WSSA Liaison to EPA (Office of Pesticide Programs – OPP)
Interim Report to the WSSA Board of Directors – Quarter 2 2011
July 12, 2011

Second quarter activities:
April 11-13: I spent three days in Washington at the EPA Office of Pesticide Programs. The week was quieter than usual due to the federal government shutdown threat that was not resolved until late on April 8. Many meetings had been cancelled in preparation for the shutdown (the office would have closed). I did meet with Dan Kenny, Skee Jones, Frank Wong (APS-SME), and Bill Chism to discuss herbicide resistance issues on April 11. I extended the invitation from NCWSS weed scientists to hold a herbicide resistance tour for agency staff and discussed other WSSA activities related to herbicide resistance, including the National Academy of Science summit. The herbicide branch has several new staff members, including Bo Davis, product manager, and two reviewers. I met briefly with them to discuss general issues in weed science and complexities of herbicide labelling. Frank Wong and I also met again with the Future Directions Workgroup about how to increase visibility of EPA and opportunities in EPA within our professional societies.
April 13: Frank Wong and I attended, with Bill Chism, Skee Jones and others from EPA, the Insecticide Resistance Action Committee (IRAC) meeting at Crop Life. Gene Reagan (ESA-SME) was on the phone; he had given a presentation to the group remotely prior to our arrival. We discussed resistance management activities; Frank talked about APS and FRAC interactions and activities and I described WSSA and HRAC activities. We discussed mechanism of action labeling on pesticides and how important that information is for educational outreach in the area of resistance management. We also talked about the resistance terminology issue and how Frank, Gene, and I are working on defining a list of terms as they are used in our respective disciplines. After the meeting, members of the EPA resistance workgroup, Frank and I held a conference call with Gene to discuss the outcomes of the IRAC meeting. We felt that the resistance terminology discussion among the three SMEs and the resistance workgroup is very important activity.

May 11-13, 2011: I spent three days in Washington at the EPA Office of Pesticide Programs and at a Crop Life forum "Judging the Quality of Scientific Research" (May 13). Kurt Getsinger, aquatic weed science subject matter expert (SMEs) was also on site during the week. This offered us an opportunity to discuss weed science issues with each other and with agency personnel.
Resistance Workgroup meeting: I attended a Resistance workgroup meeting led by Bill Chism (BEAD). We continued our discussion about the similarities and critical differences in resistance terminology and management among our disciplines. Frank Wong provided a combined list of definitions as a working document. Of interest is that there are significant differences in terms used by the different disciplines. After the meeting, I sent the combined list to the following WSSA committees and groups: Herbicide Resistance, Extension, HRAC, WSSA officers, and other individuals whose feedback is valued. The combined list is attached to this report – feedback on the list (corrections, updates, etc.) would be welcome. Please note that changes have already been made to this list by ESA (not included in this draft).
I also asked about interest in the HR tour offered by NCWSS; EPA staff have travel restrictions due to budget issues. We discussed whether they could attend if external funding was secured. I brought this information back to the organizers.
Another meeting relative to the herbicide resistance issue was with Kathy Davis (Field External Affairs Division). We discussed how FEAD interacts with states in the area of pesticide applicator training and certification. I showed her the resistance management training modules under development by WSSA and we discussed who I should speak with relative to integrating herbicide resistance training into pesticide certification programs.
May 13: I was a panel member at the forum "Judging the Quality of Scientific Research" sponsored by Crop Life. Randall Lutter moderated the forum where Chris Borgert, Applied Pharmacology and Toxicology, and Sheldon Krimsky, Tufts University, made presentations and an 'Expert Advisory Group' held a discussion on the following:

Topic: ***Judging the quality of scientific work in chemical evaluation***

- ***Regulatory science and research science: similarities and differences***
- ***Strengths and limitations of chemical testing data from regulated industry, academics and government labs***
- ***Identifying criteria for judging quality***

Goal: To assure a strong scientific basis for management of risks related to chemicals to protect the environment and public health.

Purpose

- *The advancement of science in regulatory decision-making and public policy through constructive dialogue.*
- *The identification of different views on how to ensure high quality scientific work is used to make regulatory decisions and public policy.*
- *Beyond the dialogue, to seek agreement / consensus.*

Panel members represented public policy, regulatory agencies, industry, academia, and public interest groups. The discussion was lively and centered on the need for improved transparency of methodology, data analysis and results from research used in regulatory decisions, whether the investigators are in the public or private sectors. The panelists, led by Dr. Lutter, are currently working on a consensus document that will be published as Viewpoint of *ES&T.* At this time, I am not certain that this effort will come to fruition due to differing views and agendas of the participants. I will provide a copy of the reference if the document is published.

