teach students about the changing weed flora, new herbicides, their resistance, toxicity, antidotes and residue management under different cropping systems

Theory

UNIT I

Crop-weed competition in different cropping situations; changes in weed flora, various causes and effects

UNIT II

Structural, physiological and biological aspects of herbicides: their absorption, translocation, metabolism and mode of action; selectivity of herbicides and factors affecting them

UNIT III

Climatic factors and phytotoxicity of herbicides; fate of herbicides in soil and factors affecting them, residue management of herbicides, adjuvants

UNIT IV

Advances in herbicide application techniques; herbicide resistance; antidotes and crop protection compatibility of herbicides of different groups; compatibility of herbicides with other pesticides

UNIT V

Development of transgenic herbicide resistant crops; herbicide development and registration procedures

UNIT VI

Relationship of herbicides with tillage, fertilizer and irrigation; bioherbicides, allelochemicals; herbicide bioassays

Suggested Readings


