Herbicide Mode of Action Review-Implications for Resistance Management

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Outline for this morning...

- Definitions

- Working knowledge of mode of action
  - and why it is important
    - herbicide resistance management
  - revised PNW #437 publication on herbicide resistance

- Review of common modes of action
  - chemical families
  - used in many crops/non-crop areas common to Oregon
  - plant herbicide symptomology

- History of resistance

- Current status of herbicide resistance
  - Globally and in the PNW

- Weed management implications-diversification of products
Effective Chemical Weed Management

Herbicide application and placement

Herbicide uptake

Herbicide translocation
  movement within the plant through xylem and phloem tissue

Herbicide toxicity and activity

Herbicide metabolism and degradation
Mode of Action - Definitions

- Sequence of events from herbicide absorption to plant death

- Mechanisms by which a herbicide causes plant death

- The suite of plant process interfered with by a herbicide at the tissue or cellular level

- How a herbicide kills a plant
Site of Action

- Place in the plant where the herbicide acts...
  - specific mechanisms of action...

- Organized by “Groups”
  - Group number usually printed on the label
Herbicide Classification for Resistance Management

- Herbicides are grouped by site of action
- Users are able to determine related chemistries
- EPA and Agriculture Canada are calling for voluntary labeling that would include group number
Herbicide-Resistant Weeds and Their Management

When planning a herbicide program to prevent resistance, do not use herbicides from the same group more than once within three years.

This publication contains the 

Guide for Herbicide Rotation reference poster

The authors—Carol Mallory-Smith, Professor of Weed Science, Oregon State University; Andy Hulting, Assistant Professor and Extension Weed Specialist, Oregon State University; Doni Thill, Professor of Weed Science, University of Idaho; Don Morishita, Professor of Weed Science, University of Idaho; Jen Krenz, Faculty Research Assistant, Oregon State University.

http://extension.oregonstate.edu/catalog

“PNW 437”
Modes/Sites of Action

Synthetic Auxins

Amino Acid Synthesis Inhibitors

EPSP Synthase Inhibitors

ACCase Inhibitors

PPO Inhibitors
ACCase Inhibitors-Group 1

Mode of Action
– Prevents the formation of fatty acids, which are essential for the production of lipids. Lipids are vital in the integrity of cell membranes and thus new plant growth

Chemical Families
– Cyclohexanediones: clethodim (Select)
– Aryloxyphenoxypropionates: quizalifop (Assure II)
– Phenylpyrazoline: pinoxaden (Axial XL)
ACCase Inhibitors-Group 1

Site of Action
– Inhibits the ACCase enzyme which ceases the synthesis of fatty acids

Translocation
– Symplastic movement - translocate to all areas of new growth via phloem, no soil activity

Uses / Notes
– Postemergence “grass killers” , no BL activity
– Control many annual and perennial grasses
ACCase Inhibitors-Group 1

Symptoms
Only on Grasses:
- Injury first appears on new emerging whorl leaves
- Immediate growth stoppage
- Very gradual discoloration of tissue
- Slow acting, symptoms take 7 to 14 days to show up
- Chlorosis to reddening followed by necrosis of grass whorl
- Can pull out dead whorl, an early indicator (growing point separates from rest of the plant)
Amino Acid Synthesis Inhibitors-Group 2

Mode of Action
- Inhibits a specific enzyme (single site) which prevents production of essential amino acids

Chemical Families
- Imidazolinones: imazethapyr (Pursuit), imazamox (Raptor or Beyond), imazapic (Plateau), imazapyr (Arsenal or Chopper)

- Sulfonylureas: chlorimuron (Classic), nicosulfuron (Accent), primisulfuron (Beacon), thifensulfuron (Harmony GT), halosulfuron (Permit), chlorsulfuron (Glean or Telar), mesosulfuron (Osprey)
Amino Acid Synthesis Inhibitors-Group 2

Chemical Families, con’t.

- Sulfonanilides: cloransulam-methyl (FirstRate)

- Sulfonylaminocarbonyl-triazolinone:
  - flucarbazone (Everest)
  - propoxycarbazone (Olympus)
Site of Action
- Imidazolinones, Sulfonylureas, and Sulfonanilides prevent production of three essential amino acids by inhibiting the same enzyme, acetolactate synthase (ALS)

Translocation
- Move through xylem and phloem and accumulate in meristematic region, will see injury on new leaves
ALS Inhibition

**ALS Enzyme Action**
- substrate binds to enzyme

**Inhibition of ALS Enzyme**
- herbicide blocks normal substrate
Amino Acid Synthesis Inhibitors-Group 2

Uses / Notes
- PRE/POST weed control in various crops
- Immediate growth cessation
- Slow to develop, gradual chlorosis followed by necrosis of newest growth after several days
- Death of growing point
- Stunting, slow growth, death of plant may take up to 28 days
- IMI’s and SU’s are difficult to distinguish
Symptoms
Grass symptoms:
- General stunting
- Purpling of leaves, interveinal chlorosis of newly emerging leaves
- Chlorotic bands near base of leaf blade
- Lateral root pruning = bottle-brush appearance
- Irregular leaf shape (crinkled and wavy leaf margins)
Symptoms, con’t.