Other Activities: I continued to work as a member of the Herbicide Resistance Education Committee on the Herbicide Resistance Training Modules. I have also participated in WSSA Science Policy and Federal IPM Coordinating Committee meetings by conference call. I have worked with Bryan Young and Kevin Bradley, tour organizers, Joyce Lancaster, WSSA, and EPA staff and management to coordinate travel for the herbicide resistance tour that will be sponsored by WSSA and NCWSS in August. The tour activities will include:

Dates: August 15-17, 2011

Day 1

am Flights from DC to Memphis to Colombia, MO
Noon Arrive in Colombia, MO and lunch
1pm Tour bus from Colombia, MO to glyphosate-resistant giant ragweed site (Host – Kevin Bradley)
5pm Travel on tour bus to St. Louis, MO (Host – Kevin Bradley)
7pm Check-in at hotel and dinner (Host – Kevin Bradley and Bryan Young)

Day 2

7am Breakfast at hotel
8am Travel on tour bus to glyphosate-resistant waterhemp site (Host – Bryan Young)
11:30 Depart field site
Noon Lunch (Host – Bryan Young)
1pm Depart for Arkansas while perhaps stopping to visit with other areas/people of interest (Host – Bryan Young)
6pm Arrive in Arkansas (Host – Jason Norsworthy and Bryan Young)
7pm Check-in at hotel and dinner (Host – Jason Norsworthy and Bryan Young)

Day 3

7am Breakfast at hotel
8am Travel on tour bus to glyphosate-resistant Palmer amaranth site (Host – Jason Norsworthy)
Noon Lunch and travel to Memphis airport (Host – Jason Norsworthy)
pm Return flights to DC

Individual Hosts Representing WSSA:

Dr. Kevin Bradley **Associate Professor, Weed Science** **Division of Plant Sciences** **University of Missouri**	**Dr. Jason Norsworthy** **Associate Professor, Weed Science** **Crop, Soil and Environmental Sciences** **University of Arkansas**
Dr. Jill Schroeder **Professor, Weed Science and EPA SME** **Entomology, Plant Pathology and Weed Science** **New Mexico State University**	**Dr. Bryan Young** **Professor, Weed Science** **Dept. of Plant, Soil and Agricultural Systems** **Southern Illinois University**

Planned activities for third quarter:
August 7-11: I'll be at the American Phytophathology Society meeting in Hawaii. I am involved in two activities: 1- I am presenting an invited paper (topic herbicide resistance management) on behalf of the Herbicide Resistance Action Committee (HRAC). 2- I am participating in the APS-Public Policy Board meeting to discuss the activities of the liaison (subject matter expert) and benefits of maintaining this program for the society and agency.
August 15-17: I'll be in Missouri (various locations) on a herbicide resistance tour sponsored by the North Central Weed Science Society for EPA staff that I work with as liaison. We will be touring and visiting with growers, consultants and university scientists about the issue of resistance, management approaches, and adoption of management strategies.
September 21-23: Represent, with President Mike Barrett, WSSA at the Plant Biology Research Summit, Organized by the American Society of Plant Biologists and Howard Hughes Medical Institute, Chevy Chase, MD

