Chapter 16
Seedling Root/Shoot Inhibition

1. General Information

Herbicides in this group consist of only one chemical family, the acid amides. The specific mode of action is still unknown or not proven, therefore, they are classified according to their effects on plants. Most of these herbicides inhibit growth of seedling plants, and probably affect cell division or cell enlargement processes to some degree.

2. Mode of Action

The actual cause of plant death is not understood, but documented effects of these herbicides include:

- Inhibited fatty acid and lipid biosynthesis evident by reduced cuticular wax deposition
- Inhibited protein, isoprenoid (including gibberellin), and flavenoid (including anthocyanins) biosynthesis

One proposed theory is that the above effects are a result of the herbicide binding irreversibly to acetyl coenzyme A, a very important cofactor with many uses in biological pathways. Another theory links the acid amides to inhibitory binding of P450 monooxygenases, enzymes involved in the terpenoid biosynthesis pathway. Other modes of action have also been proposed.

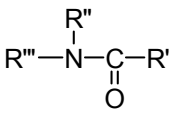
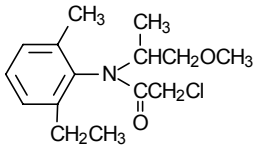
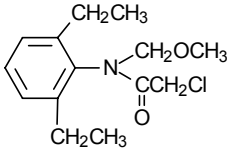
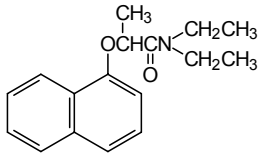
3. Site of Action

The site of action is unknown.

4. Symptoms

- germination is not inhibited but seedlings fail to emerge from soil
- if plants emerge they can have malformed leaves and grasses may be “buggy whipped” (caused by newest leaf not emerging from the whorl)
- in soybeans, new leaves may be heart shaped (puckered with “drawstring effect”)
- leaves may have a leathery texture

5. Herbicide Family

<table>
<tr><td colspan="3">Acid Amides</td></tr>
<tr><td>Base Structure</td><td>  </td><td>herbicides in this group are derivatives of acid amides; acid amides are named for their corresponding acid; if R’ is a methyl, the corresponding acid is acetic acid, the resulting acid amide is called an acetamide; if Cl is part of the R’ group then they are referred to as chloroacetamides; when one H of the amino group is repaced by a phenyl group the compound is an anilide; other acid amide herbicides include propanil POST and pronamide PRE</td></tr>
<tr><td>Examples</td><td colspan="2"> 
metolachlor (Dual)
 
alachlor (Lasso)
 
napronamide (Devrinol)</td></tr>
<tr><td>Others</td><td colspan="2">acetochlor (Harness/Surpass/Topnotch) propachlor (Ramrod)
butachlor (Machete) diphenamid
dimethenamid (Outlook/Frontier)</td></tr>
<tr><td>Metabolism</td><td colspan="2">plant – dealkylation, ring hydroxylation, conjugation
soil – microbial
half-life – metolachlor 112-124 d; alachlor 21 d; napronamide 70 d</td></tr>
<tr><td>Absorption & Translocation</td><td colspan="2">absorption by roots (broadleafs) and shoots (coleoptile of grasses) of germinating weed seedlings
translocated upward and throughout the plant in apoplast</td></tr>
<tr><td>Selectivity</td><td colspan="2">selective – not known; could be differential metabolism</td></tr>
<tr><td>Herbicide Use</td><td colspan="2">controls grasses and small-seeded broadleafs
metolachlor and alachlor – used PRE in corn, cotton, peanuts, potatoes, safflower, sorghum (safened), soybean
napronamide – used PRE in cole crops, eggplant, tomatoes, tobacco, nutbearing trees</td></tr>
</table>

6. General Comments

The herbicidal activity of the acid amides was discovered by Monsanto in 1952. Alachlor was discovered in 1966 and commercialized in 1969 for use in corn. Metolachlor, another

common acid amide, was first synthesized in 1972 by Ciba-Geigy and sold commercially in 1977.

In contrast to the herbicides discussed in the handout, the acid amide propanil (Stam) is an inhibitor of photosynthesis in Photosystem II and the amide herbicide pronamide (Kerb) is a mitosis inhibitor.

7. References

Ahrens, W. Herbicide Handbook, seventh edition. 1994. Weed Science Society of America, Champaign, IL.

Anderson, W.P. Weed Science – Principles and Applications, third edition. 1996. West Publishing, NY.

Ross, M.A. and C.A. Lembi. Applied Weed Science, second edition. 1999. Prentice Hall, NJ.