Broadleaf symptoms:

- Stunting, chlorosis, and purpling of the leaves
- Red or purple veins on the underside of broadleaf leaves (unique to IMI’s and SU’s)
- Irregular leaf shape (crinkled, puckered, and/or drawstring effect)
- Proliferation of additional shoots, new leaves are small
- Shortened internodes
Synthetic Auxins-Group 4

Mode of Action
- These herbicides disrupt hormone balance and protein synthesis in plants, leading to a variety of plant growth abnormalities

Chemical Families
- Phenoxy Acetic Acids: 2,4-D
- Benzoic Acids: dicamba (Banvel)
- Pyridines: fluroxypyr (Starane), picloram (Tordon), clopyralid (Stinger), triclopyr (Garlon 4), aminopyralid (Milestone)
Synthetic Auxins-Group 4

Site of Action
- Site(s) of action is unknown, believed to have multiple sites of action

Translocation
- Extensively translocated in xylem and phloem, herbicides accumulate in newest leaves and meristems
Synthetic Auxins - Group 4

Uses / Notes
- Primarily “broadleaf killers,” used for postemergence broadleaf control in corn, wheat, rye, barley, turf, pasture, range, roadsides
- Often have some soil activity

Symptoms
Broadleaf weeds / crops:
- Stem twisting and epinasty (downward twisting)
- Leaf malformations (leaf cupping, crinkling, strapping (parallel veins), puckering, bubbling)
- Callus tissue formation
Upward cupping of leaves on azalea. Common symptom with dicamba exposure.
Upward cupping of leaves on lilac from 2,4-D exposure.
Synthetic Auxins-Group 4

Symptoms, con’t.
Corn:
- Leaf rolling (onion-leafing), brace root fusion/malformation, stalk bending, stalk leaning, and brittleness, missing kernels in corn
- Stalk or stem brittleness

Wheat:
- Sterile heads, twisted flag leaves in small grains

*Drift, volatilization, and tank contamination are common.*
EPSP Synthase Inhibitors–Group 9

Mode of Action
– Inhibits a specific enzyme (single site) which prevents production of essential amino acids

Chemical Family
– Glycines: glyphosate
  (RoundUp formulations and others)
Site of action
- Glycines prevent production of three other essential amino acids by inhibiting EPSP Synthase

Translocation
- Move through xylem and phloem and accumulate in meristematic region, will see injury on new leaves
Uses / Notes
- Burndown applications preplant or chem fallow
- POST weed control in various glyphosate-tolerant crops
- Nonselective spot spraying applications
- Slow to develop, gradual chlorosis followed by necrosis of newest growth after several days
- Death of growing point
- Stunting, slow growth, death of plant may take up to 28 days
PPO Inhibitors-Group 14

Mode of Action
– These herbicides disrupt cell membranes

Chemical Families
– Bipyridyliums: paraquat (Gramoxone)
– Diphenylethers: oxyfluorfen (Goal)
– N-phenylphthalimides: flumioxazin (Chateau)
– Aryltriazolinonones: carfentrazone-ethyl (Aim)
  sulfentrazone (Spartan)
Site of Action
- Light causes the formation of free radicals. These radicals rupture plant cell membranes resulting in a rapid browning of tissue

Translocation
- None or very limited, necrotic spots

Uses / Notes
- Mostly foliar-applied - uptake into leaves
- Some soil-applied - root and shoot uptake
Symptoms
- Symptoms vary somewhat with herbicide and spray additive
- Rapid necrosis of plant tissue (1 to 2 hours)
- Leaves may have a water-soaked appearance or burned appearance followed by wilting an rapid desiccation
- Burnt, crispy brown tissue, leaf speckling
- Only kills the tissue it comes into contact with
- Plant parts not covered my survive
- Activity increases with sunlight, temperature, and humidity
Herbicide-Resistant Weeds and Their Management

When planning a herbicide program to prevent resistance, do not use herbicides from the same group more than once within three years.