Respectfully submitted,
Jill Schroeder
WSSA SME/liaison to EPA

DRAFT RESISTANCE TERMS

Term	Fungicide	Insecticide (changes not included)	Herbicide
Acquired Resistance	Resistance to a fungicide in a population acquired by long-term exposure to sub-lethal amounts over multiple pathogen generations. [not used commonly]	Resistance that develops over extended periods of exposure (not a very useful term).	[NU]
Altered Target-Site Resistance	Immunity or tolerance to a fungicide due to a genetic change leading to an alteration in the target enzyme, protein, or molecule that leads to reduced or no binding of the fungicide.	Resistance which develops through alteration of the target site molecule, thus resulting in reduced binding of the insecticide.	A change in enzyme or protein receptor (i.e., target site) of the herbicide in a resistant weed biotype such that the herbicide does not bind or bind as efficiently as susceptible biotypes [CU]
Artificially Induced Resistance	The development of resistance to a fungicide caused by repeated exposure to sub-lethal doses. Used commonly in the context of the selection of individuals/mutants in lab studies. (See laboratory resistance)	(not a very useful term).	
Behavioral Resistance	[not used]	The ability for an insect to detect and avoid insecticides, thus influencing a change in the behavior of the insect, with a potential population increase in non-treated areas.	[NU]
Continuous Resistance	The establishment of stable populations of fungi biochemically resistant to a fungicide. [not used commonly] This may also refer to the pattern of resistance development in populations where a continuous, quantitative range of sensitivity values can be detected (see directional resistance, quantitative resistance)	An established insect population which is resistant to an insecticide, in contrast to non-continuous which breeds back to susceptibility after a period of time.	[NU]
Cross-Resistance	Resistance to multiple fungicides that share the same biochemical mode of action or target site.	A single mechanism of resistance conferring resistance to a number of chemicals.	A weed biotype that is resistant to two or more herbicides of the same or different chemical families due to the presence of a single resistance mechanism [CU] .
Directional Resistance	A unimodal pattern of resistance development where selection within a population with a continuous, quantitative range in fungicide sensitivity results in an increased frequency of individuals with lower fungicide sensitivity (e.g. the population distribution shifts directionally towards	Development of resistance resulting in an increasing percentage of insects expressing reduced susceptibility towards an insecticide.	[NU]

	a higher frequency of less sensitive individuals, but the distribution remains a continuous range of fungicide sensitivity). Typically, increased rates of fungicide can provide some control of the population under field conditions. (See also continuous resistance, quantitative resistance)		
Discontinuous Resistance	A bimodal pattern of resistance development where selection of a population results in the increase in frequency of individuals greatly insensitive or immune to the fungicide, usually caused by a target site mutation. (See also qualitative resistance, discrete resistance, disruptive resistance)	Development of resistance with a percentage of the insects expressing reduced susceptibility in some portions of the population in contrast to other areas.	
Discrete Resistance	(See qualitative resistance)	?	[NU]
Disruptive Resistance	(See qualitative resistance)	?	[NU]
Field Resistance	A situation where the frequency of biochemically resistant individuals in a pathogen population is high enough (due to repeated selection events) so that the effectiveness of the fungicide application is compromised such that decreased control is noticed in the field. (See also practical resistance)	Resistance in an insect population which is derived from repeated exposure to an insecticide, thus with reduced efficacy of field applications, maybe resulting in control failures.	Same as field-evolved resistance or at levels recognized by a grower [MU]
Field-Evolved Resistance	Resistance development in pests in response to field applications of fungicides where their repeated use results in the selection of naturally immune or tolerant individuals to a frequency where control with fungicides is compromised. (See practical resistance) [not used commonly]	Resistance derived from repeated exposure from field applications of an insecticide.	Development of resistance in a normal pattern of herbicide use as opposed to selection for resistance via greenhouse or laboratory selection methods [CU]; evolved resistance in a weed population over time in a field as a result of past selection pressure.
Hormoligosis	[not used]	Stimulating insect activity through exposure to sublethal doses of insecticide. (?)	[NU]
Insensitivity	The development of biochemical immunity to a fungicide. (See resistance)	Reduced sensitivity at the target site, especially for insects not ever affected by the insecticide. (Target site insensitivity)	[CU], e.g., target site enzyme single-step resistance
Laboratory Resistance	Selection of resistant pests through repeated exposure under controlled conditions. This is sometimes related to the creation of mutants in the lab with mutagens and subsequent selection with	Resistance selected from controlled laboratory conditions (i.e. artificial selection).	resistance developed via artificial selection procedures [MU]