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Herbicide Resistance

- Herbicide resistance is the result of genetic, biological, ecological and management factors.
Herbicide Resistance

- Herbicide resistance is the heritable response of a weed biotype to survive a herbicide treatment to which the wild type population was susceptible
Different Than Herbicide “Tolerance”

- The wild type population was never susceptible to the herbicide
  - glyphosate (Roundup) and common mallow

- Selective herbicides are possible because of crop tolerance
  - diclofop (Hoelon) in wheat
  - atrazine in corn
Multiple- and Cross-Resistance

- Cross-resistance is resistance to different herbicides with the same site of action
  - example, imazethapyr (Pursuit) and thifensulfuron (Harmony)
  - Both Group 2 ALS Inhibitors

- Multiple-resistance is resistance to two or more chemically unrelated herbicides with different sites of action
  - example, imazethapyr (Pursuit) and Atrazine
  - Group 2 ALS Inhibitor and Group 5 Photosystem II
Kochia populations are resistant to dicamba and fluroxypyr. This is an example of cross-resistance.

Both herbicides are synthetic auxins. Atrazine is a triazine and metsulfuron is a sulfonylurea.

Kochia populations are resistant to atrazine and metsulfuron. This is an example of multiple-resistance.
What to Look for in the Field

- No obvious application/environmental problems
- Other weed species are controlled
- Irregular shaped patches
- Records show repeated use of herbicides with the same site of action
- Previous failure at the same site
- No herbicide symptoms on target weed
Historical Perspective

- 1908 - insecticide resistance
- 1940 - fungicide resistance
- 1957 - herbicide resistance
- 1967 – triazine resistant common groundsel
Herbicide Resistant Weeds

- 393 biotypes
- 211 species
  - 124 dicots
  - 87 monocots
- > 680,000 “fields”

Source: Dr. Ian Heap
www.weedscience.com
# Herbicide Resistant Weeds in Oregon

- prickly lettuce
- kochia
- Russian thistle
- wild oat
- Italian ryegrass
- common lambsquarters
- annual bluegrass
- pigweed spp.
- common groundsel
- downy brome
- small-seeded falseflax

**Probably Others?**
glyphosate and/or glufosinate resistant annual ryegrass
multiple resistant annual ryegrass in winter wheat
Risks for Oregon?

- Forestry
- Christmas Trees
- Roadside
- Crops
What To Do If You Suspect Resistance

- Do not re-spray with a herbicide with the same site of action
- Do not use a higher rate of the same herbicide
- Document resistance
  - Contact OSU Weed Group
Prevention and Management

- Prevention is the best strategy

- Requires
  - knowledge of the herbicide
    - need to know the herbicide site of action
    - need to know about persistence
    - need to know the control spectrum
  - knowledge of the weed biology
  - willingness to change system
Prevention & Management

- Limit applications of herbicides with the same site of action
- Use tank-mixtures or sequential treatments of herbicides
Tank-Mixtures Must

- Be effective on the same target weeds
- Have the same soil persistence
- Include different sites of action
- Have the same application timing
Herbicide use should be based on an IWM program that includes:

- scouting
- historical information related to herbicide use and crop rotation
- considers tillage or other mechanical control, cultural, biological and other chemical control practices
Prevention & Management

- Prevent resistant weeds from producing seeds

- Clean equipment before leaving an area with resistant weeds
Herbicide Resistance in the 21st Century

- More weed species will develop resistance

- Resistance to most if not all herbicides will occur

- The introduction of herbicide-resistant crops will increase the selection of herbicide resistant weeds due to increased selection pressure
  - RR Crops in Midwest and Southeast
    - pigweed spp. and horseweed in corn, cotton, soybean
Herbicide Resistance in the 21st Century

- Herbicides will still be used as the primary weed control method for the foreseeable future.

- No herbicides have been removed from the marketplace because of resistance.

- However, growers have lost the use/value of herbicides for control of specific weeds at some sites.
Summary

- Herbicide resistance is the result of the biology of the weed and management factors

- We can not change the biology so must change the management
Reminders-Review PNW Handbook

- Review labels/handbook for the following prior to making applications: pnwhandbooks.org/weed
  - Tank-mixing Instructions
    - N Sources (as a % of carrier and/or with surfactant loads)
    - Insecticides
    - Fungicides
    - Other Herbicides
  - Application Timings
  - Herbicide Mode of Action Info and Group Numbers
  - Pre-Harvest Intervals
    - Grazing and feeding restrictions for various herbicides
  - Changing Plantback Restrictions
Weed Management Handbook

This handbook is designed as a quick and ready reference for weed control practices and herbicides used in various cropping systems or sites in Idaho, Oregon, and Washington.

This handbook will be useful to Extension agents, company field representatives, commercial spray applicators and consultants, herbicide dealers, teachers, and producers. More about the PNW Weed Management Handbook