	fungicides. Resistant pests created in this manner may or may not reflect genetic mutations that would occur under natural conditions. (see artificially induced resistance)		
Low-Level Resistance	The development of tolerance individuals to a fungicide that may not be significant relative to the rates or doses of fungicides used for commercial control. Alternately, the development of a low frequency of immune individuals to a fungicide but at a frequency so low that control is not compromised.	The development of reduced sensitivity levels which may be insignificant to some normal control practices; most likely to be first detected in a reduced length of control period.	Mechanism of resistance that alters how the end user uses the herbicide, but level of resistance <100 fold [CU]; Suggested: resistance ratio $\leq$ 5 (moderate: 6-10; high: 11-100; very high: >100.
Major Gene Resistance	Resistance associated with changes to a single gene that affects the fungicide binding characteristics of the target molecule, protein or enzyme. Typically associated with a target site mutation that confirms immunity to the fungicide.	Resistance due to single gene, as opposed to being controlled by several (minor) genes.	Resistance controlled by a single, major gene with large phenotypic effect. [CU]
Major Resistance	Pest resistance associated with high economic or biological impact. [ambiguous term]	Resistance levels which highly impact the economics of the management system, relates to population density.	[NU]
Mechanism of Resistance	The process by which pests become biochemically resistant to fungicides. Processes are typically related to target site mutations, decreased binding to the target site, increased gene expression, detoxification or degradation of the fungicide, or efflux of the fungicide away from the target site. This may also refer to the selection process for pest populations and the patterns of increased frequencies of resistant individuals in response to fungicide use strategies.	Behavioral and physiological changes that occur within the insect, allowing for the development of resistant traits.	Response of the plant that reduces the efficacy of the herbicide (e.g. target site resistance, exclusionary resistance, over-expression target site) [CU] Biophysical, biochemical, or physiological basis for evolved resistance (e.g., target site resistance (insensitivity, over-expression); non-target site resistance (enhanced detoxification, reduced translocation, etc.) Clarification: Should sequestration or exclusion be included under the general term 'translocation'?
Metabolic Resistance	Biochemcal resistance development based on detoxification or degradation of the fungicide, or	Resistance which develops through the use of detoxifying enzymes to break down	Situation where the herbicide undergoes rapid detoxification before it reaches

	efflux of the fungicide away from the target site	insecticide molecules into smaller soluble compounds, which then can be excreted, or the sequestration of the insecticide, such as is the case with amplified esterases in mosquitoes.	target site of action; can be via oxidation, reduction, hydrolysis, and conjugation [CU]
Mode Of Action	The biological process which a fungicide specifically inhibits (biochemical mode of action). For example, the biochemical mode of action of QoIs is the inhibition of cellular respiration. See also target site of action, which describes the exact location of a molecule that a fungicide binds to. This may also describe the temporal and spatial characteristics of a fungicide in inhibiting the life or infection cycle of a pest (physical mode of action). For example, QoIs are strongest at inhibiting spore germination and infection processes and have a "preventive" mode of action.	The insect physiological process, biochemical process and/or molecular target that is affected by the insecticide and by which the insecticidal action occurs or is thought to occur. How the insecticide works.	Response of plant to herbicide [CU] The plant processes affected by the herbicide, or the entire sequence of events that results in death of susceptible plants; includes absorption, translocation, metabolism, and interaction with the site of action.
Multi-Step Resistance	This refers to the pattern of selection in a population with a continuous distribution of sensitivities to a fungicide, and when resistance is conferred by more than one target site mutation or genetic mechanism. Each of the mutations or mechanisms gives an additive effect with individuals with a few of these may have a moderately tolerant phenotype, and those with multiple mutations or mechanisms have a highly tolerant phenotype. This may also be referred at as "multigenic" resistance. The effect of the different Applications select for increasingly tolerant individuals dependent on the dose of fungicide used, e.g. low doses select out the most sensitive individuals allowing for the survival of moderately sensitive ones and higher doses select out low and moderately sensitive individuals. Very high doses may have a neutral overall effect on the pest, as the dose is so high that even the least sensitive individuals are still controlled. DMI fungicide resistance is a good example of this phenomenon. See also one-step resistance, quantitative resistance and qualitative resistance.	Resistance which develops from mutations across multiple target sites, and where each mutation exhibits an additive effect.	[NU]
Multiple	Biochemical resistance development to two or	Resistance to multiple unrelated	Description of weed populations with

Resistance	more unrelated fungicide classes. This may result from selection of individuals naturally resistant to two or more fungicides or the sequential selection of individuals resistant to one fungicide class which then naturally obtain a mutation conferring resistance to another fungicide and are subsequently selected for by the use of the latter fungicide.	insecticides, which is conferred by multiple mechanisms - multiple mechanisms here vs one mechanism in cross resistance. (Some feel the appropriate term should only be cross resistance).	two or more distinct resistance mechanisms [CU]
Negative Cross-Resistance	A situation where a fungicide only affects individuals resistant to another class of fungicides. An example is the N-phenylanilines, which are only toxic to individuals that are benzimidazole resistant and ineffective against benzimidazole sensitive individuals. Note that multiple resistance can develop to both fungicides, such in the previous case where an adjacent target site mutation in beta-tubulin also conferred resistance to N-phenylanilines in benzimidazole resistant individuals.	An occurrence where the insect develops resistance toward one insecticide but greater susceptibility towards another.	A situation where a biotype resistant to one herbicide or chemical family has greater sensitivity to other herbicides [CU]; A resistant population that is more sensitive to a herbicide than a susceptible population of the same species
Partial Cross-Resistance	A situation where biochemical resistance to one fungicide also confers low levels of resistance or tolerance to another fungicide class with a different biochemical mode of action.	An occurrence where the insect develops resistance toward one insecticide and low-level change in response toward another unrelated insecticide.	[MU]
Penetration Resistance	[not used]	A resistant trait which develops from mechanisms that prevent the insecticide from penetrating through the cuticle and/or epidermis into the hemolymph and reaching the target site.	[NU]
Practical Resistance	The situation where the frequency of biochemically resistant individuals in a population reaches a point at which field or commercial fungicide applications no longer provide aesthetically or economically acceptable control. This is a relative term as "acceptable levels of control" amongst crops can be variable depending on the use or value of the crop. (See field resistance)	The level of resistance to where conventional field applications of an insecticide no longer provide acceptable control of the insect pest.	The level of resistance that reduces the economic effectiveness of the herbicide to the grower [MU]
Progressive Resistance	See multi-step resistance and quantitative resistance. [not commonly used]	see multi-step resistance	[NU]

Qualitative Resistance	A patter of resistance development in populations where there is a distinct separation between sensitive and resistant individuals. Individuals are either susceptible to the fungicide or resistant to levels of the fungicide that could be feasibly used. This is typically associated with target site mutations that confer immunity to a fungicide for an individual.	(?)	Synonymous with major gene resistance. [MU]
Quantitative Resistance	A pattern of resistance development in populations where there is a continuous range of sensitivity amongst individuals in a population. Individuals may have increased tolerance to a fungicide but increased doses may still be effective. This is typically associated with target site mutations, metabolic resistance mechanisms or other genetic changes that confer tolerance but not immunity to a fungicide.	(?)	Continuous or quantitative variation in susceptibility to a herbicide in a weed population, caused by accumulation of minor genes with small additive effect in progeny due to outcrossing that may lead to practical field resistance. Synonymous with polygenic resistance. [CU]
Resistance Ratio	The ratio of resistant individuals relative to sensitive (wild-type) individuals in a population. This may also refer to the difference between mean population sensitivities (typically expressed as 50% effective dose (ED50) values) when populations exhibit a quantitative pattern of resistance development.	The measure of resistance in an insect population, which is calculated by dividing the LD50 of the resistant population by the LD50 of the susceptible population.	The fraction of resistant plants that survive herbicide application to the corresponding fraction of susceptible plants; [CU] suggest this definition refers to resistance frequency of individuals in a population, sometimes erroneously coined with 'level' of resistance; resistance ratio or index: - effective herbicide dose reducing growth or survival by 50% compared with the nontreated control of a resistant compared with a susceptible population of the same species.
Resistance	Decreased sensitivity to a fungicide that results in immunity or tolerance to the fungicide in an individual pathogen from a population. (see also biochemical resistance) Also may refer to the presence of resistant individuals within a population of fungal plant pathogens.	The development of an insect strain that is capable of surviving a dosage or dose that is lethal to the majority of individuals of the same species, by means of a genetic inheritable change that has increased in frequency in response to selection, and may impair control in the field.	Herbicide resistance is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. In a plant, resistance may be naturally occurring or induced by such techniques as genetic engineering or selection of variants produced by tissue culture or mutagenesis. [CU]
Resurgence	The situation where the application of a fungicide to a population containing resistance to the	A rapid increase in numbers of a pest that has previously been suppressed by an	[NU]

	fungicide causes an increase in this pest's damage or activity. This is due to the negative affect of the fungicide on other competing pests or organisms but essentially no effect on the resistant population, putting them at an ecological advantage.	insecticide. Earlier, the concept of resurgence due to elimination of natural enemies may be a bit limiting; we now know that pesticides can alter plant defense biochemical pathways that in turn may increase pest populations not because there are no enemies but because they change the plant physiology that may increase fecundity for example. This has been observed with mites and aphids. J Econ Entomol. 2002 Aug	
Single-Step Resistance	Resistance conferred by a single mechanism of resistance such as a single target site mutation or single metabolic change. This may also be referred to as "monogenic" resistance. Also see multiple-step resistance, quantitative resistance and qualitative resistance.	Resistance which develops from a single behavioral or physiological alteration.	[NU]
Sequential Resistance	Refers to multi-step or quantitative patterns of resistance development. Alternately, this could mean resistance to one class of chemistries followed by reliance on another class as the fungicides to which resistance then develops, etc. [ambiguous term]	Term used to assist with management concepts involving insecticide resistance replacement chemistry. In order to minimize the loss of multiple chemistries, a particular insecticide or class might be used (in contrast to others) which would not preclude the loss of potentially important replacement chemistries if resistance were to occur.	
Sustained Susceptibility	The situation where either a pest has failed to develop resistance to a fungicide despite repeated use of it due to biological or behavioral reasons, or a situation where biochemically resistant individuals are present but are maintained at a low level, thus practical resistance never develops. [not used commonly]	The inability for an insect to develop high levels of resistance toward an insecticide.	[NU]
Target Site Of Action	The physical site of interaction between a fungicide and the pest. For single site of action fungicides, this is a specific enzyme, protein or molecule involved in a key biological process. For example, QoIs bind specifically to the Qo-site of cytochrome bc1. Also see mode of action, which describes the biological process inhibited by the fungicide.	The specific molecule (most commonly proteins: receptor, channel, enzyme, transcription factor), or when unknown the physiological component or pathway that is directly affected by an insecticide.	Similar to mechanism of action; specific biochemical or physiological pathway disrupted by herbicide [CU]; The biochemical site within a plant with which a herbicide directly interacts. Mechanism of action is a synonym.

Tolerance	Reduced sensitivity of a individual pest to a fungicide conferred by genetic changes relative to a wild-type individual. Tolerant individuals still may be affected by increases doses of a fungicide. In contrast, immunity describes a situation where the individual has a mutation that causes the target site to no longer be able to bind to the fungicide or at such a low level that commercially useable doses of fungicide are ineffective for control.	The natural ability for an insect to withstand insecticide exposure. For synthetic insecticides, this is more commonly due to the metabolic capabilities of a particular species; for Bt toxins it may be due to differences in receptor binding, toxin excretion, etc. *It is important to distinguish it from Resistance because this term "tolerance" has been sometimes used wrongly to indicate low resistance ratios in populations. Tolerance is the estimated (upper) natural range of survivorship of a population, previous to exposure or selection by the toxicant. For example one could say that H. zea is more tolerant than H. virescens to the Cry1Ac Bt toxin. It is inherent to the species.*	Herbicide tolerance is the inherent ability of a species to survive and reproduce after herbicide treatment. This implies that there was no selection or genetic manipulation to make the plant tolerant; it is naturally tolerant. [CU]

Additional Suggested Terms

Term	Fungicide	Insecticide	Herbicide
Accession			A collection of individual plants of a weed species whose characteristics (genetic, physiological, biochemical, or biological) are yet to be determined.
Allele			An alternative form or copy of a gene.
Biochemical Resistance	Resistance to a fungicide in an individual that results from genetic changes that confer tolerance or immunity. Distinct from resistance in the context of populations that describes the frequency of biochemically resistant individuals in a population		
Biotype			A plant selection that has a unique genotypic pedigree.
Dominance			State of an allele whose phenotypic expression is similar both in the

	homozygous and heterozygous stages.
Ecotype	A biotype that has adapted to a specific growing environment.
Evolution	Progressive change in the gene pool of a given weed (species) population in response to most recent growing conditions (herbicides in this context).
Fitness	Ability of a biotype to survive and reproduce in an environment that may or may not include herbicide treatment.
Genotype	The complement of a plant's complete hereditary information.
Hormesis	Stimulation of growth processes in plants treated with low doses of herbicide(s).
Inheritance	Process of transfer of a genetic trait from one generation to the next.
Mating System	System by which pollen moves from the anthers to the stigma of the same flower or different flowers on the same plant (selfpollination), or to stigma of flowers on a different plant (cross-pollination) of a weed species.
(Gene) Mutation	An inheritable change to genetic material or the process resulting in such a change.
Population	A group of plants of a single

		weed species with potential to interbreed and inhabiting a specific geographic area.
Recessive		Condition of an allele whose expression is veiled by a dominant allele in the heterozygous stage.
Reduced Sensitivity	(See Tolerance)	
Selection Pressure		The effectiveness of natural selection in altering the genetic composition of a population over a series of generations.
Trait		A genetic characteristic of interest.